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Please see brief summary of prescribing information on the next page.

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Professional Use Information

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CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** *CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.*
2. **Congestive Heart Failure.** *Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).*
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** *Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.*
4. **Acute Hepatic Injury.** *In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)*

PRECAUTIONS

General. *CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.*

Drug Interaction. *Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)*

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. *A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.*

Pregnancy. *Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.*

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. *Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.*

Pediatric Use. *Safety and effectiveness in children have not been established*

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	*Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.*
Nervous System:	*Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.*
Gastrointestinal:	*Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.*
Dermatologic:	*Petechiae, pruritus, photosensitivity, urticaria.*
Other:	*Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.*

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

Issued 9/86

See complete Professional Use Information before prescribing.

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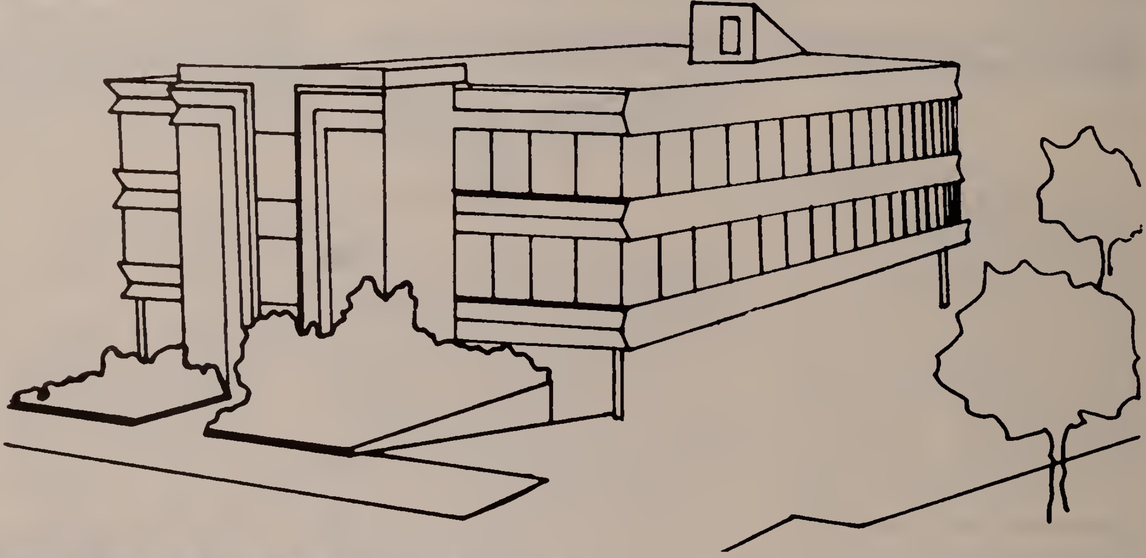

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Before prescribing, please consult complete product information, a summary of which follows:
CONTRAINDICATIONS: Hypersensitivity to trimethoprim or sulfonamides; documented megaloblastic anemia due to folate deficiency, pregnancy at term and during the nursing period; infants less than two months of age
WARNINGS: FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS.
BACTRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. Clinical signs, such as rash, sore throat, fever, pallor, purpura or jaundice, may be early indications of serious reactions In rare instances a skin rash may be followed by more severe reactions, such as Stevens-Johnson syndrome, toxic epidermal necrolysis, hepatic necrosis or serious blood disorder Perform complete blood counts frequently.
BACTRIM SHOULD NOT BE USED IN THE TREATMENT OF STREPTOCOCCAL PHARYNGITIS Clinical studies show that patients with group A ß-hemolytic streptococcal tonsillopharyngitis have a greater incidence of bacteriologic failure when treated with Bactrim than with penicillin
PRECAUTIONS: *General:* Give with caution to patients with impaired renal or hepatic function, possible folate deficiency (e.g., elderly, chronic alcoholics, patients on anticonvulsants, with malabsorption syndrome, or in malnutrition states) and severe allergies or bronchial asthma. In glucose-6-phosphate dehydrogenase deficient individuals, hemolysis may occur, frequently dose-related
Use in the Elderly May be increased risk of severe adverse reactions in elderly, particularly with complicating conditions, *e g* , impaired kidney and/or liver function, concomitant use of other drugs. Severe skin reactions, generalized bone marrow suppression (see WARNINGS and ADVERSE REACTIONS) or a specific decrease in platelets (with or without purpura) are most frequently reported severe adverse reactions in elderly In those concurrently receiving certain diuretics, primarily thiazides, increased incidence of thrombocytopenia with purpura reported Make appropriate dosage adjustments for patients with impaired kidney function (see DOSAGE AND ADMINISTRATION)
Use in the Treatment of Pneumocystis Carinii *Pneumonitis in Patients with Acquired Immunodeficiency Syndrome (AIDS):* Because of unique immune dysfunction, AIDS patients may not tolerate or respond to Bactrim in same manner as non-AIDS patients Incidence of side effects, particularly rash, fever, leukopenia, with Bactrim in AIDS patients treated for *Pneumocystis carinii* pneumonitis reported to be greatly increased compared with incidence normally associated with Bactrim in non-AIDS patients.
Information for Patients Instruct patients to maintain adequate fluid intake to prevent crystalluria and stone formation.
Laboratory Tests. Perform complete blood counts frequently; if a significant reduction in the count of any formed blood element is noted, discontinue Bactrim. Perform urinalyses with careful microscopic examination and renal function tests during therapy, particularly for patients with impaired renal function.
Drug Interactions: In elderly patients concurrently receiving certain diuretics, primarily thiazides, an increased incidence of thrombocytopenia with purpura has been reported Bactrim may prolong the prothrombin time in patients who are receiving the anticoagulant warfarin Keep this in mind when Bactrim is given to patients already on anticoagulant therapy and reassess coagulation time Bactrim may inhibit the hepatic metabolism of phenytoin Given at a common clinical dosage, it increased the phenytoin half-life by 39% and decreased the phenytoin metabolic clearance rate by 27%. When giving these drugs concurrently, be alert for possible excessive phenytoin effect. Sulfonamides can displace methotrexate from plasma protein binding sites, thus increasing free methotrexate concentrations.
Drug/Laboratory Test Interactions. Bactrim, specifically the trimethoprim component, can interfere with a serum methotrexate assay as determined by the competitive binding protein technique (CBPA) when a bacterial dihydrofolate reductase is used as the binding protein No interference occurs if methotrexate is measured by a radioimmunoassay (RIA) The presence of trimethoprim and sulfamethoxazole may also interfere with the Jaffe alkaline picrate reaction assay for creatinine, resulting in overestimations of about 10% in the range of normal values.
Carcinogenesis, Mutagenesis, Impairment of Fertility: Carcinogenesis. Long-term studies in animals to evaluate carcinogenic potential not conducted with Bactrim. *Mutagenesis.* Bacterial mutagenic studies not performed with sulfamethoxazole and trimethoprim in combination. Trimethoprim demonstrated to be nonmutagenic in the Ames assay No chromosomal damage observed in human leukocytes *in vitro* with sulfamethoxazole and trimethoprim alone or in combination; concentrations used exceeded blood levels of these compounds following therapy with Bactrim Observations of leukocytes obtained from patients treated with Bactrim revealed no chromosomal abnormalities *Impairment of Fertility* No adverse effects on fertility or general reproductive performance observed in rats given oral dosages as high as 70 mg/kg/day trimethoprim plus 350 mg/kg/day sulfamethoxazole
Pregnancy Teratogenic Effects: Pregnancy Category C Trimethoprim and sulfamethoxazole may interfere with folic acid metabolism; use during pregnancy only if potential benefit justifies potential risk to fetus.
Nonteratogenic Effects: See CONTRAINDICATIONS section.
Nursing Mothers See CONTRAINDICATIONS section
Pediatric Use Not recommended for infants under two months (see INDICATIONS and CONTRAINDICATIONS sections)
ADVERSE REACTIONS: Most common are gastrointestinal disturbances (nausea, vomiting, anorexia) and allergic skin reactions (such as rash and urticaria) **FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS (SEE WARNINGS SECTION).**
Hematologic. Agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, neutropenia, hemolytic anemia, megaloblastic anemia, hypoprothrombinemia, methemoglobinemia, eosinophilia *Allergic Reactions:* Stevens-Johnson syndrome, toxic epidermal necrolysis, anaphylaxis, allergic myocarditis, erythema multiforme, exfoliative dermatitis, angioedema, drug fever, chills, Henoch-Schoenlein purpura, serum sickness-like syndrome, generalized allergic reactions, generalized skin eruptions, photosensitivity, conjunctival and scleral injection, pruritus, urticaria and rash Periarteritis nodosa and systemic lupus erythematosus have been reported *Gastrointestinal:* Hepatitis (including cholestatic jaundice and hepatic necrosis), elevation of serum transaminase and bilirubin, pseudomembranous enterocolitis, pancreatitis, stomatitis, glossitis, nausea, emesis, abdominal pain, diarrhea, anorexia *Genitourinary:* Renal failure, interstitial nephritis, BUN and serum creatinine elevation, toxic nephrosis with oliguria and anuria, crystalluria. *Neurologic.* Aseptic meningitis, convulsions, peripheral neuritis, ataxia, vertigo, tinnitus, headache. *Psychiatric.* Hallucinations, depression, apathy, nervousness *Endocrine:* Sulfonamides bear certain chemical similarities to some goitrogens, diuretics (acetazolamide and the thiazides) and oral hypoglycemic agents; cross-sensitivity may exist. Diuresis and hypoglycemia have occurred rarely in patients receiving sulfonamides *Musculoskeletal* Arthralgia, myalgia. *Miscellaneous:* Weakness, fatigue, insomnia
DOSAGE AND ADMINISTRATION: Not recommended for use in infants less than two months of age.
URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN: *Usual adult dosage* for urinary tract infections is one DS tablet, two tablets or four teaspoonfuls (20 ml) *b.i.d.* for 10 to 14 days. Use identical daily dosage for 5 days for shigellosis *Recommended dosage for children* with urinary tract infections or acute otitis media is 8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses every 12 hours for 10 days. Use identical daily dosage for 5 days for shigellosis. *Renal Impaired* Creatinine clearance above 30 ml/min, give usual dosage; 15-30 ml/min, give one-half the usual regimen; below 15 ml/min, use not recommended.
ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS: Usual adult dosage is one DS tablet, two tablets or four teasp (20 ml) *b.i.d* for 14 days.
PNEUMOCYSTIS CARINII PNEUMONITIS: Recommended dosage is 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days See complete product information for suggested children's dosage table
HOW SUPPLIED: *DS (double strength) Tablets* (160 mg trimethoprim and 800 mg sulfamethoxazole)—bottles of 100, 250 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 20. *Tablets* (80 mg trimethoprim and 400 mg sulfamethoxazole)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40 *Pediatric Suspension* (40 mg trimethoprim and 200 mg sulfamethoxazole per teasp.)—bottles of 100 ml and 16 oz (1 pint) *Suspension* (40 mg trimethoprim and 200 mg sulfamethoxazole per teasp)—bottles of 16 oz (1 pint)
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MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

FEBRUARY 1988

AROUND THE STATE

MANHATTAN

Feb 13. **Contact B & A Scan Ultrasonography for the Clinician.** Manhattan Eye, Ear & Throat Hospital. Contact: Kenn Kostuk, Manhattan Eye, Ear and Throat Hospital, 210 East 64th St, New York, NY 10021. Tel: (212) 605-3753.

AROUND THE NATION

CALIFORNIA

Feb 4–6. **Third International Conference on Monoclonal Antibody Immunoconjugates for Cancer.** 19 Cat 1 Credits. Hotel Inter-Continental, San Diego. *Feb 11–13.* **Second Annual UCSD-Sharp Memorial Hospital International Cardiac Symposium: Clinical Strategies in Complex Valvular Heart Disease.** 16 Cat 1 Credits. U.S. Grant Hotel, San Diego. *Feb 25–27.* **Second International Conference on Intracavitary Chemotherapy.** 18 Cat 1 Credits. U.S. Grant Hotel, San Diego. Contact: Office of Continuing Medical Education, M-017, UC San Diego School of Medicine, La Jolla, CA 92092. Tel: (619) 534-3940.

Feb 10–12. **21st Annual Recent Advances in Neurology.** 15½ Cat 1 Credits. Fairmont Hotel, San Francisco. *Feb 11–13.* **Comprehensive Care of the AIDS Patient: A Workshop.** San Francisco. Contact: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Feb 13–15 and 19–21. **New Precepts and Concepts in Dermatopathology.** Palm Springs. Contact: Charles Stef-

(*continued on p 40A*)

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Before prescribing, please consult Brief Prescribing Information on next page.

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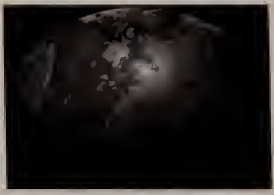
- ☐ **It's comparable to 10 mEq**—in low-dosage supplementation[3]†
- ☐ **It's economical**—less expensive than all other leading KCl slow-release supplements on a per tablet cost to the patient[1]



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For patients who can't or won't tolerate liquid KCl.

*The most common adverse reactions to potassium salts are gastrointestinal side effects.

†Pooled mean serum potassium following oral administration of 30 mEq K-Tab compared to 24 mEq Slow-K in diuretic-treated hypertensives (n = 20) over 8 weeks.

C I B A

References: 1. Data on file, CIBA Pharmaceutical Company. **2.** Skoutakis VA, Acchiardo SR, Wojciechowski NJ, et al: Liquid and solid potassium chloride: Bioavailability and safety. *Pharmacotherapy* 1980;4(6):392-397. **3.** Skoutakis VA, Carter CA, Acchiardo SR: Therapeutic assessment of Slow-K and K-Tab potassium chloride formulations in hypertensive patients treated with thiazide diuretics. *Drug Intell Clin Pharm* 1987;21:436-440.

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BRIEF SUMMARY (FOR FULL PRESCRIBING INFORMATION SEE PACKAGE INSERT)

INDICATIONS AND USAGE

BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS.

1. For therapeutic use in patients with hypokalemia with or without metabolic alkalosis; in digitalis intoxication and in patients with hypokalemic familial periodic paralysis.
2. For prevention of potassium depletion when the dietary intake of potassium is inadequate in the following conditions: patients receiving digitalis and diuretics for congestive heart failure; hepatic cirrhosis with ascites; states of aldosterone excess with normal renal function; potassium-losing nephropathy; and certain diarrheal states.
3. The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases supplementation with potassium salts may be indicated.

CONTRAINDICATIONS

Potassium supplements are contraindicated in patients with hyperkalemia, since a further increase in serum potassium concentration in such patients can produce cardiac arrest. Hyperkalemia may complicate any of the following conditions: chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e.g., spironolactone, triamterene) (see **OVERDOSAGE**).

All solid dosage forms of potassium supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract. In these instances, potassium supplementation should be with a liquid preparation. Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to an enlarged left atrium.

WARNINGS

Hyperkalemia (See **OVERDOSAGE**).

In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally. Potentially fatal hyperkalemia can develop rapidly and be asymptomatic.

The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment.

Interaction With Potassium-Sparing Diuretics

Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e.g., spironolactone or triamterene), since the simultaneous administration of these agents can produce severe hyperkalemia.

Gastrointestinal Lesions

Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths. These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage, or perforation. Slow-K is a wax-matrix tablet formulated to provide a controlled rate of release of potassium chloride and thus to minimize the possibility of a high local concentration of potassium ion near the bowel wall. While the reported frequency of small-bowel lesions is much less with wax-matrix tablets (less than one per 100,000 patient-years) than with enteric-coated potassium chloride tablets (40-50 per 100,000 patient-years) cases associated with wax-matrix tablets have been reported both in foreign countries and in the United States. In addition, perhaps because the wax-matrix preparations are not enteric-coated and release potassium in the stomach, there have been reports of upper gastrointestinal bleeding associated with these products. The total number of gastrointestinal lesions remains approximately one per 100,000 patient-years. Slow-K should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs.

Metabolic Acidosis

Hypokalemia in patients with metabolic acidosis should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, or potassium acetate.

PRECAUTIONS

General:

The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis *per* se can produce hypokalemia in the absence of a deficit in total body potassium, while acute acidosis *per* se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium.

Information for Patients

Physicians should consider reminding the patient of the following:

To take each dose without crushing, chewing, or sucking the tablets.

To take this medicine only as directed. This is especially important if the patient is also taking both diuretics and digitalis preparations.

To check with the physician if there is trouble swallowing tablets or if the tablets seem to stick in the throat.

To check with the doctor at once if tarry stools or other evidence of gastrointestinal bleeding is noticed.

Laboratory Tests

Regular serum potassium determinations are recommended. In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis.

Drug Interactions

Potassium-sparing diuretics: see WARNINGS

Carcinogenesis, Mutagenesis, Impairment of Fertility

Long-term carcinogenicity studies in animals have not been performed.

Pregnancy Category C

Animal reproduction studies have not been conducted with Slow-K. It is also not known whether Slow-K can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Slow-K should be given to a pregnant woman only if clearly needed.

Nursing Mothers

The normal potassium ion content of human milk is about 13 mEq/L. It is not known if Slow-K has an effect on this content. Caution should be exercised when Slow-K is administered to a nursing woman.

Pediatric Use

Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

One of the most severe adverse effects is hyperkalemia (see **CONTRAINDICATIONS, WARNINGS,** and **OVERDOSAGE**). There also have been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see **CONTRAINDICATIONS** and **WARNINGS**); other factors known to be associated with such conditions were present in many of these patients.

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea. These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose.

Skin rash has been reported rarely.

OVERDOSAGE

The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia. However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see **CONTRAINDICATIONS** and **WARNINGS**). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration (6.5-8.0 mEq/L) and characteristic electrocardiographic changes (peaking of **T** waves, loss of **P** wave, depression of **S-T** segment, and prolongation of the Q-T interval). Late manifestations include muscle paralysis and cardiovascular collapse from cardiac arrest (9-12 mEq/L).

Treatment measures for hyperkalemia include the following: (1) elimination of foods and medications containing potassium and of potassium-sparing diuretics; (2) intravenous administration of 300-500 ml/hr of 10% dextrose solution containing 10-20 units of insulin per 1,000 ml; (3) correction of acidosis, if present, with intravenous sodium bicarbonate; (4) use of exchange resins, hemodialysis, or peritoneal dialysis.

In treating hyperkalemia in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity.

DOSAGE AND ADMINISTRATION

The usual dietary intake of potassium by the average adult is 40-80 mEq per day. Potassium depletion sufficient to cause hypokalemia usually requires the loss of 200 or more mEq of potassium from the total body store. Dosage must be adjusted to the individual needs of each patient but is typically in the range of 20 mEq per day for the prevention of hypokalemia to 40-100 mEq or more per day for the treatment of potassium depletion. Large numbers of tablets should be given in divided doses.

Note: Slow-K slow-release tablets must be swallowed whole and never crushed, chewed, or sucked.

HOW SUPPLIED

Tablets—600 mg of potassium chloride (equivalent to 8 mEq) round, buff colored, sugar-coated (imprinted Slow-K)

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Dispense in tight, light-resistant container (USP).

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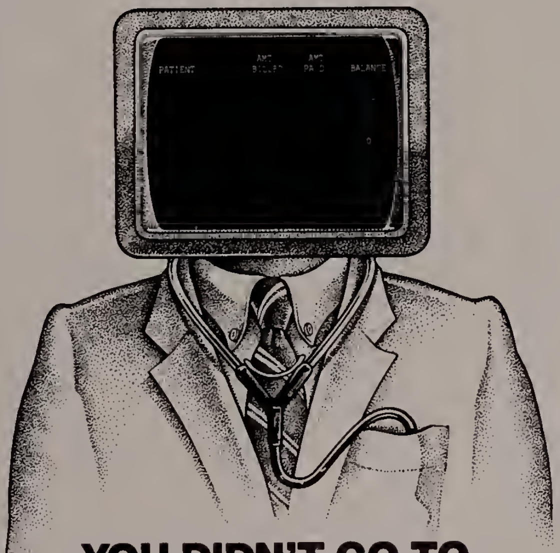

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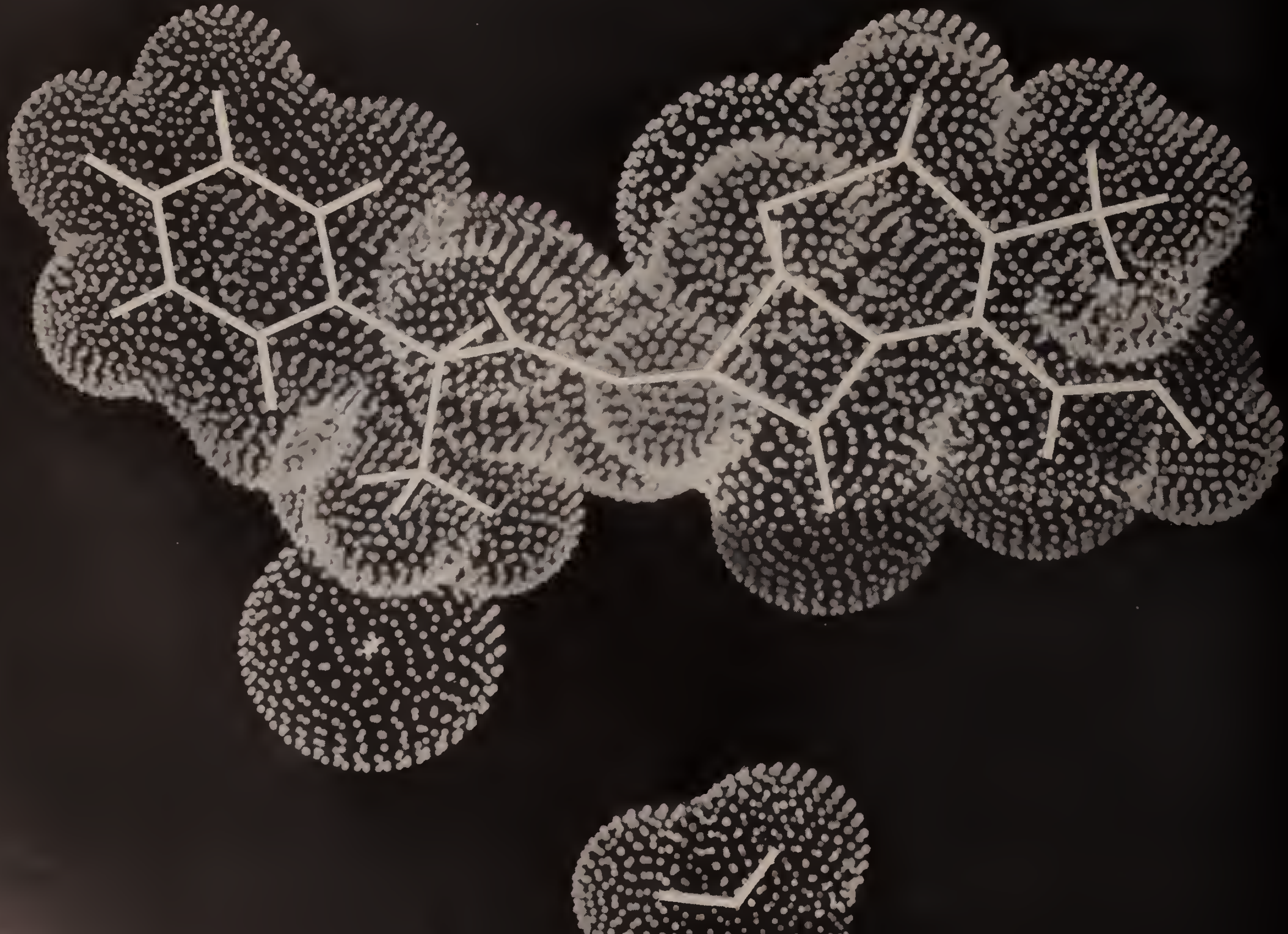

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Keftab is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-sensitive patients.

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*Due to susceptible strains of *Staphylococcus aureus* and/or β-hemolytic streptococci.
†Due to susceptible strains of *Escherichia coli*, *Proteus mirabilis*, and *Klebsiella* sp.
‡Due to susceptible strains of group A β-hemolytic streptococci.

KEFTAB™
(cephalexin hydrochloride monohydrate)

Summary: Consult the package literature for prescribing information.

Indications and Usage:

Respiratory tract infections caused by susceptible strains of *Streptococcus pneumoniae* and group A β-hemolytic streptococci.

Skin and skin structure infections caused by susceptible strains of *Staphylococcus aureus* and/or β-hemolytic streptococci.

Bone infections caused by susceptible strains of *S aureus* and/or *Proteus mirabilis.*

Genitourinary tract infections, including acute prostatitis, caused by susceptible strains of *Escherichia coli, P mirabilis,* and *Klebsiella* sp.

Contraindication: Known allergy to cephalosporins.

Warnings: KEFTAB SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:

- Discontinue Keftab in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Keftab should be administered cautiously in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy and lactation. Cephalexin is excreted in mother's milk. Exercise caution in prescribing Keftab for these patients.
- Safety and effectiveness in children have not been established.

Adverse Reactions:

- *Gastrointestinal,* including diarrhea and, rarely, nausea and vomiting. Transient hepatitis and cholestatic jaundice have been reported rarely.
- *Hypersensitivity* in the form of rash, urticaria, angioedema, and, rarely, erythema multiforme, Stevens-Johnson syndrome, or toxic epidermal necrolysis.
- *Anaphylaxis* has been reported.
- *Other reactions* have included genital/anal pruritus, genital moniliasis, vaginitis/vaginal discharge, dizziness, fatigue, headache, eosinophilia, neutropenia, and thrombocytopenia; reversible interstitial nephritis has been reported rarely.
- Cephalosporins have been implicated in triggering seizures, particularly in patients with renal impairment.
- *Abnormalities in laboratory test results* included slight elevations in aspartate aminotransferase (AST, SGOT) and alanine aminotransferase (ALT, SGPT). False-positive reactions for glucose in the urine may occur with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

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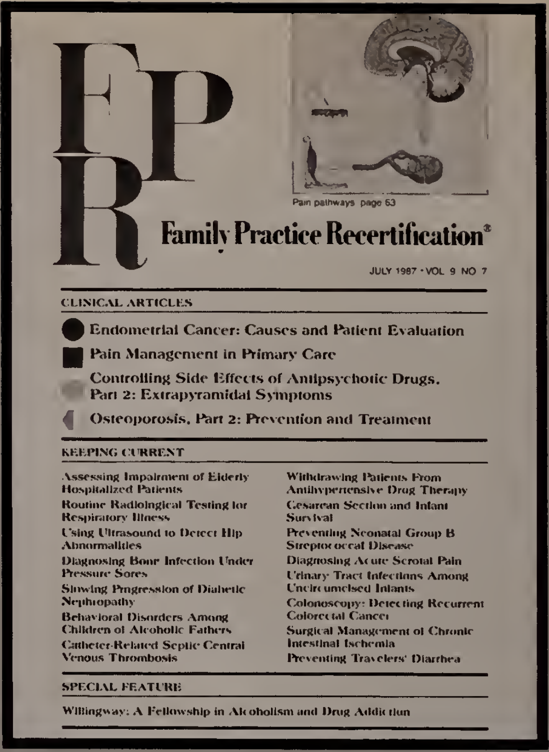

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COMMENTARIES

Preventive medicine: A new challenge for medical education

Benjamin Franklin noted long ago in *Poor Richard's Almanac* that "an ounce of prevention is worth a pound of cure." Americans respect the wisdom of this founding father, but have not taken it to heart with regard to their own health care. Medical research has clearly shown that smoking causes cancer and heart disease, invading the lives of practitioners, coworkers, and nonsmoking family members.[1,2] Smoking plus the other modifiable risk factors of hypertension, hypercholesterolemia, and sedentary life styles are directly linked to the nation's number one cause of morbidity and mortality: cardiovascular disease. The evidence is clear and compelling. Despite this, the overwhelming portion of our billions of medical dollars ($425 billion in 1985) is spent on treating preventable disease, or "underwriting medical failure."[3] At the same time, health care cost containment has emphasized limitation of medical services rather than bolstering of health promotion efforts. Improving the balance between preventive and curative approaches to health care will require fundamental changes at many levels.

Physicians have a unique opportunity of affecting health behavior on an individual patient level as well as providing leadership in health promotion for the medical care system. As a result, progress on preventive issues requires physician support. Too often, however, physicians have not fully embraced or encouraged the concept of preventive medicine.[4,5] They recognize the value of risk factor modifications, yet rarely incorporate them into their routine professional lives. Physicians, whether in private practice or academia, should do more than accept the value of risk factor modification. They must promote it in the public consciousness by encouraging and advertising health awareness beyond the examination room.

What keeps physicians from promoting wellness? It is certainly more than a collective, subliminal worry that they would all be "out of a job" should everyone stay well ("an apple a day keeps the doctor away"?). Paul A. Nutting, MD, of the Office of Primary Care Studies, US Public Health Service, has addressed the problems of health promotion and primary medical care.[5] He lists five characteristics of current medical practice that limit physician involvement in applying preventive medicine measures. These include physician attitudes and beliefs, the structure of the medical care encounter, the state of the art of health promotion, physician knowledge and skills, and the economic structure of medical care. Three of these deficiencies are related to inadequate physician understanding of preventive medicine. Although physicians have become increasingly aware of the risks of certain behaviors, they are insecure with the mechanics of risk assessment and uncomfortable with active intervention in patient behavior.

Part of the problem is that prevention does not fit well with the classic, idealized role of physicians as "healers." Curative medicine provides the drama of diagnostic intrigue and therapeutic action, while prevention is seen as passive and unimaginative. This dichotomy has existed since classic Grecian times, when the goddess of health, Hygeia, vied for attention with the god of healing, Aesculapius. Medical historians have carefully traced the roots of the disease-oriented, curative philosophy in the development of western medicine. Aesculapius, being the more exciting campaigner, has won the day. These conceptual problems are compounded by the economic structure of medical care, which does not encourage preventive interventions. Health providers are compensated far more for invasive procedures and curative treatments than for preventive interventions. In this value system, attention to prevention becomes an economic liability. Simply stated, physicians are reluctant to promote wellness because they either do not understand it or find it financially unrewarding. These attitudes can only change in part through a process of education, and at this point the infrastructure for teaching preventive medicine is insufficient.

In many ways medicine has done an inadequate job of self-education regarding the alternative, preventive approach. Few physicians are familiar with the objective advances made in understanding health-related risk factors and population behavior. Natural suspicions regarding megavitamins, copper bracelets, eye-of-newt potions, and health fads have blurred the concept of health promotion. A clearer understanding will require better education of practicing physicians, residents, and the insurance industry. To accomplish this educators will need to distill and disseminate the advances in preventive medicine by including them in the curricula of medical schools, residencies, and continuing medical education updates. Preventive medicine represents a new challenge for medical education. Meeting this challenge will require changes in physician education as well as shifts in the economic structure of medical care.

Medical school provides an important initial opportunity for this process. Professional attitudes can be strongly influenced by undergraduate curricular content. A survey in 1980[7] indicated that 99 of 125 accredited medical schools had required courses in preventive and community medicine. Most offered these topics in the first two years and a few included courses in both the preclinical and clinical years. Investigators from the University of Washington School of Medicine have examined these curricular influences on preventive care attitudes.[8] Awareness of the importance of preventive medicine was high upon entry to medical school and remained stable. Preventive medicine courses personalized this awareness by improving the levels of confidence in physicians' ability to provide preventive health care. However, good attitudes can be difficult to sustain without reinforcement. Many of the concepts gained in medical school are reshaped and tempered by the realities of clinical medicine. The initial years of postgraduate training and practice are critical in this process.

Unfortunately, postgraduate residencies may be the weakest links in teaching preventive medicine. The deficiencies of high-tech, acute care residencies have become increasingly apparent as medicine moves from the inpatient to the outpatient setting. This was clearly detailed by Schroeder et al,[9] who called for changes in the way we educate internal medicine residents. These authors listed health promotion and disease prevention as two of the many deficient areas. Awareness of risk factor screening and modification should be improved for all young physicians, not just those in primary care. Heart disease, osteoporosis, and breast and lung cancer are just as likely to occur in surgical and gynecological patients as they are in general internal and family medicine populations. Residents need the facts of risk factor assessment as well as respect for the potential of behavioral modification. They must sense from attending physicians that preventive medicine issues are relevant and worthy.

Reemphasis in physician education is essential but not sufficient. Good intentions regarding preventive medicine will become a reality only if economic incentives are established to support these measures in everyday practice. However, current reimbursement schedules are firmly rooted in the management of illness rather than in the promotion of wellness. Until third-party payors are persuaded that lowering cholesterol levels can be more cost-effective than performing bypass surgery, this system will continue. Change can occur only after the government and the insurance industry learn the value of wellness as a means of cost containment.

Health care providers are all aware of the upward spiral of medical costs caused by explosive technological advances, high public expectations, and liability issues. Government and industry, accustomed to the "bottom line" approach, have sought to control costs by placing limitations on the supply of medical services. Prospective payment for hospitals and insurance companies' limitations on access to consultants and emergency rooms are two examples. Other forms of containment by limitation are calls for restrictions on the number and type of residents based on the assumption that fewer physicians means fewer costs.[10] Physicians bristle at the external constraints and worry about the reduction in quality medical services. And yet they are all concerned by the increasing costs of medical care to society.

In the current setting, isn't it reasonable to consider that the merits of preventing illness are as worthwhile as those of restricting services? Doesn't it make sense that even small accomplishments in the prevention of illness on a national basis would mean large savings given the high cost of treating illness ("a penny saved is a penny earned!")? National foundations have established valuable and important programs regarding risk factor modification. These efforts have raised our awareness of preventive issues but have had little or no impact on the reimbursement system. One of the problems of accepting broad-based cost containment through preventive medicine is the long delay before benefits are apparent. As a result, few prospective, multicentered studies have been performed to link behavior modifications with cost savings. However, consider the impact of reducing just one risk factor, cigarette smoking. Smoking-related health care costs for 1985 were estimated at $20 billion (range, $12–35 billion).[11] Two-thirds of the costs among people over 65 are borne by government programs. Add to this the productivity loss of over 300,000 smoking-related premature deaths per year (at an average of 15 years before the life expectancy) as well as costs for higher insurance and sick leave pay. These costs certainly dwarf the current expense of smoking cessation programs. If we spent one-tenth of this amount on smoking prevention, how much would we save in human and financial resources?

The concept of preventive cost containment makes sense and is worthy of the required investment. To accomplish it, prospective studies in preventive health must be supported. Physicians should be compensated for programs developed in the promotion of good health. Funds should be made available to help physicians teach themselves preventive medicine. This will require important changes in the way society pays for medical care. Changing these attitudes will require concerted education of physicians as well as of third party payors.

Preventive medicine and the promotion of wellness are not panaceas. Illness will continue to afflict Americans even after they have all stopped smoking, entered triathlons, and developed fun-loving, mellow personalities. However, these measures are potential resources in the control of illness which are underutilized and frequently ignored. Only when physicians completely accept and promote wellness concepts will the benefits be fully realized. We all must meet the challenge to learn a new approach.

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Encouraging physician interest in preventive cardiology

Physician interest and behavior determine the course of most medical care. How thoroughly a patient is examined, which tests are performed, and how treatments are initiated have all traditionally been determined by the physician who is in charge of the patient's care. Until recently, the physician made all the decisions, with little or no input from other sources. However, dramatic changes in medical care are now underway, and in the future there will be much greater involvement of patients and their families, third party payors, state and local health departments, and the federal government. Many aspects of physician behavior are currently under scrutiny. One area that has heretofore received inadequate attention is the approach to the practice of preventive medicine. The prevention of disease states should represent the highest ideal for the practitioner, yet all too often such efforts are merely given lip service. An examination of physician behavior in the area of modification of risk factors would provide important insights into how widely preventive medicine is practiced.

The reality is that most physicians were trained to deliver curative medicine, rather than preventive medicine. In my training program, establishing an obscure diagnosis underlying the patient's esoteric signs and symptoms was the Holy Grail we sought. Once this was achieved, vigorous therapy aimed at elimination of the disease was pursued with a singleminded purpose. The patient was then discharged. Any idea of follow-up or preventive care was relegated to the doctors providing ambulatory care. The "real" action was limited to the acutely ill patient on the ward. Most training programs are still crisis-oriented.

Previous early efforts at preventive medicine were primarily oriented toward the identification, prevention, and treatment of infectious diseases. What is necessary today is an invigorated program directed at the prevention, identification, and modification of cardiovascular arteriosclerotic disease, particularly in the young and middle-aged adult.

Previous studies have identified a group of risk factors whose presence can be identified long before diagnosable clinical heart disease is discovered. Physicians and even the lay public have become generally knowledgeable about these so-called risk factors, and there have been initial efforts toward their identification and modification, albeit in a piecemeal and often desultory fashion. For a variety of reasons, the emphasis is now undergoing a significant change.

For a long time the relationship between coronary artery disease and cholesterol and saturated fat was viewed as an impossibly complex one, both by the medical community and by the population at large. Physicians and the consuming public have been exposed to misleading information promulgated by certain food processors and producers who have a vested interest in maintaining and increasing the sales of foods rich in saturated fat and cholesterol. The politics of such efforts have previously been described.[1]

The Lipid Research Clinics Coronary Primary Prevention Trial[2] provided strong evidence that the modification of serum cholesterol levels by diet and drugs results in a reduction of the risk of primary endpoints such as acute myocardial infarction and cardiac death. Such lowering also reduces the risk of development of such secondary endpoints as angina pectoris, a positive stress test, or the need for saphenous vein bypass surgery. The population studied consisted of males aged 35 to 59 who were known to be free of heart disease and hypertension, and most of whom were nonsmokers. For every 1% reduction in serum cholesterol levels there was a 2% reduction in hard and soft endpoints.[2]

Yet not all physicians are in agreement as to the need to vigorously identify and treat elevated serum lipid levels.[3] A recent study of the high cost and low yield of treating elevated cholesterol levels discussed the effects of lowering the cholesterol 6.7%. Reductions of 30–35%, readily obtainable with combinations of presently available drugs, were not discussed since the use of multiple drug therapy for lipids is still in development.[4] The release of a number of hydroxy-methyl gluterase Co-A reductase inhibitors will make such major reductions readily attainable for compliant, cooperative patients.

A major educational program about the importance of elevated serum lipid levels, directed at physicians and at the public at large, is underway. Practicing physicians must become more knowledgeable about what levels of cholesterol represent moderate and high risk for the development of coronary disease. Previously, physicians have believed that therapy need not be directed at serum cholesterol levels unless they were in excess of 300 mg. Yet as physicians gradually become more conversant about serum cholesterol levels and the recommendations of the consensus panel of the NIH, such nihilistic physician behavior will change.

A previously published report documented the casual response of many physicians to hypercholesterolemia identified during hospitalization.[5] Despite their inaction, there is little doubt of the significance of cholesterol. There is evidence implicating elevated blood cholesterol levels as a major risk factor in the development of coronary arteriosclerosis.[6,7] Although there has been disagreement on the application of dietary manipulation to the population at large, there is less disagreement on the role of serum cholesterol levels and the frequency and extensiveness of coronary artery disease.[8,9] Elevated cholesterol levels appear to be related to progressive narrowing in aorto-coronary vein grafts studied after bypass surgery.[10] Longitudinal changes in cholesterol levels also provide useful predictors of ischemic disease in man.[11] Moreover, regression of arteriosclerotic lesions in man and animals has been reported after therapy directed at lowering elevated serum cholesterol levels.[12–14]

Recent publications have documented the laissez-faire attitude of many physicians when notified of their patient's elevated cholesterol levels.[15] Given the reality that physicians have not been aggressive in the diagnosis and treatment of elevated levels of serum cholesterol, and the overwhelming evidence that reduction of elevated serum cholesterol levels would reduce the risk of cardiovascular events, what steps can be taken to encourage physicians to change their behavior? The answer would clearly seem to rest with education, both of physicians and of the medical community as a whole. For example, many laboratories still persist in labeling as normal a serum cholesterol as high as 310 mg, despite evidence to the contrary. Education of the lay public is also vital. Efforts directed toward this end are already underway. Yet for the average physician in practice, this will mean a major adjustment in attitude, from one of, "Don't worry about your cholesterol, it is only a little high," to one of time-consuming involvement. This involvement of the physician in the treatment of elevated serum cholesterol levels will require the acquisition of additional skills and experience, for most physicians another time-consuming chore. Yet the efforts will result in better treatment and a higher degree of compliance by the patient as well as a better medical outlook.

The first mode of treatment for hypercholesterolemia must be dietary, and for some patients this is all that is required. Generally, physicians tend to overestimate the value and effectiveness of the dietary treatment of hypercholesterolemia. With close cooperation between physician and patient, a 10–15% reduction of serum cholesterol levels can be obtained with a low-fat, low-cholesterol, cereal-based diet.[16] Physicians must attempt to do the best job they can for and with the patient under these difficult circumstances. Exercise as well as diet, weight loss, and restriction of alcohol intake in alcohol-sensitive individuals must be stressed. The physician's efforts are made even more difficult by the reality that hypercholesterolemia is a lifelong condition, and is usually asymptomatic. Continual support of the patient is necessary for good compliance.

Barriers persist to the physician's implementation of lipid-lowering therapy as part of a program of preventive cardiology. Some of these have been described previously.[17] The major barrier is related to the physician's previous education and experience. Physicians do not generally understand or accept that their own patients will benefit from the intervention of lowering the level of serum cholesterol in the blood. The physician may not have the time, interest, or skill to help patients change their risk factors. Physicians have received ambiguous information about elevated levels of cholesterol from their old medical school textbooks, from some less-than-current laboratory normal-range lists, and from the current media drive on cholesterol education.

Equally important is the lack of compensation mechanisms for practicing good, preventive cardiology. A physician who engages in any invasive technique, or who puts his or her patients through an extensive non-invasive diagnostic workup, is much more richly rewarded by current fee schedules. The physician who takes the time to counsel patients on the importance of diet and exercise will receive far less income for the same expenditure of time and effort. Physicians must be adequately reimbursed by third party payors for supplying time-consuming preventive services.

Furthermore, if the physician does not truly believe that the intervention can change the patient's behavior or risk, there is little motivation to persist in that difficult task. This is particularly true for physicians who lack confidence in their ability to deliver preventive intervention. Many physicians choose to doubt the legitimacy of the preventive approach to patient care, rejecting it as part of their professional responsibility.

Finally, many physicians are convinced that they are too busy with their practices to provide a service that patients are not clamoring for in the first place. If the physician chooses to believe that patients do not really wish the intervention that requires behavioral change, then they can be free from the burden of doing something the patient does not seem to want. Yet if the patient does not want an operation or a cardiac catherization, how many good doctors will feel free to disregard their own medical judgment in the matter without trying to influence their patient's choice? The most recent intervention, that of urging patients to lower their cholesterol by whatever dietary, behavioral, and pharmaceutical means are necessary, will soon be the keystone of joint efforts to reduce the toll of coronary artery disease. This effort will involve many players including industry, business, nurses, other nonphysician health professionals, and a variety of medical and health-related community groups. We can hope that this will provide impetus similar to the explosion of interest and effort that followed the NIH consensus on the treatment of hypertension.

Our patients who avoid future heart attacks may not be completely aware of the benefits of our efforts on their behalf, but the true measure of a physician is both the illnesses we prevent as well as those we effectively cure.

"We are not required to complete the task at hand—Neither are we free from making a start."[18]

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Hospital-physician relationships: A changing dynamic

The turbulence that is affecting health care is also changing the relationship between physicians and hospital administrators. In the past, their interactions have swung on a pendulum between collaborative and adversarial. Now, there seems to be a definite need for collegiality to prevail. Rubright[1] cogently states, "the physician is the quintessential guest and customer, whether in private or group practice, or employed by an HMO [health maintenance organization] or similar organization. And given what physicians know and what they do, pursuing timely, thorough physician-relations programs will serve not only the current but the future needs of hospitals." With this awareness, it is incumbent on physicians to scan the environment in order to gain further insight. Areas to consider include impasse resolution, the role of physicians in hospital planning and decision making, physicians as a source of capital, and marketing management.

Several publications have been devoted to conflict resolution,[2-4] but perhaps the one with greatest relevance to this issue is the *Report of the Joint Task Force on Hospital-Medical Staff Relationships.*[5] This represents a collaborative effort of the American Hospital Association and the American Medical Association. The report recommends that concerns be promptly communicated, that the reasons underlying the concerns and their degree of importance be evaluated, that the significance of compromise and accommodation be stressed, and that third-party consultations be jointly employed as needed. It notes that the key actions are *communicate*, *compromise*, and *consult jointly*. To develop the process, Kovner and Chinn[6] advocate encouraging physician leadership in hospitals. They propose three models: fractionated, dual domain, and integrated. In the fractionated model, a few key personnel make the decisions and there is no formal planning system. In the dual domain model, there are separate decision-making roles for both physicians and administrators. There is a formal planning system but the arrangement may lead to an adversarial encounter between the two groups.

The investigators favor the integrated model, in which there is joint decision making, formal planning, and encouragement of active physician leadership. The integrated model is thought to be a slow process, however. Miscommunication and misunderstanding are observed to be problems because of the many individuals involved. It is the authors' opinion that interaction of the models with the environment, strategic decisions, and so on would often determine the outcome of the relationship. Another important variable is the informal power relationship among the players.[6]

Some institutions have made conscious decisions to involve the medical staff at the beginning of the planning effort. Clemenhagen and Champagne[7] describe such an undertaking at an acute care teaching hospital. The decision makers and information users were identified and organized. The planners worked actively, reactively, and adaptively with the decision makers and information users. The planners sought to balance the input of the decision makers while ensuring their cooperation. This approach has opened lines of communication between the medical staff and the administration. It has also increased the awareness of planning principles and promoted the exchange of information. It has been described as a time-consuming endeavor, but it has nonetheless charted the path for hospital growth. Grubbs et al[8] cite some of the barriers to physician understanding that have to be overcome if the planning process is to be successful. They include time constraints, specific perspectives, skepticism, and different levels of interest.

As administrators search for a means of financing the renovation of their institutions, Fried[9] notes that physicians represent a source of capital through tax shelter financing, limited partnerships, and joint ventures in acquiring medical equipment as possible investment vehicles. With the new tax laws, joint ventures such as medical office buildings have come under increased scrutiny but they still remain a possibility. One source[10] lists as investment winners HMOs, preferred provider organizations (PPOs), utilization review services, and others. The losers would be medical office buildings and imaging and diagnostic centers.

Hospital-physician partnerships in which costs, risks, and benefits are shared may increasingly become a means of resolving conflicts between physicians who want new equipment and hospitals that are unable or reluctant to risk new capital expenditures. In the future there may be more physician-hospital venture capital relationships based on mutual trust and shared risk and reward.[11]

The relationship between doctors and their hospitals has been affected by the changing market conditions of an aging population, an interest in wellness, and an increasing number of hospitals. The picture has been further complicated by changing payment programs and the growth of HMOs. Mac Stravic[12] suggests that hospital administrators can foster more effective communication through newsletters, through individual and collective meetings with physicians, and by accompanying physicians on rounds. He favors offering marketing assistance for the physicians as well as management assistance in billing collections.

Many executive directors have been convinced of the importance of marketing their hospitals. Sturm[13] notes that four strategies have emerged in support of this objective. They are aptly named, "The Physician First," "The Great American Consumer," "The Blend," and "The Turtle." Whether the emphasis is on the physician, the patient, or both, or the institution has taken a wait-and-see approach, marketing management has taken hold in US hospitals. There also appears to be segmentation of the market, whereby age, sex, and other patient characteristics will determine who makes the decision as to hospitalization.[14]

Briefly, then, we have surveyed some of the changes taking place in the area of hospital-physician relationships. These changes may enhance the relationship for the well being of patients and the mutual benefit of physicians and hospitals. There is an undeniable interdependency among physicians and their patients and hospitals. In truth, it can be said that we are all in this together.

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New York State emergency department physician survey

Implications for graduate medical education

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ABSTRACT. A survey conducted in late 1986 of 219 (75%) New York State hospital emergency departments sought to determine the nature of physician staffing and the volume and the acuteness of patient problems. The results are grouped by Health System Agency (HSA) areas to reflect regional variations of staffing and patient visits. Overall, only 15.5% of emergency department staffing is provided by board-certified emergency physicians in New York State emergency departments, compared with the national norm of 36%. In addition to a lack of board-certified emergency physicians, the survey identified an absolute deficiency of emergency department physician staffing throughout all regions, representing a shortage of nearly 400 physicians statewide. Average annual patient visits varied throughout the state, but acuteness was consistently high as reflected in the large percentage of patients admitted to the hospital and to intensive care units. The implications of these findings for graduate medical education in New York State are discussed.

(NY State J Med 1988; 88:7–10)

Increased attention has been focused in recent years on the qualifications and training of physicians who staff emergency departments. The Joint Commission on Accreditation of Hospitals (JCAH) now mandates that "at least one physician experienced in emergency care [be] on duty in the emergency care area." [1] The New York State Department of Health Hospital Review and Planning Council has proposed standards for emergency departments requiring that

> The emergency department staff physicians must have a license to practice medicine and: a) board certification in emergency medicine, or b) three years post graduate experience in emergency medicine, surgery, family practice, or pediatrics in addition to current certification in Advanced Cardiac Life Support (ACLS), and Advanced Trauma Life Support (ATLS) or certification by the American Board of Emergency Medicine or equivalent training and experience.[2]

The American College of Emergency Physicians has adopted the following position with respect to emergency department physician staffing:

> It is ideal for all physicians working in emergency departments to be board-certified in Emergency Medicine. Until that goal is realized, however, the following are recommended qualifications, in order of preference, for emergency physicians: 1) residency training in emergency medicine; 2) training and experience necessary to take the Emergency Medicine board certification examination; 3) residency training in a related specialty; and 4) experience in, and full-time commitment to, emergency medicine.[3]

New York State public health authorities are expected to propose new regulations regarding improved supervision of house officers in emergency departments by at least one senior attending physician trained in emergency medicine. This action comes in response to recommendations by a Manhattan grand jury investigating the death of a young woman at a New York hospital.[4] In New York City, the Emergency Medical Services Agency will require, over the next couple of years, all emergency departments that are part of the city's 911 emergency ambulance system to be staffed by fully trained attending physicians.[5] The net result of this renewed public and regulatory interest in the training and qualifications of emergency department physicians will be to increase the demand for fully trained and qualified emergency physicians.

Specialty recognition for the field of emergency medicine was achieved in 1980 with the designation of the American Board of Emergency Medicine (ABEM). The ABEM has granted certification to approximately 6,000 physicians since 1980, many of whom qualified as practice-eligible to take the certification exam. After 1988, however, only emergency medicine (EM) residency graduates will be eligible to take the board examination, limiting the number of new board-certified physicians to about 450 per year from approximately 70 training programs nationwide. Data from 1984–1985 indicate that more than 36% of all emergency physicians nationwide are now board-certified in emergency medicine.[6] More than 88% of emergency physicians are US medical school graduates, compared to 78% for all other specialties.[6]

New York State, with its 13 medical schools and associated medical centers, plays a major role in specialty train-

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ing. Although New York has about 7.8% (17.5 million people) of the nation's population, it annually graduates 17.1% (3,141) and 17.2% (1,421) of the nation's internists and surgeons, respectively.[7] Moreover, New York trains a disproportionately large number of residents in all major specialties except for emergency medicine and family practice, with 4.6% and 5.4%, respectively, of all residency positions in the United States.[7] The total number of emergency medicine residency positions in New York State is 51, with 21 new graduates annually (personal communication, Paul R. Gennis, MD, Joel R. Gersheimer, MD, Thomas G. Kwiatkowski, MD, Ralph Altman, MD, April 1987). They account for only 4.6% of the total of 453 final-year emergency medicine residents annually trained in the US (personal communication, Anne E. Crowley, PHD, March 19, 1987). Only four emergency medicine residency programs exist in medical centers in New York State: Albert Einstein College of Medicine, Long Island Jewish Medical Center, New York Medical College, and Metropolitan Hospital Center, the latter two programs consolidating during 1987. No programs exist in upstate New York. Thus, it is clear that medical centers in New York State have not developed emergency medicine training programs to the extent that they have those in the more established specialties.

Since medical centers in New York State train proportionately few emergency physicians, we chose to evaluate the nature of physician staffing and patient visits in emergency departments throughout New York State to determine the need for an expansion of emergency medicine training programs in the state.

METHODS

A telephone survey of 219 hospital emergency departments was conducted in New York State during the months of November and December 1986. An attempt was made to interview the emergency department medical director or designee in each instance. The hospitals surveyed represented 75% of the 294 nonfederal hospital emergency departments in the state. A total of 181 hospitals (62%) participated, while 38 hospitals (13%) refused to participate in the study.

The following questions were asked by a surveyor, in addition to identifying the name and title of the person interviewed:

- What is the total number of annual visits to your emergency department?
- What percentage of these total visits results in admission to the hospital?
- Of the total number of patients admitted, what percentage is admitted to a critical care unit?
- Provide the total number of full-time physicians needed to fully staff the emergency department, consolidating any part-time positions to make up full-time physician equivalents.
- What is the average number of hours worked by a full-time physician (per week or per month), excluding vacation time?
- Of the full-time physicians, how many are board-certified in emergency medicine?
- Of the full-time physicians who are not board-certified in emergency medicine, how many are board-certified in another specialty? Which specialties, if any?
- Is the department currently short-staffed? If yes, by how many full-time physician equivalents?

A data base was constructed from information obtained from the phone interviews using the Lotus 1-2-3 software system and an IBM AT computer. Information was generated regarding frequency, distribution, percentage, mean, and standard deviation of the data obtained.

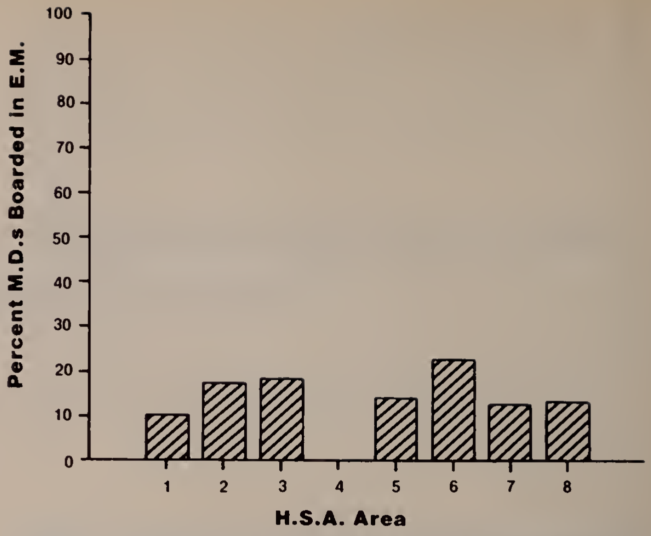

FIGURE 1. Percentage of physicians certified in emergency medicine by Health Systems Agency (HSA) area, New York State, 1986.

RESULTS

The average percentage of physician coverage provided by board-certified emergency physicians in New York State emergency departments is 15.5%. The range is from 10.5% in Health Systems Agency (HSA) region 1 (Western New York) to 22.8% in HSA region 6 (Lower Hudson Valley) (Fig 1). The geographic locations of HSA regions may be seen in Figure 2. The percentage of board-certified physicians (any board except emergency medicine) providing emergency department coverage is 43.6%. The range is from 31.4% in HSA region 1 to 56% in HSA region 8 (Nassau and Suffolk Counties, Long Island).

The average number of physician full-time equivalents (FTEs) necessary to staff hospital emergency departments in New York State is 5.91 ± SD (standard deviation) 3.43 FTE, with a range of 3.89 ± SD 1.87 FTE in HSA region 1 to 9.16 ± SD 5.58 FTE in HSA region 7. The actual number of FTEs currently staffing emer-

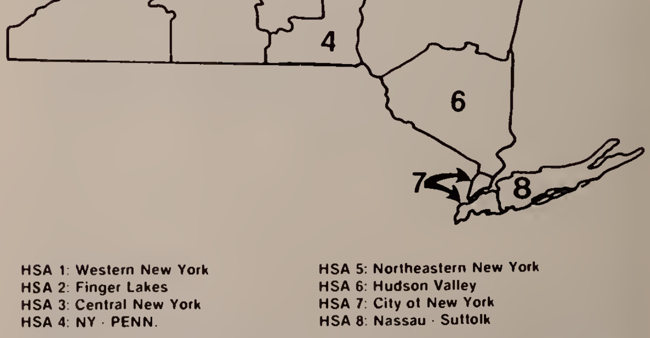

FIGURE 2. Health Systems Agency (HSA) areas In New York State.

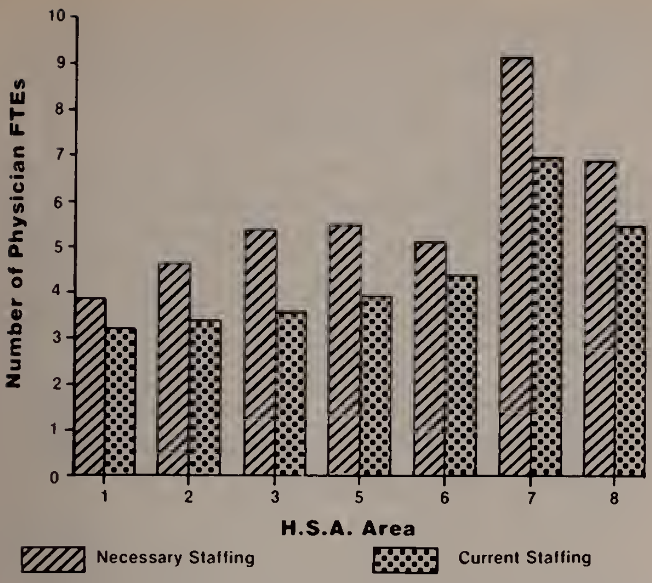

FIGURE 3. Comparison of current versus necessary emergency department physician staffing requirement by Health Systems Agency (HSA) area.

gency departments in the state is 4.60 ± SD 3.80 FTE, with a range of 3.19 ± SD 2.16 FTE in HSA region 1 to 6.98 ± SD 6.5 FTE in HSA region 7 (Fig 3).

Thus, a total of 1,352 FTE physicians are currently engaged in the practice of emergency medicine in New York State. Of this group, 15.5%, or about 210 physicians, are board-certified in emergency medicine. The total number of physicians falls 386 short of the number actually needed to fully staff emergency departments in New York State.

The average number of annual patient visits to each New York State emergency department was 27,615 ± SD 32,379. The range is from 16,143 ± SD 8,892 in HSA region 1, to 55,020 ± SD 61,261 in HSA region 7 (Fig 4).

The average percentage of patients admitted from an emergency department based on total annual visits is 18.5%, with a range of 14.7% in HSA region 5 to 24% in HSA region 8. Thus, about one in every five patients seen in an emergency department in New York State is admitted to the hospital. Of those admitted, another one in five (19%) are admitted to a critical care unit.

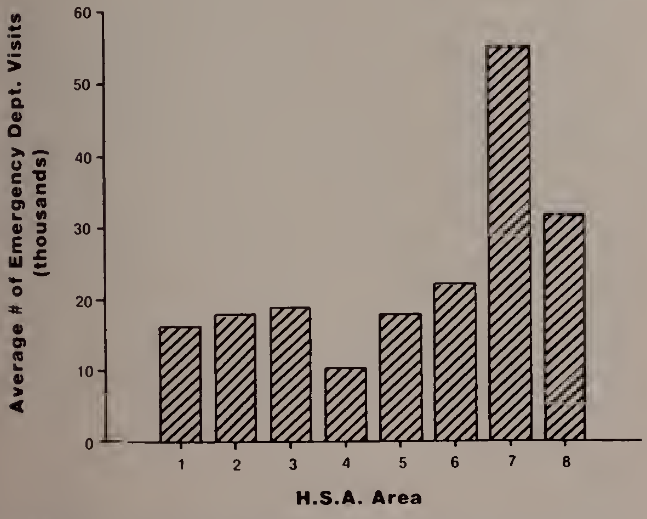

FIGURE 4. Average annual number of emergency department visits by Health Systems Agency (HSA) area, New York State, 1986.

Each FTE provides 43.59 hours ± SD 6.74 hours of emergency department coverage each week. On average, each FTE sees 138 patients per week, about 25 of whom will require admission, with five going to a critical care unit.

DISCUSSION

Hospital emergency departments provide a variety of essential services to the public on a demand basis. The staff and physicians must be capable of managing a range of illnesses and injury with efficiency and a high degree of skill. Moreover, no patient can be denied access to care in an emergency department in New York State on the basis of inability to pay. A large proportion of the health care provided to the medically indigent is obtained through emergency departments. Thus, hospital emergency departments meet an array of medical and social needs.

It is clear that the medical control and quality of an emergency department is a reflection of its nursing and physician staff. The results of our survey are troubling in this regard. Not only are emergency departments in New York State short-staffed by nearly 400 full-time physicians, but the training and qualifications of those emergency physicians currently in practice fall far short of the national norm.

The national average percentage of physician emergency department coverage provided by board-certified emergency physicians is 36%. To increase the current staffing level of 15.5% emergency medicine board-certified physicians to 36% in New York State would require the addition of 277 emergency medicine board-certified physicians. Therefore, simply to raise New York State emergency departments up to full staffing and meet national norms for qualifications would require the addition of nearly 400 physicians, about two-thirds of whom would need to be board-certified in emergency medicine.

Unfortunately, the teaching medical centers are poorly prepared to meet the demand for additional qualified emergency physicians. If the state were to rely on its own emergency medicine training programs to eliminate the emergency physician shortfall, it would take 13 years, based on 21 graduates per year, provided every residency graduate remained in New York State and there was no physician turnover, both events being unlikely. Clearly, a rapid acceleration is needed in the development of emergency medicine training programs in New York State in order to meet the standards of the regulatory agencies and the needs of the patients.

The *Report of the New York State Commission on Graduate Medical Education* has recommended the establishment of a council on graduate medical education to "provide continuing policy guidance to State policy makers regarding the composition, training, supply, and distribution of physicians in New York State." [7] It appears, therefore, that the council may be empowered to allocate the number and distribution of graduate medical education (GME) positions within any particular medical school and its affiliated hospitals (described as a consortium). New programs such as emergency medicine may achieve inadequate growth or even risk elimination if the proposals of the committee on GME are adopted.

Should the council reduce the total number of training positions, well-established programs will struggle to maintain their quota of house staff. Moreover, if the total is reduced and available positions are redistributed toward primary care, competition within any consortium will become even more intense for the allocation of subspecialty positions. Finally, emergency medicine may not qualify in the view of the council as a primary care program. Thus, as a new specialty, not well represented in teaching centers, and perceived as a nonprimary care specialty, emergency medicine training positions may become severely constrained in New York State.

Once formed, the Council on Graduate Medical Education will be in a position to address the deficiency of qualified emergency physicians in New York State. The information provided in this study should prove valuable to the council by providing a reliable data base on the need for emergency medicine graduate medical education in the state. Only through improved planning and coordination of graduate medical educational activities in emergency medicine can there be any hope of addressing the pressing training and patient care issues identified in this survey.

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Characteristics of shifts and second-year resident performance in an emergency department

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ABSTRACT. There is currently much concern, especially in New York State, about house staff work schedules in emergency departments, as they might adversely affect patient care and physician well-being. The literature, however, has scarce information about the effects of residents' work conditions and stresses on patient care performance. In this study, the author examined relationships between second-year resident performance in an emergency department, several shift work characteristics (number of patients seen, time-in-shift, and busyness of the shift), and the number of shifts worked. The study found that as busyness of the shift increased, there was a decrease in the number of history and physical examination items recorded in the medical record. As the number of hours worked increased, there was a decrease in the comprehensiveness of the physical examination. These results were found although the shifts studied were not extraordinarily long (ten to 14 hours). Most shifts did not involve overnight duty, and time off between shifts seemed adequate to prevent sleep deprivation.

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Recently, there has been rising concern about the work conditions and stresses of internship and residency, and their potentially adverse effects on patient care and physician well-being. Following a grand jury's findings regarding an emergency room patient's death,[1] the New York State Department of Health took under consideration recommendations for regulations regarding the supervision of house staff and limiting interns' and residents' shifts in emergency departments to 12 hours, separated by at least eight hours off.[2] In October 1987, the Ad Hoc Advisory Committee to New York State's Commissioner of Health, after hearing testimony from hospital leaders and medical school deans, proposed a weekly limit of 80 hours.[3]

The American Medical Association House of Delegates adopted a resolution at its June 1987 annual meeting recognizing the importance of the quality of patient care in the nation's teaching hospitals and the urgent need to address the working hours and supervision of house officers.[4] The resolution disapproved of administrative regulation or governmental legislation, acknowledging instead the ability of the medical profession to propose policies assuring the quality of patient care and graduate medical education.

While there is a modest body of research literature describing the work conditions and stresses of internship and residency,[5-7] there is remarkably little literature examining the effects on house staff performance. Studies of the effects of work conditions on house staff performance primarily focus on the effects of sleep deprivation and fatigue on performance with various cognitive, motor, or simulation tasks.[5,8,9] What is clearly scarce are studies of the

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effects on actual patient care, not only of sleep deprivation and fatigue, but of other work characteristics such as workload, which also has been identified as a major source of stress for physicians-in-training.[10] This scarcity is not surprising, since the complexity and variability of a physician's work are significant barriers to work measurement studies. A teaching hospital emergency department (ED) in particular encounters variable patient loads and medical problems that vary in type and severity. There is a crucial need for empirical studies of those factors that can affect the quality of both patient care and the training and educational experiences of house staff.

Because of the need for empirical studies, the author reexamined a data set from the Johns Hopkins Hospital emergency department which included second-year resident performance data. Although the data is from the years 1976–1977, the hospital had already reduced the time duration of medical resident shifts to a minimum of ten and a maximum of 14 hours, which is comparable to the recommended times currently under consideration in New York State. Considering the urban, inner-city setting of a major hospital and the prestigious nature of the training program, the findings can still be instructive today.

In this study, the relationships between resident performance in the ED for selected patients and several shift work characteristics were examined. This included the number of patients seen prior to a selected patient, an estimate of the duration of a shift already worked when a selected patient was seen, and an indicator of a shift's busyness. Also examined was the relationship between the number of shifts worked and performance, to assess whether any decrements in performance occurred over time with the type of shift schedule.

A previously published analysis of data used in this report found many negative but small correlations between resident performance, patient rate of entry to the ED, and total number of patients seen during a shift.[11] But unlike the previous report, the analysis in this paper takes into consideration individual differences in performance among residents and patient sociodemographic and economic characteristics. It also examines time-in-shift, a measure of shift busyness, and the number of shifts worked. Further, it is possible that the previously reported findings of negative correlations were misinterpreted as resident performance decrements in response to increasing work demands rather than the actual result of resident intentions, unrelated to a shift's work demands. Residents could decrease performance per patient, thus leading to their ability to see more patients. The analysis in this paper accounts for such a possibility, although in the ED setting there were no formal productivity requirements which might have influenced residents' intentions, and patients still waiting to be seen at the end of a resident's shift would be seen by another resident. Finally, the analyses take into account the potential effects of circadian rhythm on performance.

METHODS

Assessment of the effects of shift characteristics on resident performance could be approached by using the various tasks described in the literature as well as patient simulators (ie, persons trained to provide standardized patient presentations).[12] Such tasks and patient simulators could be presented to residents in a planned schedule under varying work conditions. But costs and practicality limit the number of residents and shifts that could be assessed with patient simulators, and the results from special tests are difficult to generalize to patient care. The research approach of this study was akin to using patient simulators, except that performance was measured for real patients who had the same medical problems. These patients are referred to as "test" patients. By selecting frequently occurring medical problems, test patients can be expected to have presented to ED residents at different times within many shifts, assuring that residents saw these patients under different working conditions. Variables such as patient sociodemographic and economic characteristics are included in the analyses to account for other differences among test patients, differences which would have been minimized with patient simulators. By using medical records, one can generate adequate sample sizes more efficiently.

The data set was derived from the medical records of ten second-year residents randomly sampled from the 38 second-year residents who had a month-long rotation on the medicine service of the ED during the year July 1, 1976–June 30, 1977. Residents were assigned to shifts in the ED which were set for 12 to 14 hours on weekdays and ten to 14 hours on weekends. Assignment was planned so that ED staffing levels corresponded to heaviest patient usage of the ED in the afternoons and evenings. The sampled residents had worked from 20 to 25 shifts during their rotation in the ED. Eight of the ten residents had each spent from five to seven overnight shifts, which usually were scheduled on consecutive days. One resident had no overnight shifts, and one had only overnight shifts.

The shift work characteristics studied were number of patients seen (PTSEEN), time-in-shift (TMINSHFT), and shift busyness (TIME). PTSEEN is the number of patients a resident had seen during a shift prior to a test patient. TMINSHFT is an estimate of the proportion of a shift already worked at the time a test patient was seen. TMINSHFT was calculated by dividing PTSEEN plus one by the total number of patients seen during a shift (TOTALPTS). An estimate was necessary, because the medical records did not include the actual time a patient was seen.

The data set included information on the times of entry to the ED of the first and last patients seen by a resident during his or her assigned shift. The time duration (TIME) between these two times of entry was used as an indicator of the busyness of a shift. All else being equal (such as the total number of patients seen during a shift), the smaller the value of TIME the busier the shift. Shift busyness, then, is inversely correlated with TIME. When larger TIME values occur, it is more likely that the last patient seen by a resident was admitted more recently, indicating shorter waiting lines and hence less busy shifts and less need for residents to hurry or decrease their work per patient to more quickly attend to waiting patients or in response to a hectic environment.

Performance data were collected for the initial visits of patients with a single diagnosis of either upper respiratory infection or viral syndrome who were seen only by a second-year resident. As noted, these patients are referred to as "test" patients. Upper respiratory infection and viral syndrome are typically nonsevere, self-limiting medical problems.

The performance measures included the number of history items recorded in the medical record (HISTORY), number of physical examination items recorded (PHYSICAL), comprehensiveness of the physical examination (COMPREHN, ie, the number of body systems mentioned), and a dichotomous variable TESTS indicating if no laboratory and radiologic tests (TESTS = 0) or one or more tests (TESTS = 1) were ordered. History and physical examination items included abnormal symptoms and signs as well as those described as absent or within normal limits.

The analysis was based on multivariate regression, in which each of the shift work characteristics was related separately to each of the performance variables in the form of partial correlation coefficients (r_p). Adjustments were made for patient age, type of diagnosis (DISEASE, ie, upper respiratory infection or viral syndrome), time of day patients were seen (TIMEODAY), total number of patients seen by a resident during a shift (TOTALPTS), and individual differences among the residents. The squares of the shift measures also were entered into regression equations, recognizing the parabolic relationship between work demands and performance postulated by activation theory.[13] Simple correlations are presented for comparative purposes. In the analyses of number of shifts worked, the partial correlation coefficients are adjusted only for individual differences among residents and differences in the type of diagnosis (none of the other variables played a role in the relationships). Data analyses were conducted using the statistical package SPSS/PC+ for the IBM Personal Computer.

Patient characteristics other than age were examined and included sex, race, marital status, and method of payment. These patient characteristics were not retained in the multivariate regression findings presented in this report, because preliminary regressions found that they were not statistically significant at $p \leq 0.10$ in any equation, and their exclusion did not alter the findings.

DISEASE was included to adjust for performance differences between upper respiratory infection and viral syndrome. TOTALPTS adjusts for the possibility discussed above that the resident's intended performance determined the total number of patients seen during a shift. TIMEODAY was a dichotomous variable included to adjust for possible circadian rhythm effects on resident performance. TIMEODAY was measured as 10 AM to 10 PM and 10 PM to 10 AM, corresponding approximately to periods of high and low arousal, respectively.[14] Nine dummy variables representing the ten residents were entered in the regressions to account for individual differences in practice styles among residents.

Repeat data collection on a random sample of 54 patient records found test-retest correlations for the performance variables all greater than 0.90. Billing tape data was used to check the validity of the TESTS measure. Access to billing tape data was by resident identifier. Two of the ten residents were randomly selected for verifying the TESTS data. For these residents there was agreement between the TESTS measure obtained from billing tape records and from patient records for 95% of the cases.

RESULTS

The ten residents worked a total of 228 shifts in the ED, during 141 of which they saw at least one test patient. There were 289 test patients, 145 with the diagnosis of upper respiratory infection and 144 with viral syndrome. Patients were mostly young (mean age, 29.2 years), female (85%), black (87%), not living with a spouse (76%; includes single, separated, divorced, and widowed), and not paying for services with private insurance (72%; includes Medicaid, Medicare, and self-pay patients).

Resident performance data are shown in Table I. Overall, the mean number of history items was 8.0 (SD, standard deviation = 4.7), physical examination items, 13.6 (SD = 10.4), and body systems included in the physical examination, 5.6 (SD = 2.8). The number of patients with no tests was 180 (63%). There was ample variation among residents, indicating differences in practice styles and the necessity to account for this in the analyses.

TIME ranged from 1.5 to 14.0 hours. With shifts scheduled from ten to 14 hours, small TIME values were unexpected and signaled the possibility that some shifts were unusual for unknown reasons. Hence, shifts with small TIME values, identified as less than 4.0 hours, were excluded from the analyses. This reduced the number of cases from 289 to 252. After these exclusions, TIME ranged from 4.1 to 14.0 hours with a mean of 9.1 (SD = 2.2); PTSEEN ranged from 0 to 31, with a mean of 6.7 (SD = 4.8); and TMINSHFT from 0.06 to 1.00, with a mean of 0.57 (SD = 0.28).

Table II gives the simple and partial correlation coefficients of the shift work characteristics with the performance measures. For the partial correlation coefficients, with the exception of TESTS, the results were in the direction indicating that performance decreased as the number of patients seen, time-in shift, and shift busyness (by definition the negative of TIME) increased. However, the partial correlations were small and most were not statistically significant ($p < 0.05$). Statistical significance does support the conclusion that physical examination comprehensiveness decreased as time in a shift passed ($r_p = -0.13$), and the busier a shift (the negative of TIME) the fewer history ($r_p = 0.21$ with TIME and $r_p = 0.22$ with TIME squared) and physical examination items ($r_p = 0.13$) were recorded.

TABLE I. The Number of Test Patients, Mean (and SD) of HISTORY, PHYSICAL, and COMPREHN, and Number of Patients with 0 and 1 TESTS, by Resident

Resident	Number of Test Patients	HISTORY Mean (SD)	PHYSICAL Mean (SD)	COMPREHN Mean (SD)	TESTS* 0	TESTS* 1
1	30	9.4 (3.6)	13.0 (10.6)	5.0 (2.3)	15	15
2	42	4.3 (2.1)	4.2 (2.4)	2.2 (1.0)	39	3
3	25	14.4 (4.3)	34.9 (13.8)	11.6 (1.5)	14	10
4	29	9.0 (3.1)	16.9 (9.7)	6.7 (1.7)	7	22
5	17	12.3 (3.3)	18.2 (3.7)	6.6 (1.2)	9	8
6	28	5.4 (2.1)	11.5 (7.3)	5.4 (1.8)	16	12
7	25	9.8 (3.6)	12.1 (7.3)	4.9 (1.4)	10	15
8	49	3.3 (2.2)	12.5 (3.9)	5.9 (2.1)	47	2
9	28	12.4 (2.9)	10.1 (2.5)	4.8 (0.7)	10	18
10	16	7.6 (2.8)	11.5 (5.0)	4.8 (1.3)	13	2
Total	289	8.0 (4.7)	13.6 (10.4)	5.6 (2.8)	180	107

* For two patients, the TESTS value is missing.

To gain some appreciation of the potential clinical significance of the statistically significant findings, HISTORY and PHYSICAL were calculated using the multiple regression equations, entering mean values for all but TIME, and entering TIME alternately two standard deviations above and below its mean. On average, then, as TIME decreased from 13.5 to 4.7 hours, HISTORY decreased from 9.8 to 6.7 items, and PHYSICAL decreased from 16.7 to 12.4 items. This represents reductions of 32% and 26% in history and physical examination items, respectively. For the finding between TMINSHFT and COMPREHN, the calculation compared the beginning (TMINSHFT = 0) and end (TMINSHFT = 1) of an average shift. On average, between the beginning and end of a shift, COMPREHN decreased from 6.0 to 5.3 body systems, a decrease of 12%.

Comparison of the simple and partial correlation coefficients in Table II illustrates the importance of the adjustments. Some correlations that were statistically significant as simple correlations changed to nonsignificant partial correlations, whereas others had their statistical significance enhanced. Alternatively, some statistically nonsignificant simple correlations emerged statistically significant as partial correlations.

All ten residents had worked on at least 12 consecutive days without a day off, and the fewest number of shifts worked by any resident was 20. Table III presents the partial correlations between these numbers of shifts worked and the performance measures. The partial correlations were adjusted for individual differences among residents and differences between the two diagnoses (DISEASE). Test

TABLE II. Correlation Coefficients of Shift Work Characteristics with Performance Measures*
(N = 248)

Shift Work Characteristic	HISTORY Simple	HISTORY Partial	PHYSICAL Simple	PHYSICAL Partial	COMPREHN Simple	COMPREHN Partial	TESTS Simple	TESTS Partial
PTSEEN	−0.15[†]	−0.08	−0.10	−0.03	−0.15[†]	−0.11	−0.07	0.05
(PTSEEN)^2	−0.17[†]	−0.09	−0.09	−0.01	−0.11	−0.06	−0.08	0.05
TMINSHFT	−0.03	−0.09	0.01	−0.03	−0.05	−0.13[†]	0.05	0.03
(TMINSHFT)^2	−0.03	−0.10	0.00	−0.04	−0.05	−0.13[†]	0.07	0.04
TIME	0.17[†]	0.21[‡]	0.01	0.13[†]	−0.03	0.09	−0.03	−0.07
(TIME)^2	0.18[†]	0.22[‡]	−0.01	0.12	−0.05	0.09	−0.01	−0.06

* Partial correlation coefficients are with adjustments for patient age, DISEASE, TIMEODAY, TOTALPTS, and individual differences among residents.
[†] $p = <0.05$.
[‡] $p = <0.001$.

patients were also classified as having been seen during either the first half or the last half of a shift. Test patients with TMINSHFT values less than 0.5 were assigned to the first half of a shift, and the remaining test patients to the last half.

No statistically significant partial correlations were found with either a schedule of 12 consecutive daily shifts or a total of 20 shifts (Table III). Borderline statistical significance ($p = 0.07$) was found with the TESTS measure for the first half of shifts in the 12 consecutive daily shifts schedule. The prominence of the partial correlation in the first half of shifts in the 12-shift schedule could not be explained due to lack of testing during the last half of shifts. There was no difference in frequency of testing between the first and last half of shifts (chi-square corrected = 0.63, $p = 0.43$). Borderline statistical significance also was found in the case of physical examination comprehensiveness with the 20-shift schedule for the full shift ($r_p = -0.12$, $p = 0.07$) and the last half shift data ($r_p = -0.17$, $p = 0.06$).

DISCUSSION

Small decreases in second-year resident performance correlated with shift work characteristics in an ED where shifts were not extraordinarily long. There was minimal rotation through the shift schedule; most shifts did not involve overnight duty, and time off between shifts appeared adequate to prevent sleep deprivation. The latter remains to be proved. The schedule was not unlike that considered for adoption as state regulations in New York State.[1,2] As the length of time worked within a shift increased, approximated by the TMINSHFT measure, only comprehensiveness of physical examinations showed a small, statistically significant negative correlation. However, what was more important than the number of hours worked or the number of patients seen in a shift, which had no effect on performance, was the busyness of a shift. This recalls complaints cited in the literature of excessive workload and not having adequate time to see patients.[6,10,15] It is not known if the residents included in this study had such complaints, but the findings show that as shifts became busier, fewer history and physical examination items were recorded.

TABLE III. Partial Correlation Coefficients of Number of Shifts Worked with Performance Measures for Full Shifts, First Half of Shifts, and Last Half of Shifts*

Performance Measure	Number of Shifts Worked 12 Consecutive Shifts Full Shift	 12 Consecutive Shifts First Half	 12 Consecutive Shifts Last Half	 20 Shifts[†] Full Shift	 20 Shifts[†] First Half	 20 Shifts[†] Last Half
HISTORY	0.08	0.16	0.07	0.03	0.11	−0.01
(N)	(151)	(56)	(95)	(226)	(88)	(138)
PHYSICAL	−0.05	−0.06	−0.08	0.02	0.10	−0.07
(N)	(151)	(56)	(95)	(226)	(88)	(138)
COMPREHN	−0.12	−0.16	−0.09	−0.12[‡]	−0.06	−0.17[§]
(N)	(151)	(56)	(95)	(226)	(88)	(138)
TESTS	−0.09	−0.27[‡]	−0.07	0.02	0.03	−0.01
(N)	(141)	(56)	(95)	(224)	(87)	(137)

* Partial correlation coefficients are with adjustments for individual differences among residents and DISEASE.
[†] Unlike the "12 consecutive shifts," the 20 shifts, while consecutive, were not all on consecutive days, since residents had days off.
[‡] $p = 0.07$.
[§] $p = 0.06$.

Fatigue might have played some role in the type of shift schedule studied. Physical examination comprehensiveness decreased with an increase in the number of hours worked within a shift and as the number of shifts worked increased (the findings were of borderline significance in the 20-shift schedule). With the latter, the effect was most evident during the last half of shifts, when accumulating fatigue might be expected to be most prominent. TESTS also decreased as number of shifts worked increased with the 12 consecutive daily shift schedule, but only during the first half of shifts, and again the finding was of borderline significance. Finding an effect only during the first half of shifts, if one did exist, is difficult to explain. It could not be due to a lack of testing in the second half of shifts, since the frequency of testing was similar between both halves of shifts. The effect on TESTS did not persist in the 20-shift schedule, perhaps because the effect was attenuated due to days off.

Decrements in performance related to the busyness of an ED can occur independent of fatigue, although the two conceivably can interact and augment their deleterious effect on performance. The more significant finding in this study was of negative effects on performance related to shift busyness. It should lead one to explore the adequacy of staffing levels during the busier shifts, which is a separate issue from the duration of hours or number of shifts worked. The important implication of these findings for teaching hospital EDs is that as working hours in shifts are reduced to the ten- to 14-hour range, appropriate attention must be given to adequate staffing levels. If not, the negative effects of increased workload per physician on quality of care could offset and perhaps exceed any quality gains from decreased working hours.

It is possible that the effects were selectively limited to recording rather than actual performance, although such selectivity would have to have been specific to the history and physical examination. The recorded information on tests was corroborated by billing data. Medical records may not be complete, but studies do support the validity of their content.[16] Nevertheless, the findings are consistent with complaints of overwork and deserve further study.

Further study also is indicated to determine if these findings can be generalized to other types of patient care problems, more demanding shift schedules, and other work settings. This is important since those aspects of resident performance found to be affected in this study were the basic physician tasks of information gathering essential to arriving at valid diagnoses and instituting effective therapy.

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A QUESTION OF ETHICS

Medical advancement in the past fifty years has been great. Hospitals have multiplied and expanded their services, medical schools have fitted themselves to give better training to their graduates, and the practitioner has equipment and methods at his disposal that were unheard of at the opening of the century. However, according to the *Military Surgeon* [1950; 106:315],

> There are signs in the air that there has been no parallel life in the spirit of the individual that would practice medicine. Indeed there are indications of serious defect in the mental attitude of the medical student body of today toward the vocation they have chosen to follow. The text of this discourse is found in an item of "Current Comment" in a recent issue of the *Journal of the American Medical Association.* This item relates to the proceedings of a meeting of the Association of Interns and Medical Students. This pressure group in convention went on record for a program of benefits for its membership including minimum pay for interns and residents, for vacations and time off, for accident insurance and extra pay for work regarded as hazardous. However reasonable these objectives may be, there can be but abhorrence and concern at the methods being employed for their accomplishment. At a stage in their careers when the members of this group should have no interest but that of perfecting their professional preparation, they are banding themselves together for exacting material advantages by means of political and industrial pressures. . . .

For more detailed information about the activities of the group in question, we refer our membership to an article in the *Saturday Evening Post* of February 11, 1950, by Vic Reinemer, "How Our Commies Defame America Abroad," and the more recent article in *Medical Economics*, March, 1950, entitled, "Leftist Minority Woos Future Doctors," a behind-the-scenes look at the Association of Interns and Medical Students. . . .

It may be argued that this Association of Interns and Medical Students is just another medical society, but it would be a jaundiced eye that could see it as such. It more closely resembles a labor union in that the benefits it demands are personal ones for its members. . . .

The mental query that arises from this discussion is what manner of professional ethics may be expected in the years to come from individuals who in their years of student work and of internship bind themselves together into pressure units for the furtherance of their interests? We feel that an inherent sense of personal dignity will keep the right-minded medical student and intern out of such an organization.

EDITORIAL
(*NY State J Med* 1950; 50:1569-1570)

Reducing neurologic trauma in sports

LAWRENCE B. LEHMAN, MD

The full spectrum of sports-related injuries that affect the nervous system has been reviewed in previous publications.[1-7] This includes both acute (and often dramatic) injuries as well as chronic types. Acute sports-related injuries of the nervous system include closed head trauma, which is particularly common in contact sports such as boxing, football, and ice hockey. Closed head trauma is also seen in many other activities such as horseback riding and cycling. Spinal cord injury with or without vertebral column fracture is particularly common in diving and gymnastics, as well as in team sports such as rugby and football. Peripheral nerve injuries are seen in almost every contact and competitive sport. Chronic sports injuries of the nervous system are also well known. A good example of these is the chronic encephalopathy of athletes who engage in contact sports, particularly boxing.

The goal of this article, however, is not to reiterate earlier works on this subject, nor to discuss the pathophysiologic mechanisms underlying these injuries. Rather, it is to discuss certain conservative measures that may be implemented by personal and team physicians and consultants in order to reduce or eliminate nervous system morbidity and mortality. These measures include preseasonal medical evaluation and interval reexamination, conditioning and proper training regimens, adequate supervision, the institution of available mechanical safety devices, and restriction or elimination of some particularly dangerous maneuvers.

EVALUATION AND REEVALUATION

The single most important intervention in preventing serious athletic injuries is the evaluation of the individual prior to final selection of a suitable sports activity. Unfortunately, not all individuals are physically competent to participate in all sports. It is the responsibility of the consulting physician not only to perform a thorough history and physical examination, but also to analyze how well suited for a particular sport a prospective player is, or may become.[8,9] The preseason examination should include a general physical examination with documentation of the individual's vital signs, height, and weight; routine cardiovascular, pulmonary, abdominal, and extremity examinations; and a comprehensive neurologic assessment. The neurologic assessment should evaluate and document the individual's mental status (including attitude and judgment, memory, and perception); cranial nerve functioning (with particular attention directed towards assessing vision and hearing); motor and sensory functioning; and balance, coordination, and reflexes. It should serve not only as a preparticipation screening examination, but also as a baseline examination for comparison throughout the season.

A person with prior major surgery on his brain or spine should not participate in contact sports for a period of time dictated by the primary nervous system disorder, postoperative neurologic status, and physical integrity and stability of the craniospinal axis. A person with an untreated cerebral aneurysm or spinal arteriovenous malformation, or a poorly controlled seizure disorder, should not participate in vigorous competitive or contact sports. Other prospective participants may need specific instructions about limitations, restrictions, or protective devices, such as the scuba diver or skydiver with an indwelling ventriculoperitoneal shunt, the gymnast with congenital scoliosis, the avid equestrian with a seizure disorder, and similar cases. With careful and thoughtful guidance and supervision, even moderately and severely physically and mentally impaired individuals may benefit from organized sports, including the Special Olympics and similar projects.

Persons who are participating concurrently in several sports activities, as high school and college athletes often do, may incur repeated nervous system insults in a relatively short period of time; these injuries may be almost synergistic in nature. The cerebral concussion or spinal cord injury that results from each activity is similar, and hence the athlete who is subjected to injury in boxing one day should be advised against participating in football or rugby the next day.

The periodic reexamination of the athlete, perhaps in midseason, and certainly annually, may serve to reveal more subtle deficits in vision, coordination, endurance, strength, balance, and other parameters that may contribute to these injuries. Clinical reexamination of the nervous system is mandatory following significant injuries such as the loss of consciousness. A player who sustains a cerebral concussion with loss of consciousness during a game or

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activity should be removed from that activity at once to prevent further risk. On occasion, certain additional studies may be indicated, including plain radiographs, tomography, computer assisted tomography, magnetic resonance imaging, neurophysiologic diagnostic studies, and neuropsychologic studies. These supplemental studies may be indicated for evaluating an athlete who manifests a significant or new focal or lateralizing clinical finding elicited during the neurologic examination. They are most useful in discerning the presence of mass lesions such as intracranial hemorrhages, or bony fractures of the skull and vertebral column. A clinical reexamination after a course of physical therapy may also be of use.

CONDITIONING AND TRAINING

Proper conditioning and training of the athlete is another important component that must be considered in reducing neurologic sports-related morbidity and mortality.[1,3,10,11] Both general overall physical fitness and specific regionalized conditioning which may be sport-specific need to be addressed. An example of the latter is cervical muscle conditioning in sports such as football, rugby, and soccer, where particular stresses are placed on the neck. Another example is lumbar conditioning in such sports as gymnastics and skydiving, which place particular stresses and strains on the lower back.

Conditioning and training must be the joint effort and responsibility of team coaches, trainers, and physicians. A regimen of appropriately selected warm-up, muscle strengthening, endurance, and other exercises should be devised and frequently updated for each player. These may combine the use of physical and occupational therapy techniques with the use of specialized equipment.

An integral part of the process is didactic and not physical and consists of the discussion of injury prevention. The trainer or coach should emphasize that participants must take certain measures in order to insure their safety and the safety of others. These measures include using equipment only in optimal condition and avoiding certain dangerous maneuvers.[2] A player found using one of these prohibited techniques should be suspended from play. Training drills should also include a rehearsal of emergency resuscitation and evacuation of an injured athlete.

SUPERVISION

Athletes of all ages and levels of skill require adequate supervision. Supervision is necessary to enforce the rules of the game; avoid infractions of technique, sportsmanship, or equipment; assess the competence and progress of the participants; and effect the early and safe evacuation of an injured athlete. Supervision is not only required at "formal" games, but also at sandlot games, Little League games, team exercises and practice sessions, and the like. Those providing supervision should be medically knowledgeable and prepared to handle minor on-field emergencies. They should have ready access to team trainers and physicians for immediate back-up support in the case of more severe injuries, and they should have a thorough understanding of the physical restrictions and limitations, as well as the goals of each participant during the activities. At a minimum, they should be familiar with basic and advanced first aid techniques, and the fundamentals of evaluation and evacuation of an injured athlete from the field, as taught in a number of regional emergency medicine technician courses. For higher risk sports activities such as boxing, wrestling, gymnastics, auto racing, and diving, a physician or paramedic team should be present, and an ambulance should be immediately available for transport to a predesignated trauma facility with general surgery, neurosurgery, and orthopedic surgery consultants available.

MECHANICAL MEANS

Among the most controversial of the measures suggested to help reduce neurologic morbidity is the compulsory employment of certain mechanical means.[7,12-14] These range from the widely accepted and approved belt and harness of race drivers, to the more controversial protective skull helmet. Although the mandatory use of the helmet in many sports has dramatically reduced the occurrence and severity of head injuries,[15-21] many clinicians and investigators have unjustly associated the use of the helmet with an increase in overall cervical spine injuries.[4,22] In recent years, the consensus has been that there is not a direct causal relationship between helmet use and cervical spine injury,[16,23] particularly if the helmet is of appropriate design, construction, and fit.

Although to date there is no satisfactory cervical protective device, some advocate the use of cervical rolls in an attempt to prevent extreme neck extension and rotation. Some have advocated structural changes in the design of stadiums, have demonstrated flaws in equipment design, and have suggested new types of surface padding.[5]

PROHIBITING DANGEROUS MANEUVERS

A number of dangerous maneuvers have been described which appear to cause an unacceptable amount of severe and catastrophic nervous system injury.[2,4,7,22-25] Among the most dangerous of these maneuvers is "spearing," which involves using the helmeted, buttressed head as a sort of battering ram, with the neck held in flexion. This maneuver is seen in football, rugby, and a number of other contact sports. It accounts for a large number of devastating cervical spine as well as brain injuries. Many investigators have found that the forces applied to the cervical spine in this maneuver are maximal, and frequently result in a burst fracture with subluxation and spinal cord compression.[21] When the intentional use of this maneuver was made illegal in collegiate football[4,7] and Canadian ice hockey,[21] the incidence and severity of these injuries diminished.

"Stinging" may be produced by the intentional depression of the shoulder, or unintentional blow to the depressed trailing shoulder, which results in a stretch injury to the upper portion of the brachial plexus. It is accompanied not only by arm weakness, but also by severe, painful dysesthesias. This maneuver has been utilized in several contact sports, including hockey and football.

The "clothesline" or "guillotine" seen in football, professional wrestling, and other sports has resulted in a number of serious cervical hyperextension injuries, according to investigators.[4] The pathophysiology and biomechanics of this maneuver have been refuted and questioned by others.[21]

The "pyramid formation" in cheerleading, traditionally a low risk activity, has been the cause of an alarming number of profound neurologic injuries.

Direct blows to the head and neck, as seen in boxing, wrestling, and altercations in other sports such as hockey and rugby, have repeatedly been shown to be related to severe neurologic dysfunction.[24,25] Many have suggested banning certain direct blows directly, and even banning entirely such high risk sports as boxing.

The use of foreign objects to produce neurologic trauma, such as the "beanball" in baseball, and "sticking" in ice hockey, can only be labelled as assault, and should not be tolerated in the realm of sportsmanship.

Certain acrobatic gymnastic activities and skiing maneuvers have been responsible for an alarming number of neurologic disorders.[21,26] The "Fosbury flop" is one such well known example of this type of dangerous maneuver. In this maneuver, in high jumping or pole vaulting, for example, the athlete leaps and twists, landing on the upper back and neck. Recently, there has been a strong movement to eliminate the trampoline entirely from the spectrum of gymnastic competition for this reason.[7]

The participation of youngsters in all-terrain-vehicle competition, motorcross, and other such activities must also be considered dangerous. Similarly, scuba diving without a companion, and sports diving by people who are ill trained and ill prepared, are unwise and potentially injurious activities.

Participating in any sports activity for which the participant lacks proper training, skill, equipment, and supervision, and for which he or she has not been medically "cleared" must be thought of as potentially dangerous. Even when these criteria are met, the individual must be periodically reassessed for evidence of fatigue, intoxication, and equipment flaws which may preclude participation until reevaluated.

CONCLUSIONS

Preventing and reducing the severity of sports injuries should be a concern of all of us. It is imperative to recognize that there are inherent risks in all sporting activities, and that these risks can be minimized only by a multidisciplinary approach to the problem. This approach begins with the thorough evaluation of the appropriateness of an athlete for a particular sport; extends through the on- and off-field training and conditioning process; involves qualified, competent, and caring supervision; and meticulous application of mechanical safety measures. It does not end there, however, because further work is needed to improve on each of these areas. In certain cases, the only practical solution may be the curtailment or elimination of certain high risk maneuvers or activities.

Health care professionals must also act to inform and educate members of the community about the potentially devastating and costly public health aspects of the neurologic sequelae of sports trauma. The assistance of educators, team and school district administrators, local politicians, and parents' groups should be enlisted in order to increase the public's awareness of these tragic injuries and of measures to reduce them.

By following the five critical steps outlined in this review, significant levels of neurologic morbidity and mortality can be eliminated. There are some who claim that increased safety measures will detract from spectator interest and player participation. However, experience has shown that the mandatory use of batting helmets in baseball, the prohibition of "spearing" in college football, and rigorous prematch physical examinations in boxing have resulted in the opposite effect. Just as important, however, are efforts at educating and informing athletes, their families and supporters, and the public at large about potential dangers and how they can be minimized.

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Abdominal tuberculosis

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With the advent of antituberculosis chemotherapy in the early 1940s, the frequency and severity of intestinal tuberculosis in the United States and Western Europe has significantly diminished.[1] However, abdominal tuberculosis commonly occurs in the Third World. (There is usually evidence of pulmonary disease and the diagnosis is easily made.) Although abdominal tuberculosis is now only rarely seen in medical practice in the United States, physicians should be aware of its existence and understand its nonspecific and protean clinical manifestations. Severe problems arise when a patient presents with tuberculosis of the intestines or liver in the absence of apparent pulmonary disease. Intestinal tuberculosis may often be confused with other diseases, such as Crohn disease and intestinal neoplasm. The misdiagnosis of abdominal tuberculosis as Crohn disease is particularly unfortunate because the corticosteroid therapy used for the latter risks miliary dissemination of the former.

EPIDEMIOLOGY

There are no recent studies of the incidence of enteric tuberculosis in the United States. Older studies from the United States and Great Britain[2] have emphasized that immigrants from areas of the world endemic for tuberculosis (Asia) constitute a large proportion of patients with this entity. These studies also indicate that immigrants from developing areas such as India, Africa, and Asia will have a high frequency of primary intestinal tuberculosis—ie, enteric tuberculosis, without evidence of extraintestinal involvement. In urban areas with large populations of alcohol abusers or immigrants from endemic countries, the incidence of abdominal tuberculosis is high. In more affluent parts of the country, the disease is rare. Most granulomatous disease of the bowel in the United States and Western Europe is due to Crohn disease, whereas in India and other developing countries, the most common cause is intestinal tuberculosis.

PATHOGENESIS

The pathogenesis of intestinal tuberculosis is unclear. However, it is likely that the disease is a consequence of reactivation of previously inactive tuberculosis rather than a reinfection.[3] Possible routes of infection include direct invasion by ingested organisms, hematogenous seedings, and extension from contiguous organs.

Direct Invasion. Although isolated cases of primary tuberculous enteritis are occasionally reported in developing countries, the widespread pasteurization of milk and the disappearance of bovine tuberculosis in the United States have excluded contaminated milk as a cause of tuberculosis in this country. Secondary infection of the intestine may arise from contamination of chyme by bacteria from other sites, generally the lungs, through swallowing of sputum. This mechanism of spread is supported by the strong clinical impression that there is an association between the severity of pulmonary tuberculosis and the frequency of enteric infection. The factors that permit initiation of enteric tuberculosis by swallowing sputum are unclear. Intestinal involvement is probably a function of the number and virulence of the ingested organisms and the metabolic status of the patient. After the patient ingests the organism, the bacillus enters the small bowel, where the sites of infection appear to be influenced by the presence of physiologic stasis, the rate of water and electrolyte absorption, the abundance of lymphoid tissue, and the amount of time that the bacillus is in contact with the intestinal mucosal surface. In decreasing order of frequency, the most common sites of enteric infections are the ileum (especially the ileocecal area), colon, jejunum, rectum, and duodenum.

Hematogenous Spread. This form of spread of the tubercle bacillus is supported by the observation that the enteric lesion is usually found in the submucosa with a normal overlying mucosa. Perhaps a "silent" bacteremia occurs during the active phase of pulmonary tuberculosis and allows for hematogenous spread.

Extension. Although enteric tuberculosis can occur by direct extension from infected adjacent organs—eg, the female adnexa—invasion of the bowel does not seem to occur in the setting of tuberculous involvement of the peritoneum.

PATTERNS OF INVOLVEMENT

In considering abdominal tuberculosis, it is generally useful to classify it as intestinal, peritoneal, or hepatic.

Intestinal Tuberculosis. Like Crohn disease, with which it is so frequently confused, tuberculosis may affect any portion of the intestinal tract from the mouth to the anus, but tends to affect the terminal ileum most often. This is probably because the terminal ileum contains much lymphoid tissue and is an area of stasis. While the disease is most commonly confined to the ileocecal area,

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there are cases in which the disease may be more confluent. There may be proximal or distal intestinal involvement, or there may be "skip lesions," in which apparently normal bowel is found between areas of obviously diseased bowel. On gross examination, the bowel wall is thickened, and the lumen shows evidence of stenosis. Caseating granulomas are the histologic hallmark of tuberculosis. However, they are rarely seen, and cultures of even the most actively diseased bowel are usually negative. Inflammation is transmural, giant cells are common, and caseation is rare, except in the setting of draining mesenteric lymph nodes.

The symptoms of intestinal tuberculosis are not specific, and a pathognomonic syndrome does not occur. Abdominal pain, fever, weight loss, weakness, and nausea are the most common symptoms. Although radiographic evaluation of the chest is normal in many patients with intestinal tuberculosis, the symptoms of abdominal pain, diarrhea, and anorexia in a patient with pulmonary tuberculosis should suggest the possibility of intestinal involvement.

The clinical hallmark of intestinal tuberculosis is abdominal pain. Because the disease process is most commonly localized to the ileocecal area, abdominal pain is usually located in the right lower quadrant. This pain is due to a combination of intestinal obstruction and the inflammatory reaction. The pain is variable in severity, site, and duration. Some patients will present with colicky pain typical of small bowel obstruction. A few patients may present with acute intestinal obstruction or, occasionally, with peritonitis due to cecal perforation. Tuberculous appendicitis may simulate the symptom pattern of ordinary appendicitis, but more commonly presents as a chronic, relapsing disorder with symptoms being intermittent. Some patients present with insidious generalized pain extending over years with a paucity of physical findings. This group of patients is often misdiagnosed as having irritable bowel syndrome or nontuberculous inflammatory bowel disease. Most patients tend to be constipated; diarrhea occurs less often than might be expected in view of the inflammatory nature of the disease. When patients complain of diarrhea, the characteristics are similar to those of Crohn disease, although gross blood or pus is seldom seen. The most helpful physical signs are a right iliac fossa mass which is tender, and fever.

Peritoneal Involvement. Ascites occurs in this setting, but it is not clinically significant. The classic pattern of "doughy ascites" with fever is rare. Weight loss and fever in a patient with ascites who has a high ascitic fluid albumin should suggest this condition. Peritoneal involvement may also cause intestinal adhesions and the development of an obscure abdominal mass as a result of omental inflammation.

Hepatic Tuberculosis. Tuberculosis is a classic cause of fever of unknown origin. Liver involvement is relatively common, and liver biopsies reveal ill-defined, noncaseating granuloma. Liver function remains normal, and changes are nonspecific. The tubercle bacillus is only rarely seen or cultured in this condition.

LABORATORY FINDINGS

Biochemical and hematologic determinations reflect the disturbances of organ function and the severity of the disease process, but do not establish a diagnosis of tuberculous enteritis. Thus, the hemoglobin concentration is generally slightly depressed because of "anemia of chronic disease." The erythrocyte sedimentation rate is usually elevated. The white blood count is generally normal, although there will often be a monocytosis. The serum albumin concentration is depressed. Some studies suggest that patients who have lower values of serum albumin are at greatest risk of death following laparotomy.[4] A raised serum alkaline phosphatase level suggests hepatic involvement or, less likely, bony involvement.

Whereas patients with active pulmonary tuberculosis almost invariably have a positive tuberculin reaction, the opposite is true of those with abdominal tuberculosis. A negative tuberculin skin test reduces the likelihood of a diagnosis of abdominal tuberculosis, but does not exclude it. Perhaps these patients are immunologically hyporesponsive because of the severity of the illness and the malnutrition. On the other hand, a positive skin test indicates tuberculous infection but does not establish active disease, nor does it permit diagnosis of the patient's symptoms as being due to intestinal involvement of tuberculosis.

Culture of acid-fast bacilli from sputum, urine, gastric contents, pleural fluid aspirate, excretions from fistulae, and stool are helpful when positive. However, these studies are generally unrewarding. The availability of biopsies obtained through colonoscopy has helped somewhat in making a diagnosis.[5] However, tuberculous enteritis or enterocolitis may be difficult or impossible to distinguish from Crohn disease, particularly in a superficial mucosal biopsy specimen obtained via colonoscopy. In comparing 139 patients with intestinal tuberculosis and ten with Crohn disease, Tandon and Prakash[6] found caseating granulomas, which are not always present in cases of tuberculosis, to be the only absolute distinguishing feature. Pathologic features favoring a tuberculous cause include multiple circumferential ulcers, overlying Peyer patches and lymphatics, and confluent granulomas with caseous centers.

Examination of peritoneal fluid may suggest tuberculous infection if there is a predominance of lymphocytes and an increased protein content. Any ascitic (or pleural) fluid with a protein content greater than 2.5 g% should be cultured for tuberculosis.

RADIOGRAPHIC SIGNS

In the absence of active pulmonary disease, the chest radiograph is usually normal. Thoeni and Margulis[7] reported that only 50% of patients with gastrointestinal tuberculosis show radiographic evidence of pulmonary involvement. Even if small calcifications are seen, they often fail to suggest the diagnosis of tuberculosis. Although there are no pathognomonic signs for enteric tuberculosis, certain features suggest the disease. Kolawole and Lewis[8] listed a hyperplastic form with a "pipestem" colon and a retracted, cone-shaped cecum as the most common radiologic manifestation. Often, there is an irregular, ulcerating terminal ileal stricture with cecal distortion or involvement with a mass effect, indistinguishable from that of Crohn disease. In the patient who has isolated large bowel disease, the disease tends to be segmental and is manifested radiographically by massive, annular constrictions and ulcerations. Again, the appearance may be indistinguishable from that of Crohn disease, especially when "skip lesions" are present. The presence of an enteric, enterocutaneous, or enterovesicular fistula supports the diagnosis, but, again, is indistinguishable from that of Crohn disease.

DIAGNOSIS OF ABDOMINAL TUBERCULOSIS

The most important factor in making the diagnosis of abdominal tuberculosis is to consider this disease in the differential diagnosis of obscure abdominal disease. The diagnosis of tuberculous enteritis is not difficult in the presence of known pulmonary tuberculosis; however, making the diagnosis in the absence of active pulmonary disease is difficult. In clinical practice, bacteriologic proof may be unavailable, and the decision to treat is based on a general clinical impression rather than a rigid diagnosis. As mentioned previously, a negative chest film and negative tuberculin skin reaction do not exclude intestinal tuberculosis from the differential diagnosis. It should also be mentioned that a specific diagnosis may be difficult to make in a patient who has recently received antituberculosis therapy, since such therapy has an inhibitory effect on culture of the bacillus and on the characteristic histopathologic features.

The most helpful diagnostic factor is probably the patient's ethnic and social background. Abdominal pain, fever, and a mass are likely to be due to tuberculosis in an immigrant from India or Asia, while the same symptoms in an indigenous American are usually due to Crohn disease or appendiceal abscess. In the older patient, ileocecal tuberculosis must be differentiated from carcinoma of the right colon.

The studies required to make a specific diagnosis of abdominal tuberculosis include tuberculin skin testing; chest radiograph; barium contrast studies of the small and large bowel; culture of appropriate body fluids, excretions, and tissue; and histologic examination of resected tissue. Computed tomographic scanning of the abdomen can be useful in the diagnosis of intra-abdominal tuberculosis.[9]

DIFFERENTIAL DIAGNOSIS

Disease entities that by their signs and symptoms and their radiographic, morphologic, and histologic features must be differentiated from tuberculous enteritis most commonly include Crohn disease, appendiceal abscess, and carcinoma of the cecum and right colon. A more extensive list is found in Table I. The differential diagnosis of tuberculosis of the liver includes the diseases that cause granulomas in the liver, namely drug reactions, sarcoidosis, and lymphoma.

TABLE I. Differential Diagnosis of Tuberculous Enteritis

Small bowel	Lymphoma Fungal and parasitic infection Crohn disease Vascular insufficiency
Ileocecal area and colon	Crohn disease Ulcerative colitis Carcinoma of the large bowel Lymphoma Periappendiceal abscess Amebiasis Fungal infection Sarcoidosis Actinomycosis
Rectosigmoid	Crohn disease Ulcerative colitis Carcinoma Amebiasis Ischemic colitis Diverticulitis Foreign body reaction Fungal infection Schistosomiasis Lymphogranuloma venereum Actinomycosis Endometriosis

THERAPY

In contrast to pulmonary tuberculosis, in which the response to therapy may take some time, most patients with abdominal tuberculosis respond rapidly to therapy. This is because the abdominal sites of involvement, as well as other sites of extrapulmonary disease, usually contain smaller numbers of tubercle bacilli than pulmonary sites, and pharmacokinetic studies have indicated that first-line antituberculosis drugs, especially isoniazid and rifampin, penetrate virtually all extrapulmonary sites at levels well above the minimal inhibitory concentrations for *Mycobacterium tuberculosis*.[10] Clinically, the majority of patients recover from their fever and notice an increase in well-being and diminution of pain within a week.[1] Within two weeks, the majority of patients are considerably improved. The rapid response to therapy is the basis for the suggestion that a therapeutic trial in the appropriate clinical setting is reasonable. Thus, when confronted with a patient in whom the differential diagnosis is tuberculosis or inflammatory bowel disease, little is lost in treating the latter condition with antituberculosis therapy, whereas treating the former condition with corticosteroids can be disastrous.[11] In fact, Crohn et al,[12] in the original publication on regional enteritis, commented on the significance of the finding of tubercle bacilli to exclude regional enteritis.

Although the published experience with antituberculosis therapy of intestinal tuberculosis is limited, a recently published consensus statement from the American College of Chest Physicians[13] has suggested that modern, optimal chemotherapy consists of nine months of a "core" of isoniazid (5–10 mg/kg up to 300 mg orally or intramuscularly) and rifampin (10–20 mg/kg, up to 600 mg orally), supplemented for the first three months with pyrazinamide, ethambutol, or streptomycin. There are advantages and disadvantages to each of the three drugs suggested as a supplement to the "core" (Table II). Ethambutol is the least toxic and will help prevent emergence of drug-resistant organisms, but has the potential for causing ocular disease and, when given in low doses (15 mg/kg/day), is not bactericidal. Streptomycin is slightly more potent than ethambutol, but also slightly more ototoxic and nephrotoxic. Injections of streptomycin make it easier to supervise therapy, but are inconvenient for both the patient and the physician. Pyrazinamide is also more potent than ethambutol but slightly more toxic (hepatotoxicity,

TABLE II. First-line Antituberculosis Drugs*

Drug	Daily Dose (Adult)	Most Common Side Effects	Tests for Side Effects	Remarks
Isoniazid	5–10 mg/kg up to 300 mg orally or intramuscularly	Peripheral neuritis Hepatitis Hypersensitivity: fever, skin rash, arthralgias	Liver tests† monthly if high risk for developing toxic hepatitis	Bactericidal Pyridoxine, 10–50 mg as prophylaxis for neuritis Discontinue if symptomatic hepatitis or 3-fold elevation of AST
Rifampin	10–20 mg/kg up to 600 mg orally	Hepatitis Thrombocytopenia Fever Orange urine, increased tears and saliva	Liver tests† Platelet count	Bactericidal Discontinue if jaundice or 3-fold elevation of AST Negates effect of birth control pills
Pyrazinamide	15–30 mg/kg up to 2 g orally	Hyperuricemia Hepatotoxicity	Liver tests† Uric acid acid	Combination with an aminoglycoside is bactericidal
Ethambutol	15–25 mg/kg orally	Optic neuritis (very rare at 15 mg/kg; reversible with discontinuation of drug) Skin rash	Periodic test of visual acuity and red-green color discrimination	Use with caution in presence of renal insufficiency or when eye testing is not feasible
Streptomycin	15–20 mg/kg up to 1 g intramuscularly	Eighth cranial nerve damage Nephrotoxicity Hypersensitivity: skin rash, anaphylaxis	Audiograms Vestibular function Blood urea nitrogen/serum creatinine	Use with caution in older patients or those with renal disease

* Modified from Snider et al.[13]

† Alanine aminotransferase (ALT); aspartate aminotransferase (AST).

hyperuricemia), and there is less experience with this drug than with the other two.[13] This regimen will cure virtually all patients with susceptible organisms; in fact, bacterial resistance is so rare that lack of response to this regimen in a patient with abdominal tuberculosis implies an incorrect diagnosis.

In an attempt to further reduce the cost of therapy and to reduce the risk of side effects, Dutt, Moers, and Stead[14] recently reported their experience with isoniazid and rifampin alone for extrapulmonary tuberculosis. For newly diagnosed and drug-susceptible disease, they used isoniazid, 300 mg, and rifampin, 600 mg, daily for one month, followed by isoniazid, 900 mg, and rifampin, 600 mg, twice weekly for another eight months. They reserved the use of streptomycin and pyrazinamide for suspected drug-resistant cases. Their good results suggest that this may become the standard protocol.

As mentioned, most patients with gastrointestinal tuberculosis who are treated with medication do well. Surgery is usually reserved for those patients with massive hemorrhage, free perforation of an ulcer, obstruction caused by stenosis or kinking of the bowel, confined perforation with abscess or fistula formation, and, rarely, for diagnostic purposes in a patient with an ileocecal or colonic lesion whose chest film and laboratory studies are normal.

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Many in search of a few

PASCAL JAMES IMPERATO, MD

Scholars and editors share common cause in seeking primary sources of information. Why settle for secondhand accounts or for someone else's interpretation of the meaning of a word? Search it out and look it up yourself. That admonition, which I've followed for over 35 years, was given by a high school English teacher named Mr John McMurray. I frankly never enjoyed his droning on about Silas Marner because I was among the youth on whom good literature is so often sown before the field is ready. Mr McMurray, and this is how I still refer to him so many years later, exhorted us to purchase a Thorndike-Barnhart dictionary and to frequent libraries. I may not have appreciated Silas Marner then, but I did buy the dictionary and became a regular visitor to my local library.

Just a few weeks ago I turned to that 1951 Thorndike-Barnhart dictionary because the newer and bigger dictionaries I now own had let me down. An author had employed the expression "parsimonious explanation" in a case report and it struck me that the adjective was being inappropriately used. I looked up the word *parsimonious* in three different dictionaries and was amazed to find that it wasn't listed. *Parsimony* was there, but not *parsimonious*, having been edged out by the more commonly used words of an ever-expanding language. However, the Thorndike-Barnhart dictionary had it sandwiched in between *Parsi* and *parsley*. It not only defined the word, but also gave its Latin roots, and provided synonyms and antonyms for good measure. Thank you, Mr McMurray, I thought to myself.

As a teenager I wasn't greeted in local neighborhood libraries with open arms as Mr McMurray had predicted. Librarians of the day seem to have been of the mind that teenage boys and books didn't mix. Boys were full of mischief and high spirits, disrupted the tranquility of the reading room with giggles and horseplay, and took perverted pleasure in misfiling books on the shelves. There was truth in this assessment, because as young teenagers my friends and I rolled up magazines displayed in the back of the library and shoved them through a hole in the floor created when a steam pipe was removed. Only the *National Geographic* escaped this fate, not because it was too bulky to go through the hole but because we enjoyed reading it. Librarians responded to a group of 15-year-old boys entering the library much as a colony of prairie dogs reacts to a pack of coyotes on the horizon. A high state of alert bordering on alarm swept from the checkout desk to the reference section in a matter of seconds. Boys that age found themselves under constant visual surveillance. Even if they chose a book there was no guarantee they would succeed in checking it out, as librarians freely exercised censorship of what teenagers read. As I grew older my horizon of libraries increased, as did my skills in retrieving information.

One of the libraries I came to know is the Forty-Second Street Branch of the New York Public Library, which is a wonderful retreat for scholars and even editors. It contains more information in its stacks than could ever be crammed into the collective heads of Oxford's dons. Finding the information is both a challenge and fun, provided you know how to use the systems that can bring it to light. The library's retrieval system consisted for most of this century of the card catalog, which was user-friendly for those who knew its idiosyncrasies. It was rather easy to use, materials being arranged alphabetically and by subject, author, and title. The greatest challenge a user faced was following the letters as the collection expanded. First the z's moved away from the walls to the center of the room, soon followed by the y's and x's. Finally the t's stood in the center, threatening to annihilate the remaining table space for users. Clearly something had to be done, and eventually computers came to the rescue.

The accumulated wisdom of mankind which was once compressed into thousands of well-patinated oak catalog drawers is now on soft disks or else printed in large tomes that line the walls. Being a paper person at heart (although I am changing), as contrasted with a floppy disk person, I at first mourned the loss of the venerable card catalog. This library will never be the same, I thought to myself, convinced that computerization would somehow lessen the value of the collections in ways I hadn't thought about. I even considered it sacrilegious when they renovated the stately catalog room, ripped out the old drawers, and invited any and all to take one as a souvenir. I refused to be a party to this desecration and told the reference librarian as much. She smiled and said a few soft words of consolation that told me that she was one of them, a floppy disk person, but one at least who was sensitive to the feelings of cellulose aficionados. The library administration had shown great consideration and wisdom in this transition. They put post-1971 acquisitions on computer and printed the pre-1972 catalog cards by offset and bound

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them in large volumes. The cards are still there, or at least an offset image of them, but one must now turn pages to find them instead of fingering their fellows. The sliding and clicking of wooden drawers is but a memory. Also gone is the ability to tell how often an item was used by others over the years by the patina on the upper right-hand corner of the card. Some heavily used cards had their upper right-hand corners fingered into oblivion along with the class marks. So in some ways the good old days were not so good after all. Retrieval on a computer screen is unlikely to obliterate any part of the green-lettered entry with use patina, especially the all-important class mark.

I came to realize that like most others of our species I was a creature of habit. Before long the pleasant sound of fingers tapping on the keyboards of video terminals struck me as being more in harmony with the tranquility required by scholars than the opening and closing of catalog drawers. Frustration often took a toll on the drawers as disappointed seekers angrily slammed them back into the cabinets on not finding what they wanted. It remains to be seen how disappointed users will take out their frustrations on terminal keyboards.

The acid content of paper used in printing since the late 19th century has made it necessary to microfilm large numbers of documents, as has the need to optimally use space. The two long tables of microfilm machines that once stood at the far end of the North Hall have now grown to six. I've never liked using microfilm machines for a number of reasons. The machines are often old, in poor repair, have dull lights, and require intuitive mechanical skills to use. Some of those at the Forty-Second Street Library are so old in fact that they would qualify for a place at the Smithsonian. Regular users know the defects of the various machines. Machine one at table two doesn't rewind, which means you need a strong index finger to rewind the reel yourself. Machines four and six on table three have dull lights, while machines one and five on table five have broken reversal knobs, which means you can't read films on which the columns run lengthwise, not unless you want to tilt your head sideways. Often users don't rewind films, which means that the next user must start at the end of the reel and flip it for the print to read from left to right.

For many years I thought that the library had poor quality microfilms, that is until I used some of them on interlibrary loan on a machine in my medical school library. The problem was clearly the machines. Making copies of microfilmed material was until recently a frustrating experience at the library. The copy machines produced a negative, much like the old photostats that neighborhood photographers did years ago. The print was white on black, blurred or smudged, and often difficult to read. At 30 cents a shot, people complained, but there was little alternative. I once tried to console a disgruntled young lawyer by telling her that in the 1940s one had to pay a dollar for a photostatic copy, wait two or three days for it, and be happy with the result, good or bad. I remember once that my father sent me to have a document photostated at a local photographer called Hollywood Studios, which was situated under the elevated line on Liberty Avenue in Ozone Park, Queens. The sunlight never reached the facade of the building because of a peculiar relationship between the street and the "El." Hollywood—an amusing name in retrospect considering its sunless location—was the most expensive photographer in the area, but the photostat he produced was barely legible. Still my father was happy with it.

Recently the library acquired new microfilm copying machines that produce positives. Not only are the copies excellent, but the reading screens on the machines are so superior to those on the regular microfilm reading machines that people use them just for reading. This in turn has led to a newer problem. Would-be copiers must wait on line for viewers-only to finish. Those who think they might copy invariably save time by viewing on a copier. Not only is the projection screen far superior, but they save themselves a walk down the hall, a wait on another line, and the need to unwind on one machine and wind up on another.

Card catalogs and computer terminals are relatively easy to use compared to microfilm machines and microfilm copiers. First-time users standing in front of the latter two find no screen full of instructions neatly printed in green waiting for them. This, added to the poor condition of many of the machines, has led them to turn to more experienced neighbors.

I never met anyone in the library at the card catalog and have yet to meet someone in front of a computer terminal. Yet I have met many in the microfilm reading area, either because they have sought my help or because I needed theirs. Knowing how to use these machines is no small asset because most users have limited time and can't afford to lose any of it floundering around with a mechanical retrieval system.

One day a Roman Catholic priest from Providence, Rhode Island, sat down next to me on my left in front of a machine that I knew didn't rewind. I could have minded my own business and said nothing, but because I struck up a conversation with him he was able to leave the library ten minutes later and spend his afternoon sightseeing instead of being glued to a microfilm machine. I told him that the machine didn't rewind. He thanked me, looked around for another, but before getting up to leave mentioned that he was researching the history of chaplain services at the United States Public Health Service Hospital on Staten Island in New York City. He only had a few hours to examine a number of old, microfilmed documents. I told him there was no need for him to even put the reels on another machine. A colleague of mine had just written a detailed history of the hospital and had a chapter on the subject. I gave him her name, address, and telephone number and he thanked me, saying that Divine Providence had obviously directed him to the faulty machine. He followed up on my advice, got what he needed, and later told me in a letter that he had enjoyed the city's sights thanks to our chance meeting and the defective machine. He added that the knob that didn't rewind had unwittingly promoted both scholarship and leisure.

On another day I was at a microfilm machine tracking down a primary source that I needed for the *New York State Journal of Medicine*. A young man in a pinstriped suit was busily grinding away, on my right, advancing his film, when a stranger approached. The stranger asked him something and he answered that he didn't know, referring him to the clerks who give out the microfilm rolls at a central desk. The three clerks shook their heads negatively, almost in unison. So back he came to my neighbor for further advice. He was told to check with the clerks at the photocopy counter and enroute met a security guard who also shook his head no. The guard went up to the three clerks behind the desk and they shook their heads as they had before. Clearly the four of them didn't know.

Back the stranger came, reporting no luck with the staff in the North Hall. He announced that he was going out to the catalog room to find the answer. After he left, my neighbor turned to me and said, "You wouldn't happen to know what *paucity* means, would you?" I told him it meant fewness, scarcity, a small number. He seemed genuinely impressed, but I was perplexed. Surely most adults and especially microfilm users must know the meaning of *paucity*, I thought. I looked around at the other tables and began to wonder.

The stranger later reappeared, smiling in triumph. However, before he could speak my neighbor pointed to me and blurted out, "He knows what it means."

"You know what *paucity* means?" the stranger asked.

"Yes, fewness, a small number."

Wow!" he replied. "I should have asked you in the first place. Not even the librarians out in front would tell me. They said it was against the rules. I had to look it up in the dictionary."

For some reason, the dictionary had been his last resort. He was willing to settle for secondary sources that included a security guard, several clerks, a librarian, and a microfilm user when the primary source was within easy reach.

He leaned over and in a lowered voice said, "You wouldn't happen to be a lawyer, would you?"

"No," I told him, "I'm not."

Still curious, he pressed me further. "Then what do you do?"

Not wanting to identify myself, I thought for a few seconds and said, "I spend a lot of time reading what other people write and correcting their syntax."

"Ah, an English teacher!" he exclaimed. "I should have known it. Next time I'll ask you. I wish you could have taken my SATs for me."

Although driven this one time to search in a dictionary out of necessity, he had obviously not been convinced of the value of its regular use. Meanwhile, the security guard and two of the desk clerks had taken the trouble to look up *paucity* in the large dictionary in the center of the catalog room and celebrated their triumph with the stranger. Mr McMurray would be happy, I thought, that so many at least had taken the time to properly search for a few.

Recommendations for prevention of HIV transmission in health-care settings*

INTRODUCTION

Human immunodeficiency virus (HIV), the virus that causes acquired immunodeficiency syndrome (AIDS), is transmitted through sexual contact and exposure to infected blood or blood components and perinatally from mother to neonate. HIV has been isolated from blood, semen, vaginal secretions, saliva, tears, breast milk, cerebrospinal fluid, amniotic fluid, and urine and is likely to be isolated from other body fluids, secretions, and excretions. However, epidemiologic evidence has implicated only blood, semen, vaginal secretions, and possibly breast milk in transmission.

The increasing prevalence of HIV increases the risk that health-care workers will be exposed to blood from patients infected with HIV, especially when blood and body-fluid precautions are not followed for all patients. Thus, this document emphasizes the need for health-care workers to consider *all* patients as potentially infected with HIV and/or other blood-borne pathogens and to adhere rigorously to infection-control precautions for minimizing the risk of exposure to blood and body fluids of all patients.

The recommendations contained in this document consolidate and update CDC recommendations published earlier for preventing HIV transmission in health-care settings: precautions for clinical and laboratory staffs[1] and precautions for health-care workers and allied professionals[2]; recommendations for preventing HIV transmission in the workplace[3] and during invasive procedures[4]; recommendations for preventing possible transmission of HIV from tears[5]; and recommendations for providing dialysis treatment for HIV-infected patients.[6] These recommendations also update portions of the "Guideline for Isolation Precautions in Hospitals"[7] and reemphasize some of the recommendations contained in "Infection Control Practices for Dentistry."[8] The recommendations contained in this document have been developed for use in health-care settings and emphasize the need to treat blood and other body fluids from *all* patients as potentially infective. These same prudent precautions also should be taken in other settings in which persons may be exposed to blood or other body fluids.

DEFINITION OF HEALTH-CARE WORKERS

Health-care workers are defined as persons, including students and trainees, whose activities involve contact with patients or with blood or other body fluids from patients in a health-care setting.

HEALTH-CARE WORKERS WITH AIDS

As of July 10, 1987, a total of 1,875 (5.8%) of 32,395 adults with AIDS, who had been reported to the CDC national surveillance system and for whom occupational information was available, reported being employed in a health-care or clinical laboratory setting. In comparison, 6.8 million persons—representing 5.6% of the U.S. labor force—were employed in health services. Of the health-care workers with AIDS, 95% have been reported to exhibit high-risk behavior; for the remaining 5%, the means of HIV acquisition was undetermined. Health-care workers with AIDS were significantly more likely than other workers to have an undetermined risk (5% versus 3%, respectively). For both health-care workers and non-health-care workers with AIDS, the proportion with an undetermined risk has not increased since 1982.

AIDS patients initially reported as not belonging to recognized risk groups are investigated by state and local health departments to determine whether possible risk factors exist. Of all health-care workers with AIDS reported to CDC who were initially characterized as not having an identified risk and for whom follow-up information was available, 66% have been reclassified because risk factors were identified or because the patient was found not to meet the surveillance case definition for AIDS. Of the 87 health-care workers currently categorized as having no identifiable risk, information is incomplete on 16 (18%) because of death or refusal to be interviewed; 38 (44%) are still being investigated. The remaining 33 (38%) health-care workers were interviewed or had other follow-up information available. The occupations of these 33 were as follows: five physicians (15%), three of whom were surgeons; one dentist (3%); three nurses (9%); nine nursing assistants (27%); seven housekeeping or maintenance workers (21%); three clinical laboratory technicians (9%); one therapist (3%); and four others who did not have contact with patients (12%). Although 15 of these 33 health-care workers reported parenteral and/or other non-needlestick exposure to blood or body fluids from patients in the 10 years preceding their diagnosis of AIDS, none of these exposures involved a patient with AIDS or known HIV infection.

RISK TO HEALTH-CARE WORKERS OF ACQUIRING HIV IN HEALTH-CARE SETTINGS

Health-care workers with documented percutaneous or mucous-membrane exposures to blood or body fluids of HIV-infected patients have been prospectively evaluated to determine the risk of infection after such exposures. As of June 30, 1987, 883 health-care workers have been tested for antibody to HIV in an ongoing surveillance project conducted by CDC.[9] Of these, 708 (80%) had percutaneous exposures to blood, and 175 (20%) had a mucous membrane or an open wound contaminated by blood or body fluid. Of 396 health-care workers, each of whom had only a convalescent-phase serum sample obtained and tested ≥90 days post-exposure, one—for whom heterosexual transmission could not be ruled out—was seropositive for HIV antibody. For 425 additional health-care workers, both acute- and convalescent-phase serum samples were obtained and tested; none of

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* These guidelines were developed by the United States Public Health Service Centers for Disease Control and are reprinted from *Morbidity and Mortality Weekly Report* 1987; 36(2S):3S–18S.

74 health-care workers with nonpercutaneous exposures seroconverted, and three (0.9%) of 351 with percutaneous exposures seroconverted. None of these three health-care workers had other documented risk factors for infection.

Two other prospective studies to assess the risk of nosocomial acquisition of HIV infection for health-care workers are ongoing in the United States. As of April 30, 1987, 332 health-care workers with a total of 453 needlestick or mucous-membrane exposures to the blood or other body fluids of HIV-infected patients were tested for HIV antibody at the National Institutes of Health.[10] These exposed workers included 103 with needlestick injuries and 229 with mucous-membrane exposures; none had seroconverted. A similar study at the University of California of 129 health-care workers with documented needlestick injuries or mucous-membrane exposures to blood or other body fluids from patients with HIV infection has not identified any seroconversions.[11] Results of a prospective study in the United Kingdom identified no evidence of transmission among 150 health-care workers with parenteral or mucous-membrane exposures to blood or other body fluids, secretions, or excretions from patients with HIV infection.[12]

In addition to health-care workers enrolled in prospective studies, eight persons who provided care to infected patients and denied other risk factors have been reported to have acquired HIV infection. Three of these health-care workers had needlestick exposures to blood from infected patients.[13-15] Two were persons who provided nursing care to infected persons; although neither sustained a needlestick, both had extensive contact with blood or other body fluids, and neither observed recommended barrier precautions.[16,17] The other three were health-care workers with non-needlestick exposures to blood from infected patients.[18] Although the exact route of transmission for these last three infections is not known, all three persons had direct contact of their skin with blood from infected patients, all had skin lesions that may have been contaminated by blood, and one also had a mucous-membrane exposure.

A total of 1,231 dentists and hygienists, many of whom practiced in areas with many AIDS cases, participated in a study to determine the prevalence of antibody to HIV; one dentist (0.1%) had HIV antibody. Although no exposure to a known HIV-infected person could be documented, epidemiologic investigation did not identify any other risk factor for infection. The infected dentist, who also had a history of sustaining needlestick injuries and trauma to his hands, did not routinely wear gloves when providing dental care.[19]

PRECAUTIONS TO PREVENT TRANSMISSION OF HIV

Universal Precautions. Since medical history and examination cannot reliably identify all patients infected with HIV or other blood-borne pathogens, blood and body-fluid precautions should be consistently used for *all* patients. This approach, previously recommended by CDC,[3,4] and referred to as "universal blood and body-fluid precautions" or "universal precautions," should be used in the care of *all* patients, especially including those in emergency-care settings in which the risk of blood exposure is increased and the infection status of the patient is usually unknown.[20]

1. All health-care workers should routinely use appropriate barrier precautions to prevent skin and mucous-membrane exposure when contact with blood or other body fluids of any patient is anticipated. Gloves should be worn for touching blood and body fluids, mucous membranes, or non-intact skin of all patients, for handling items or surfaces soiled with blood or body fluids, and for performing venipuncture and other vascular access procedures. Gloves should be changed after contact with each patient. Masks and protective eyewear or face shields should be worn during procedures that are likely to generate droplets of blood or other body fluids to prevent exposure of mucous membranes of the mouth, nose, and eyes. Gowns or aprons should be worn during procedures that are likely to generate splashes of blood or other body fluids.
2. Hands and other skin surfaces should be washed immediately and thoroughly if contaminated with blood or other body fluids. Hands should be washed immediately after gloves are removed.
3. All health-care workers should take precautions to prevent injuries caused by needles, scalpels, and other sharp instruments or devices during procedures; when cleaning used instruments; during disposal of used needles; and when handling sharp instruments after procedures. To prevent needlestick injuries, needles should not be recapped, purposely bent or broken by hand, removed from disposable syringes, or otherwise manipulated by hand. After they are used, disposable syringes and needles, scalpel blades, and other sharp items should be placed in puncture-resistant containers for disposal; the puncture-resistant containers should be located as close as practical to the use area. Large-bore reusable needles should be placed in a puncture-resistant container for transport to the reprocessing area.
4. Although saliva has not been implicated in HIV transmission, to minimize the need for emergency mouth-to-mouth resuscitation, mouthpieces, resuscitation bags, or other ventilation devices should be available for use in areas in which the need for resuscitation is predictable.
5. Health-care workers who have exudative lesions or weeping dermatitis should refrain from all direct patient care and from handling patient-care equipment until the condition resolves.
6. Pregnant health-care workers are not known to be at greater risk of contracting HIV infection than health-care workers who are not pregnant; however, if a health-care worker develops HIV infection during pregnancy, the infant is at risk of infection resulting from perinatal transmission. Because of this risk, pregnant health-care workers should be especially familiar with and strictly adhere to precautions to minimize the risk of HIV transmission.

Implementation of universal blood and body-fluid precautions for *all* patients eliminates the need for use of the isolation category of "Blood and Body Fluid Precautions" previously recommended by CDC[7] for patients known or suspected to be infected with blood-borne pathogens. Isolation precautions (e.g., enteric, "AFB" [7]) should be used as necessary if associated conditions, such as infectious diarrhea or turberculosis, are diagnosed or suspected.

Precautions for Invasive Procedures. In this document, an invasive procedure is defined as surgical entry into tissues, cavities, or organs for repair of major traumatic injuries 1) in an operating or delivery room, emergency department, or outpatient setting, including both physicians' and dentists' offices; 2) cardiac catheterization and angiographic procedures; 3) a vaginal or cesarean delivery or other invasive obstetric procedure during which bleeding may occur; or 4) the manipulation, cutting, or removal of any oral or perioral tissues, including tooth structure, during which bleeding occurs or the potential for bleeding exists. The universal blood and body-fluid precautions listed above, combined with the precautions listed below, should be the minimum precautions for *all* such invasive procedures.

1. All health-care workers who participate in invasive procedures must routinely use appropriate barrier precautions to

prevent skin and mucous-membrane contact with blood and other body fluids of all patients. Gloves and surgical masks must be worn for all invasive procedures. Protective eyewear or face shields should be worn for procedures that commonly result in the generation of droplets, splashing of blood or other body fluids, or the generation of bone chips. Gowns or aprons made of materials that provide an effective barrier should be worn during invasive procedures that are likely to result in the splashing of blood or other body fluids. All health-care workers who perform or assist in vaginal or cesarean deliveries should wear gloves and gowns when handling the placenta or the infant until blood and amniotic fluid have been removed from the infant's skin and should wear gloves during post-delivery care of the umbilical cord.

2. If a glove is torn or a needlestick or other injury occurs, the gloves should be removed and a new glove used as promptly as patient safety permits; the needle or instrument involved in the incident should also be removed from the sterile field.

Precautions for Dentistry.* Blood, saliva, and gingival fluid from *all* dental patients should be considered infective. Special emphasis should be placed on the following precautions for preventing transmission of blood-borne pathogens in dental practice in both institutional and non-institutional settings.

1. In addition to wearing gloves for contact with oral mucous membranes of all patients, all dental workers should wear surgical masks and protective eyewear or chin-length plastic face shields during dental procedures in which splashing or spattering of blood, saliva, or gingival fluids is likely. Rubber dams, high-speed evacuation, and proper patient positioning, when appropriate, should be utilized to minimize generation of droplets and spatter.
2. Handpieces should be sterilized after use with each patient, since blood, saliva, or gingival fluid of patients may be aspirated into the handpiece or waterline. Handpieces that cannot be sterilized should at least be flushed, the outside surface cleaned and wiped with a suitable chemical germicide, and then rinsed. Handpieces should be flushed at the beginning of the day and after use with each patient. Manufacturers' recommendations should be followed for use and maintenance of waterlines and check valves and for flushing of handpieces. The same precautions should be used for ultrasonic scalers and air/water syringes.
3. Blood and saliva should be thoroughly and carefully cleaned from material that has been used in the mouth (e.g., impression materials, bite registration), especially before polishing and grinding intra-oral devices. Contaminated materials, impressions, and intra-oral devices should also be cleaned and disinfected before being handled in the dental laboratory and before they are placed in the patient's mouth. Because of the increasing variety of dental materials used intra-orally, dental workers should consult with manufacturers as to the stability of specific materials when using disinfection procedures.
4. Dental equipment and surfaces that are difficult to disinfect (e.g., light handles or X-ray-unit heads) and that may become contaminated should be wrapped with impervious-backed paper, aluminum foil, or clear plastic wrap. The coverings should be removed and discarded, and clean coverings should be put in place after use with each patient.

Precautions for Autopsies or Morticians' Services. In addition to the universal blood and body-fluid precautions listed above, the following precautions should be used by persons performing postmortem procedures:

1. All persons performing or assisting in postmortem procedures should wear gloves, masks, protective eyewear, gowns, and waterproof aprons.
2. Instruments and surfaces contaminated during postmortem procedures should be decontaminated with an appropriate chemical germicide.

Precautions for Dialysis. Patients with end-stage renal disease who are undergoing maintenance dialysis and who have HIV infection can be dialyzed in hospital-based or free-standing dialysis units using conventional infection-control precautions.[21] Universal blood and body-fluid precautions should be used when dialyzing *all* patients.

Strategies for disinfecting the dialysis fluid pathways of the hemodialysis machine are targeted to control bacterial contamination and generally consist of using 500–750 parts per million (ppm) of sodium hypochlorite (household bleach) for 30–40 minutes or 1.5%–2.0% formaldehyde overnight. In addition, several chemical germicides formulated to disinfect dialysis machines are commercially available. None of these protocols or procedures need to be changed for dialyzing patients infected with HIV.

Patients infected with HIV can be dialyzed by either hemodialysis or peritoneal dialysis and do not need to be isolated from other patients. The type of dialysis treatment (i.e., hemodialysis or peritoneal dialysis) should be based on the needs of the patient. The dialyzer may be discarded after each use. Alternatively, centers that reuse dialyzers—i.e., a specific single-use dialyzer is issued to a specific patient, removed, cleaned, disinfected, and reused several times on the same patient only—may include HIV-infected patients in the dialyzer-reuse program. An individual dialyzer must never be used on more than one patient.

Precautions for Laboratories.† Blood and other body fluids from *all* patients should be considered infective. To supplement the universal blood and body-fluid precautions listed above, the following precautions are recommended for health-care workers in clinical laboratories.

1. All specimens of blood and body fluids should be put in a well-constructed container with a secure lid to prevent leaking during transport. Care should be taken when collecting each specimen to avoid contaminating the outside of the container and of the laboratory form accompanying the specimen.
2. All persons processing blood and body-fluid specimens (e.g., removing tops from vacuum tubes) should wear gloves. Masks and protective eyewear should be worn if mucous-membrane contact with blood or body fluids is anticipated. Gloves should be changed and hands washed after completion of specimen processing.
3. For routine procedures, such as histologic and pathologic studies or microbiologic culturing, a biological safety cabinet is not necessary. However, biological safety cabinets (Class I or II) should be used whenever procedures are conducted that have a high potential for generating droplets. These include activities such as blending, sonicating, and vigorous mixing.
4. Mechanical pipetting devices should be used for manipulating all liquids in the laboratory. Mouth pipetting must not be done.
5. Use of needles and syringes should be limited to situations in which there is no alternative, and the recommendations

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* General infection-control precautions are more specifically addressed in previous recommendations for infection-control practices for dentistry (ref 8).

† Additional precautions for research and industrial laboratories are addressed elsewhere (refs 22,23).

for preventing injuries with needles outlined under universal precautions should be followed.

6. Laboratory work surfaces should be decontaminated with an appropriate chemical germicide after a spill of blood or other body fluids and when work activities are completed.
7. Contaminated materials used in laboratory tests should be decontaminated before reprocessing or be placed in bags and disposed of in accordance with institutional policies for disposal of infective waste.[24]
8. Scientific equipment that has been contaminated with blood or other body fluids should be decontaminated and cleaned before being repaired in the laboratory or transported to the manufacturer.
9. All persons should wash their hands after completing laboratory activities and should remove protective clothing before leaving the laboratory.

Implementation of universal blood and body-fluid precautions for *all* patients eliminates the need for warning labels on specimens since blood and other body fluids from all patients should be considered infective.

ENVIRONMENTAL CONSIDERATIONS FOR HIV TRANSMISSION

No environmentally mediated mode of HIV transmission has been documented. Nevertheless, the precautions described below should be taken routinely in the care of *all* patients.

Sterilization and Disinfection. Standard sterilization and disinfection procedures for patient-care equipment currently recommended for use[25,26] in a variety of health-care settings—including hospitals, medical and dental clinics and offices, hemodialysis centers, emergency-care facilities, and long-term nursing-care facilities—are adequate to sterilize or disinfect instruments, devices, or other items contaminated with blood or other body fluids from persons infected with blood-borne pathogens including HIV.[21,23]

Instruments or devices that enter sterile tissue or the vascular system of any patient or through which blood flows should be sterilized before reuse. Devices or items that contact intact mucous membranes should be sterilized or receive high-level disinfection, a procedure that kills vegetative organisms and viruses but not necessarily large numbers of bacterial spores. Chemical germicides that are registered with the U.S. Environmental Protection Agency (EPA) as "sterilants" may be used either for sterilization or for high-level disinfection depending on contact time.

Contact lenses used in trial fittings should be disinfected after each fitting by using a hydrogen peroxide contact lens disinfecting system or, if compatible, with heat (78 C-80 C [172.4 F-176.0 F]) for 10 minutes.

Medical devices or instruments that require sterilization or disinfection should be thoroughly cleaned before being exposed to the germicide, and the manufacturer's instructions for the use of the germicide should be followed. Further, it is important that the manufacturer's specifications for compatibility of the medical device with chemical germicides be closely followed. Information on specific label claims of commercial germicides can be obtained by writing to the Disinfectants Branch, Office of Pesticides, Environmental Protection Agency, 401 M Street, SW, Washington, D.C. 20460.

Studies have shown that HIV is inactivated rapidly after being exposed to commonly used chemical germicides at concentrations that are much lower than used in practice.[27-30] Embalming fluids are similar to the types of chemical germicides that have been tested and found to completely inactivate HIV. In addition to commercially available chemical germicides, a solution of sodium hypochlorite (household bleach) prepared daily is an inexpensive and effective germicide. Concentrations ranging from approximately 500 ppm (1:100 dilution of household bleach) sodium hypochlorite to 5,000 ppm (1:10 dilution of household bleach) are effective depending on the amount of organic material (e.g., blood, mucus) present on the surface to be cleaned and disinfected. Commercially available chemical germicides may be more compatible with certain medical devices that might be corroded by repeated exposure to sodium hypochlorite, especially to the 1:10 dilution.

Survival of HIV in the Environment. The most extensive study on the survival of HIV after drying involved greatly concentrated HIV samples, i.e., 10 million tissue-culture infectious doses per milliliter.[31] This concentration is at least 100,000 times greater than that typically found in the blood or serum of patients with HIV infection. HIV was detectable by tissue-culture techniques 1-3 days after drying, but the rate of inactivation was rapid. Studies performed at CDC have also shown that drying HIV causes a rapid (within several hours) 1-2 log (90%-99%) reduction in HIV concentration. In tissue-culture fluid, cell-free HIV could be detected up to 15 days at room temperature, up to 11 days at 37 C (98.6 F), and up to 1 day if the HIV was cell-associated.

When considered in the context of environmental conditions in health-care facilities, these results do not require any changes in currently recommended sterilization, disinfection, or housekeeping strategies. When medical devices are contaminated with blood or other body fluids, existing recommendations include the cleaning of these instruments, followed by disinfection or sterilization, depending on the type of medical device. These protocols assume "worst-case" conditions of extreme virologic and microbiologic contamination, and whether viruses have been inactivated after drying plays no role in formulating these strategies. Consequently, no changes in published procedures for cleaning, disinfecting, or sterilizing need to be made.

Housekeeping. Environmental surfaces such as walls, floors, and other surfaces are not associated with transmission of infections to patients or health-care workers. Therefore, extraordinary attempts to disinfect or sterilize these environmental surfaces are not necessary. However, cleaning and removal of soil should be done routinely.

Cleaning schedules and methods vary according to the area of the hospital or institution, type of surface to be cleaned, and the amount and type of soil present. Horizontal surfaces (e.g., bedside tables and hard-surfaced flooring) in patient-care areas are usually cleaned on a regular basis, when soiling or spills occur, and when a patient is discharged. Cleaning of walls, blinds, and curtains is recommended only if they are visibly soiled. Disinfectant fogging is an unsatisfactory method of decontaminating air and surfaces and is not recommended.

Disinfectant-detergent formulations registered by EPA can be used for cleaning environmental surfaces, but the actual physical removal of microorganisms by scrubbing is probably at least as important as any antimicrobial effect of the cleaning agent used. Therefore, cost, safety, and acceptability by housekeepers can be the main criteria for selecting any such registered agent. The manufacturers' instructions for appropriate use should be followed.

Cleaning and Decontaminating Spills of Blood or Other Body Fluids. Chemical germicides that are approved for use as "hospital disinfectants" and are tuberculocidal when used at recommended dilutions can be used to decontaminate spills of blood and other body fluids. Strategies for decontaminating spills of blood and other body fluids in a patient-care setting are different than for spills of cultures or other materials in clinical, public health, or research laboratories. In patient-care areas, visible material should first be removed and then the area should be decontaminated. With large spills of cultured or concentrated infectious agents in the laboratory, the contaminated area should be flooded with a liquid germicide before cleaning, then decontaminated with fresh germicidal chemical. In both settings, gloves should be worn during the cleaning and decontaminating procedures.

Laundry. Although soiled linen has been identified as a source of large numbers of certain pathogenic microorganisms, the risk of actual disease transmission is negligible. Rather than rigid procedures and specifications, hygienic and common-sense storage and processing of clean and soiled linen are recommended.[26] Soiled linen should be handled as little as possible and with minimum agitation to prevent gross microbial contamination of the air and of persons handling the linen. All soiled linen should be bagged at the location where it was used; it should not be sorted or rinsed in patient-care areas. Linen soiled with blood or body fluids should be placed and transported in bags that prevent leakage. If hot water is used, linen should be washed with detergent in water at least 71 C (160 F) for 25 minutes. If low-temperature (≤70 C [158 F]) laundry cycles are used, chemicals suitable for low-temperature washing at proper use concentration should be used.

Infective Waste. There is no epidemiologic evidence to suggest that most hospital waste is any more infective than residential waste. Moreover, there is no epidemiologic evidence that hospital waste has caused disease in the community as a result of improper disposal. Therefore, identifying wastes for which special precautions are indicated is largely a matter of judgment about the relative risk of disease transmission. The most practical approach to the management of infective waste is to identify those wastes with the potential for causing infection during handling and disposal and for which some special precautions appear prudent. Hospital wastes for which special precautions appear prudent include microbiology laboratory waste, pathology waste, and blood specimens or blood products. While any item that has had contact with blood, exudates, or secretions may be potentially infective, it is not usually considered practical or necessary to treat all such waste as infective.[23,26] Infective waste, in general, should either be incinerated or should be autoclaved before disposal in a sanitary landfill. Bulk blood, suctioned fluids, excretions, and secretions may be carefully poured down a drain connected to a sanitary sewer. Sanitary sewers may also be used to dispose of other infectious wastes capable of being ground and flushed into the sewer.

IMPLEMENTATION OF RECOMMENDED PRECAUTIONS

Employers of health-care workers should ensure that policies exist for:

1. Initial orientation and continuing education and training of all health-care workers—including students and trainees—on the epidemiology, modes of transmission, and prevention of HIV and other blood-borne infections and the need for routine use of universal blood and body-fluid precautions for *all* patients.
2. Provision of equipment and supplies necessary to minimize the risk of infection with HIV and other blood-borne pathogens.
3. Monitoring adherence to recommended protective measures. When monitoring reveals a failure to follow recommended precautions, counseling, education, and/or retraining should be provided, and, if necessary, appropriate disciplinary action should be considered.

Professional associations and labor organizations, through continuing education efforts, should emphasize the need for health-care workers to follow recommended precautions.

SEROLOGIC TESTING FOR HIV INFECTION

Background. A person is identified as infected with HIV when a sequence of tests, starting with repeated enzyme immunoassays (EIA) and including a Western blot or similar, more specific assay, are repeatedly reactive. Persons infected with HIV usually develop antibody against the virus within 6–12 weeks after infection.

The sensitivity of the currently licensed EIA tests is at least 99% when they are performed under optimal laboratory conditions on serum specimens from persons infected for ≥12 weeks. Optimal laboratory conditions include the use of reliable reagents, provision of continuing education of personnel, quality control of procedures, and participation in performance-evaluation programs. Given this performance, the probability of a false-negative test is remote except during the first several weeks after infection, before detectable antibody is present. The proportion of infected persons with a false-negative test attributed to absence of antibody in the early stages of infection is dependent on both the incidence and prevalence of HIV infection in a population (Table I).

The specificity of the currently licensed EIA tests is approximately 99% when repeatedly reactive tests are considered. Repeat testing of initially reactive specimens by EIA is required to reduce the likelihood of laboratory error. To increase further the specificity of serologic tests, laboratories must use a supplemental test, most often the Western blot, to validate repeatedly reactive EIA results. Under optimal laboratory conditions, the sensitivity of the Western blot test is comparable to or greater than that of a repeatedly reactive EIA, and the Western blot is highly specific when strict criteria are used to interpret the test results. The testing sequence of a repeatedly reactive EIA and a positive Western blot test is highly predictive of HIV infection, even in a population with a low prevalence of infection (Table II). If the Western blot test result is indeterminant, the testing sequence is considered equivocal for HIV infection. When this occurs, the Western blot test should be repeated on the same serum sample, and, if still indeterminant, the testing sequence should be repeated on a sample collected 3–6 months later. Use of other supplemental tests may aid in interpreting of results on samples that are persistently indeterminant by Western blot.

Testing of Patients. Previous CDC recommendations have emphasized the value of HIV serologic testing of patients for: 1) management of parenteral or mucous-membrane exposures of health-care workers, 2) patient diagnosis and management, and

TABLE I. Estimated Annual Number of Patients Infected with HIV Not Detected by HIV-Antibody Testing in a Hypothetical Hospital with 10,000 Admissions/Year*

Beginning Prevalence of HIV Infection	Annual Incidence of HIV Infection	Approximate Number of HIV-Infected Patients	Approximate Number of HIV-Infected Patients Not Detected
5.0%	1.0%	550	17–18
5.0%	0.5%	525	11–12
1.0%	0.2%	110	3–4
1.0%	0.1%	105	2–3
0.1%	0.02%	11	0–1
0.1%	0.01%	11	0–1

* The estimates are based on the following assumptions: 1) the sensitivity of the screening test is 99% (i.e., 99% of HIV-infected persons with antibody will be detected); 2) persons infected with HIV will not develop detectable antibody (seroconvert) until 6 weeks (1.5 months) after infection; 3) new infections occur at an equal rate throughout the year; 4) calculations of the number of HIV-infected persons in the patient population are based on the mid-year prevalence, which is the beginning prevalence plus half the annual incidence of infections.

TABLE II. Predictive Values of Positive HIV-Antibody Tests in Hypothetical Populations with Different Prevalences of Infection

	Prevalence of Infection	Predictive Value of Positive Test*
Repeatedly reactive enzyme immunoassay (EIA)	0.2%	28.41%
	2.0%	80.16%
	20.0%	98.02%
Repeatedly reactive EIA followed by positive Western blot (WB)	0.2%	99.75%
	2.0%	99.97%
	20.0%	99.99%

* Proportion of persons with positive test results who are actually infected with HIV.
Assumes EIA sensitivity of 99.0% and specificity of 99.5%.
Assumes WB sensitivity of 99.0% and specificity of 99.9%.

3) counseling and serologic testing to prevent and control HIV transmission in the community. In addition, more recent recommendations have stated that hospitals, in conjunction with state and local health departments, should periodically determine the prevalence of HIV infection among patients from age groups at highest risk of infection.[32]

Adherence to universal blood and body-fluid precautions recommended for the care of all patients will minimize the risk of transmission of HIV and other blood-borne pathogens from patients to health-care workers. The utility of routine HIV serologic testing of patients as an adjunct to universal precautions is unknown. Results of such testing may not be available in emergency or outpatient settings. In addition, some recently infected patients will not have detectable antibody to HIV (Table I).

Personnel in some hospitals have advocated serologic testing of patients in settings in which exposure of health-care workers to large amounts of patients' blood may be anticipated. Specific patients for whom serologic testing has been advocated include those undergoing major operative procedures and those undergoing treatment in critical-care units, especially if they have conditions involving uncontrolled bleeding. Decisions regarding the need to establish testing programs for patients should be made by physicians or individual institutions. In addition, when deemed appropriate, testing of individual patients may be performed on agreement between the patient and the physician providing care.

In addition to the universal precautions recommended for all patients, certain additional precautions for the care of HIV-infected patients undergoing major surgical operations have been proposed by personnel in some hospitals. For example, surgical procedures on an HIV-infected patient might be altered so that hand-to-hand passing of sharp instruments would be eliminated; stapling instruments rather than hand-suturing equipment might be used to perform tissue approximation; electrocautery devices rather than scalpels might be used as cutting instruments; and, even though uncomfortable, gowns that totally prevent seepage of blood onto the skin of members of the operative team might be worn. While such modifications might further minimize the risk of HIV infection for members of the operative team, some of these techniques could result in prolongation of operative time and could potentially have an adverse effect on the patient.

Testing programs, if developed, should include the following principles:

- Obtaining consent for testing.
- Informing patients of test results, and providing counseling for seropositive patients by properly trained persons.
- Assuring that confidentiality safeguards are in place to limit knowledge of test results to those directly involved in the care of infected patients or as required by law.
- Assuring that identification of infected patients will not result in denial of needed care or provision of suboptimal care.
- Evaluating prospectively 1) the efficacy of the program in reducing the incidence of parenteral, mucous-membrane, or significant cutaneous exposures of health-care workers to the blood or other body fluids of HIV-infected patients and 2) the effect of modified procedures on patients.

Testing of Health-Care Workers. Although transmission of HIV from infected health-care workers to patients has not been reported, transmission during invasive procedures remains a possibility. Transmission of hepatitis B virus (HBV)—a blood-borne agent with a considerably greater potential for nosocomial spread—from health-care workers to patients has been documented. Such transmission has occurred in situations (e.g., oral and gynecologic surgery) in which health-care workers, when tested, had very high concentrations of HBV in their blood (at least 100 million infectious virus particles per milliliter, a concentration much higher than occurs with HIV infection), and the health-care workers sustained a puncture wound while performing invasive procedures or had exudative or weeping lesions or microlacerations that allowed virus to contaminate instruments or open wounds of patients.[33,34]

The hepatitis B experience indicates that only those health-care workers who perform certain types of invasive procedures have transmitted HBV to patients. Adherence to recommendations in this document will minimize the risk of transmission of HIV and other blood-borne pathogens from health-care workers to patients during invasive procedures. Since transmission of HIV from infected health-care workers performing invasive procedures to their patients has not been reported and would be expected to occur only very rarely, if at all, the utility of routine testing of such health-care workers to prevent transmission of HIV cannot be assessed. If consideration is given to developing a serologic testing program for health-care workers who perform invasive procedures, the frequency of testing, as well as the issues of consent, confidentiality, and consequences of test results—as previously outlined for testing programs for patients—must be addressed.

MANAGEMENT OF INFECTED HEALTH-CARE WORKERS

Health-care workers with impaired immune systems resulting from HIV infection or other causes are at increased risk of acquiring or experiencing serious complications of infectious disease. Of particular concern is the risk of severe infection following exposure to patients with infectious diseases that are easily transmitted if appropriate precautions are not taken (e.g., measles, varicella). Any health-care worker with an impaired immune system should be counseled about the potential risk associated with taking care of patients with any transmissible infection and should continue to follow existing recommendations for infection control to minimize risk of exposure to other infectious agents.[7,35] Recommendations of the Immunization Practices Advisory Committee (ACIP) and institutional policies concerning requirements for vaccinating health-care workers with live-virus vaccines (e.g., measles, rubella) should also be considered.

The question of whether workers infected with HIV—especially those who perform invasive procedures—can adequately and safely be allowed to perform patient-care duties or whether their work assignments should be changed must be determined on an individual basis. These decisions should be made by the health-care worker's personal physician(s) in conjunction with the medical directors and personnel health service staff of the employing institution or hospital.

MANAGEMENT OF EXPOSURES

If a health-care worker has a parenteral (e.g., needlestick or cut) or mucous-membrane (e.g., splash to the eye or mouth) exposure to blood or other body fluids or has a cutaneous exposure involving large amounts of blood or prolonged contact with blood—especially when the exposed skin is chapped, abraded, or afflicted with dermatitis—the source patient should be informed of the incident and tested for serologic evidence of HIV infection after consent is obtained. Policies should be developed for testing source patients in situations in which consent cannot be obtained (e.g., an unconscious patient).

If the source patient has AIDS, is positive for HIV antibody, or refuses the test, the health-care worker should be counseled regarding the risk of infection and evaluated clinically and serologically for evidence of HIV infection as soon as possible after the exposure. The health-care worker should be advised to report and seek medical evaluation for any acute febrile illness that occurs within 12 weeks after the exposure. Such an illness—particularly one characterized by fever, rash, or lymphadenopathy—may be indicative of recent HIV infection. Seronegative health-care workers should be retested 6 weeks post-exposure and on a periodic basis thereafter (e.g., 12 weeks and 6 months after exposure) to determine whether transmission has occurred. During this follow-up period—especially the first 6–12 weeks after exposure, when most infected persons are expected to seroconvert—exposed health-care workers should follow U.S. Public Health Service (PHS) recommendations for preventing transmission of HIV.[36,37]

No further follow-up of a health-care worker exposed to infection as described above is necessary if the source patient is seronegative unless the source patient is at high risk of HIV infection. In the latter case, a subsequent specimen (e.g., 12 weeks following exposure) may be obtained from the health-care worker for antibody testing. If the source patient cannot be identified, decisions regarding appropriate follow-up should be individualized. Serologic testing should be available to all health-care workers who are concerned that they may have been infected with HIV.

If a patient has a parenteral or mucous-membrane exposure to blood or other body fluid of a health-care worker, the patient should be informed of the incident, and the same procedure outlined above for management of exposures should be followed for both the source health-care worker and the exposed patient.

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Bacterial endocarditis as a complication of acute leukemia

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The cause of fever in a patient with cancer and neutropenia is often unknown, but the fever is usually responsive to broad spectrum antibiotic therapy. Bacterial endocarditis in such patients is a relatively rare entity. In one study of 108 patients at Memorial Sloan-Kettering Cancer Center who had underlying leukemia or lymphoma and bacteremia, there was only one case of subacute bacterial endocarditis.[1] Klastersky et al[2] found that two of 47 patients with solid tumors and fever had endocarditis.

Recently, bacterial endocarditis has been reported as a complication of flow-directed pulmonary artery catheterization resulting from catheter-induced endocardial damage and associated bacteremia.[3] The Hickman/Broviac catheter has been in use for several years as a means of central venous access in patients with cancer. Bacterial infections related to the Broviac catheters have been reported.[4-6] Liepman et al[7] reported three cases of bacterial endocarditis in patients with leukemia, thought to be a complication of an indwelling catheter. We report a case of *Streptococcus viridans* endocarditis of the aortic valve in a leukemic patient with a Broviac catheter.

CASE REPORT

A 35-year-old man was in excellent health until January 1985, when the presence of petechiae was noted; he was subsequently found to be thrombocytopenic. A bone marrow aspiration was performed, and the diagnosis of acute myeloblastic leukemia was made. The physical examination was unremarkable; no heart murmur was noted. There was no history of rheumatic fever or valvular heart disease. A Broviac catheter was placed, and the patient was begun on chemotherapy with 4-DMDR (4-demethoxydanorubicin) and Ara-C (arabinosylcystoscin). He achieved complete remission. His hospital stay was remarkable for fever with neutropenia, negative blood cultures, and resolution of the fever with broad spectrum antibiotic therapy.

In March and May 1985, the patient underwent consolidation chemotherapy with 4-DMDR and Ara-C without complications. On May 20, 1985, after a fever of four days' duration, the patient presented to Memorial Sloan-Kettering Cancer Center, where he was found to be neutropenic, without specific localizing symptoms. He was admitted for therapy. On admission, he had a temperature of 38.5°C, and the Broviac catheter site was slightly erythematous, with no other skin lesions noted. The fundoscopic examination was normal, and the lungs were clear. Cardiac examination revealed a normal S1, S2 without an S3, S4, and a new II/VI systolic murmur at the left lower sternal border without radiation. Laboratory results included hemoglobin, 9.8 g; white blood cell count, 700/mm^3; platelet count, 4,000/mm^3. A chest radiograph was within normal limits, and the electrocardiogram revealed a normal sinus rhythm. Three sets of blood cultures and a urine culture were obtained, and the patient was empirically placed on ticarcillin, 3 g every four hours, vancomycin, 500 mg every six hours, and tobramycin, 120 mg loading dose, then 75 mg every six hours. All blood cultures from both the Broviac catheter and the peripheral blood drawn on admission, prior to antibiotic therapy, grew *Streptococcus viridans*. On May 22, an echocardiogram revealed normal mitral and tricuspid valves. A vegetation was seen on the noncoronary cusp of the tricuspid aortic valve, without evidence of valvular dysfunction (Fig 1). The left atrium and left ventricular chamber size was normal; a small pericardial effusion was noted.

The patient's temperature returned to normal while he was receiving antibiotic therapy and was still neutropenic. On the sixth day of hospitalization, he received two units of packed red blood cells. A dental examination revealed several sites of odontogenic disease; and symptomatic treatment was offered. After ten days on ticarcillin/tobramycin/vancomycin, the patient was no longer neutropenic, and therapy was changed to intravenous penicillin, 3 million units every four hours through the Broviac catheter for a total of four weeks. (The patient completed the antibiotic course at home.) Subsequent blood cultures drawn during and after therapy were negative. The patient remained afebrile, and the physical examination remained unremarkable, with resolution of the heart murmur. On July 25, 1985, a repeat echocardiogram was performed, which demonstrated no change in either the aortic valve vegetation or in valve function or chamber size. Further follow-up by echocardiogram on September 3, 1985, showed complete resolution of the aortic valve vegetation.

DISCUSSION

Bacterial endocarditis in the leukemic patient is uncommon. Roberts et al,[8] in a review of 420 autopsies of the heart in acute leukemia, failed to demonstrate infective endocarditis.

In the patient without an underlying malignancy, bacterial endocarditis is usually dependent on underlying valvular disease. A review of 100 patients with bacterial endocarditis revealed 61 patients with underlying heart disease.[9] The most common organisms that cause acute and subacute infective endocarditis include streptococcal species, *Staphylococcus aureus*, gram-negative organisms, and an increasing number of diptheroids and fungi.[10] The valves usually involved, in order, are the mitral valve, aortic valve, tricuspid, and pulmonary valves. In the intravenous drug addict population, the most common infective organisms include *S aureus*, streptococci (both viridans strains and enterococcus), *Pseudomonas aeruginosa*, gram-negative organisms, and polymicrobial endocarditis.[11] The heart

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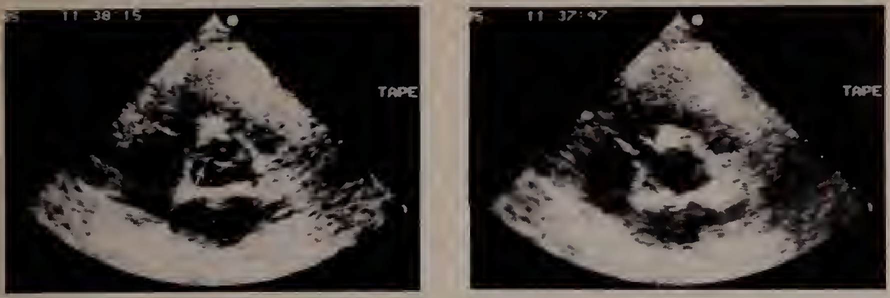

FIGURE 1. Echocardiogram performed at time of diagnosis. **Left**, diastole, arrow indicates valvular lesion on the noncoronary cusp; **Right**, systole.

valve most frequently involved with parenteral drug use is in right-sided endocarditis, and is primarily the tricuspid valve.

Several factors may account for the seldom diagnosed bacterial endocarditis in the leukemic patient. Thrombocytopenia may prevent the formation and propagation of vegetations on the cardiac valves, thereby failing to provide a nidus for bacterial colonization. The frequent initial use of broad spectrum antibiotic therapy in febrile neutropenic patients may also suppress bacterial growth on heart valves and allow early eradication of bacteremia prior to valvular colonization.

Frequently, patients with leukemia or lymphoma present with fever and a "flow" murmur, which is thought to be a reflection of anemia. Those patients with positive blood cultures and a heart murmur should be considered for echocardiography to evaluate the heart for vegetations and possible endocarditis.

In patients with leukemia, the combination of chemotherapy-induced neutropenia, an indwelling Broviac catheter, and frequent self-administration of heparin-flush for catheter patency makes these patients particularly vulnerable to a spectrum of organisms potentially capable of infecting the heart valves. Therefore, until the neutropenia resolves, appropriate broad spectrum antibiotic therapy should be started and continued until adequate marrow recovery is documented. When the neutropenia resolves, specific antibiotic(s) should be used depending on the organisms' sensitivities and the clinical setting.

The treatment for blood-culture-positive endocarditis in patients with leukemia is controversial.[12] As in this case, the patient with left-sided endocarditis does not necessarily require removal of the central venous catheter. Although granulocyte recovery improves survival in such patients, we feel that total treatment should include four weeks of specific therapy directed against the identified organisms, and the Broviac catheter should not be removed unless antibiotic therapy fails and catheter colonization occurs.

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Perirenal hematoma following judo training

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JOJI UTSUNOMIYA, MD

Both acute injury and repeated chronic injury can cause peritoneal hematoma. Eventually the blood clot can disintegrate, and the liquified hematoma may continue to expand. We present a former judo player whose back pain—which had persisted for more than 30 years—stemmed from an extracapsular, perirenal hematoma.

CASE REPORT

A 73-year-old retired policeman complained of mild, chronic, noncolicky pain in the left flank. The pain, which had been present approximately 30 years, had gradually worsened. The patient had been practicing judo since he was in his 20s, but because of increasing back pain he had stopped training in the past two years. Several orthopedists had been consulted but were unable to detect any abnormality. The patient denied any fall or direct trauma to that region except for that incurred during judo training. There were no urinary problems or weight loss.

Microscopic examination of the urine was negative. The patient's blood pressure was

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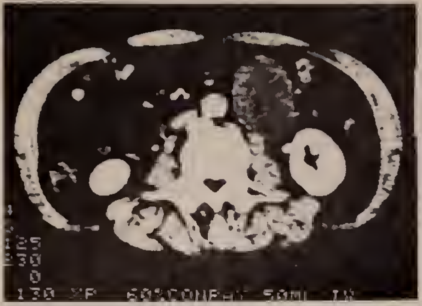

FIGURE 1. Computed tomographic scan showing a large mass adjacent to the left psoas muscle and anteromedial to the left renal pelvis.

140/94 mm Hg; bleeding time, 2 min; coagulation time, 5 min. No anomalies were revealed on a plain radiograph of the abdomen, but abdominal ultrasonography revealed an echolucent mass about 5 cm in diameter just medial to the left kidney. Computed tomography confirmed the presence of a large mass lying adjacent to the left psoas muscle and anteromedial to the left renal pelvis (Fig 1). Left renal arteriography and aortography revealed a slight upward displacement of the renal artery and vein, but there was no irregularity in the left renal filling.

Surgical exploration of the left retroperitoneal space revealed a large hematoma (8 × 7 × 4 cm) lying between the true capsule and the renal fascia. It was slightly adherent to the renal pelvis and was supplied by a small vessel arising from the left renal vein. Only the hematoma was removed (Fig 2). The external surface of the kidney, upper ureter, and renal pelvis appeared normal. The mass was identified by histologic examination as an old, organized, solid hematoma containing yellowish gel.

The postoperative course was uneventful; the patient has been doing well and has remained symptom free for two years. His blood pressure is 130/86 mm Hg.

DISCUSSION

In 1882, judo was established as a formal sport by Jigoro Kano in order to preserve what had hitherto been one of the traditional Japanese martial arts. In 1964, judo was included in the Olympics and has become a major world sport. In Japan, all policemen are required to have a working knowledge of judo, which is regarded as a method of self-defense training. Some injuries from judo are unavoidable. Ankle joint sprains are the most common injury; wrist sprains, shoulder joint dislocation, teeth and mouth injuries, fractures of the ribs, and fractures of the clavicle follow in order of frequency.[1]

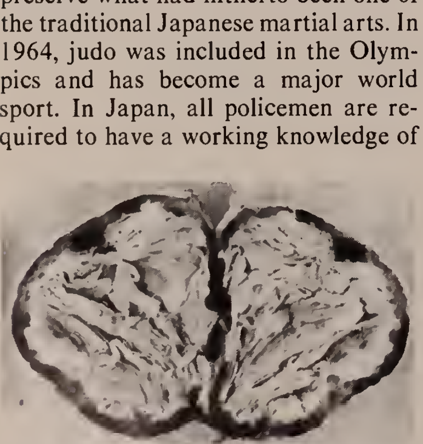

FIGURE 2. Cross-section of surgical specimen.

To prevent these injuries, a method of falling, "ukemi," has been developed so that the opponent is, in most cases, thrown in a circular motion and lands on his back. The art of learning how to fall properly, therefore, lies in the proper landing on one's back without injuring any part of the body.

With progress in judo and the mastery of ukemi, there has been a decreasing incidence of most types of injuries except for lumbar pain; about 60% of the skilled judo players in Japan complain of mild lumbar pain of unknown cause.[2]

In 1982, attention was given to the kidneys of judo players by De Meersman and Wilkerson.[3] An arm throw produced 1,819 joules of compressive force on landing on an ordinary mat, 2.5 cm thick; this resulted in "judo nephropathy"—microscopic hematuria and inhibition of the normal rate of glomerular filtration.[3] Unfortunately, however, most judo players have never undergone periodic urinalysis. In this patient, lumbar pain began approximately 20 years after his first judo training. Because there was no history of trauma to the region, beyond what might have been caused by judo, we feel this lesion is probably related to the frequent trauma of ukemi.

The first authoritative description of perirenal hematoma is attributed to Wunderlich, in 1856.[4] Since that time, numerous case reports and several reviews have been published.[4,5] Perirenal hematoma is either confined by the true capsule of the kidney (subcapsular) or lies between the true capsule and the renal fascia (extracapsular). The latter account for about 80% of these hematomas.[5] Most extracapsular hematomas are found lateral to the kidney and are supplied by a prominent arterial arcade formed within the perirenal fat lateral to the kidney and communicate with the renal branches that perforate the capsule.[6]

Thirteen cases of extracapsular hematoma, called the Antopol-Goldman lesion,[7,8] have been found medial to the kidney. Antopol and Goldman[8] first defined this lesion as a subepithelial hematoma of the renal pelvis and peripelvic tissue, with concomitant hematuria and renal pelvis filling defects visible on radiograph. They suggested that trauma, even though mild, could trigger the onset of the hemorrhage, since the pelvis and hilar blood vessel in the sinus of the kidney are poorly supported by loose connective tissue. In the case described here, an extracapsular hematoma was found medial to the Antopol-Goldman lesion, where anterior and posterior renal fascia fuses with the psoas fascia as it blends with the dense connective tissue.[6] The renal artery and vein perforates through the dense connective tissue to supply the loose connective tissue around the renal pelvis. The vessels supplying the loose connective tissue must tolerate strong shearing forces when the kidney moves suddenly. Both acute injury and repeated chronic injury can cause peritoneal hematoma.[10]

Hematomas that are too large to be absorbed organize into an encapsulated liquified mass having rich vascular beds on the capsule's inner surface. The blood clot within the hematoma disintegrates and becomes completely liquified after several months.[8] The residual liquid develops a characteristic yellow color. A perirenal hematoma may expand by drawing in fluid from the capillary bed of the capsule, as does a subdural hematoma.[9,10] In this case, the hematoma seems to have developed more than 30 years ago, and the back pain gradually worsened.

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Results of modified Martin anoplasty

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Anal stricture is a distressing condition. Patients experience gradually increasing difficulty with bowel movements associated with pain and often bleeding. The inelastic cicatrix is prone to repeated tearing and healing by more scar tissue. Attempts at dilation are not only futile, often they have been the actual cause of the stricture and, in fact, exacerbate the condition. Various plastic surgical procedures have been proposed for the correction of anal stricture. Presented here is a case series of patients treated with modified Martin mucosal advancement anoplasty.

CASE REPORT

Twenty-eight women and 13 men underwent mucosal advancement anoplasty for the correction of anal stenosis over a six-year period from 1979 to 1985. Their ages ranged from 33 years to 86 years; the mean age of the group was 64.4 years. Previous operations on the anorectum, usually a hemorrhoidectomy, were the cause of stenosis in 32 (78%) of the patients. The interval between the initial operation and the treatment for anal stenosis ranged from four months to 32 years; the mean interval was 11.3 years.

All the patients were operated on in jackknife position. The choice of anesthesia was left to the discretion of the anesthesiologist; local anesthesia was not employed. The technique used was similar to one proposed by Martin.[1] An incision was placed in the posterior commissure and carried cephalad within the anal canal deep enough to obtain an adequate lumen to accept a medium-sized Simm vaginal retractor (Fig 1). With the retractor in place, the mucosa was undermined so as to obtain a tension-free anastomosis. The mucosa was advanced to but not beyond the anal verge to avoid ectropion of the mucosa. The scar tissue at the mucosal fringe was excised so as to obtain a pliable tissue for anastomosis. The wound was closed transversely using interrupted mucocutaneous sutures of 3° polyglycolic acid. The sutures were made to include the lower edge of the internal sphincter muscle (Fig 2). This is an extremely important step. It not only anchors the anastomosis and places the anastomosis approximately one third of an inch from the anal verge and within the anal canal but also prevents a keyhole deformity.

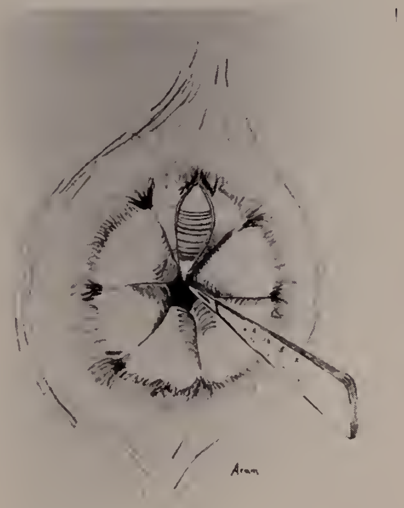

FIGURE 1. The incision is placed exactly in the posterior midline of the anus.

Neither cleansing enemas nor asperients were used before surgery. Similarly, neither drains nor anal packing were used. All patients were allowed to walk and were offered a regular diet on the day of operation. On the first postoperative day, bulk laxatives and warm sitz baths were started. All patients were followed at weekly intervals for from four to six weeks and then at six months after surgery. Anal dilations were not performed.

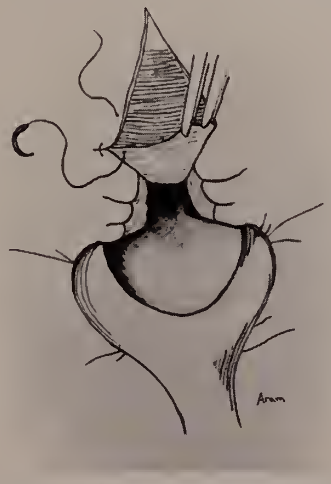

FIGURE 2. The mucocutaneous sutures include the lower edge of the internal sphincter muscle.

The postoperative stay in the hospital ranged from one to six days; the mean stay for the group was 2.3 days. Excluding urinary difficulties, there were no complications during the early postoperative period.

Two patients died during the follow-up period from unrelated causes. The remaining 39 patients were followed for from six months to 68 months; the mean follow-up period was 19.6 months. There was no incidence of overt sepsis or infection during the follow-up period. Anastomotic dehiscence with subsequent healing by granulation occurred in two (5.1%) patients, and partial disruption occurred in three (7.7%) patients. The anastomosis had healed completely in all patients within eight weeks of operation, at which time warm sitz baths were discontinued.

Results were deemed good in 34 (87.1%) patients. These patients were completely free of symptoms and tolerated a standard 19-mm sigmoidoscope without anal discomfort. In three patients the results were judged fair; these patients were relieved of symptoms but experienced anal discomfort during sigmoidoscopy. Finally, in two (5.1%) patients the stenosis recurred and required surgical correction (Table I). There was no incidence of mucosal ectropion or incontinence.

DISCUSSION

Anal stenosis continues to occur in

TABLE I. Results of the Modified Martin Anoplasty in 39 Patients

	Number	Percent
Good	34	87.1
Fair	3	7.7
Poor	2	5.1
Total	39	99.9

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patients following hemorrhoidectomy. Less frequent causes of anal stenosis include trauma and obstetric injuries, certain types of pull-through procedures, Crohn disease, tuberculosis, lymphogranuloma venerum, radiation therapy, overzealous treatment of extensive condylomata acuminata, and senility. An anal stenosis develops following closed hemorrhoidectomy due to compromise to the integrity of the mucocutaneous bridges. Following Whitehead operation, on the other hand, stenosis results from disruption of the anastomosis and subsequent healing of the gap by granulation. The incidence of anal stricture following hemorrhoidectomy is reported to be from 3.5% to 10%.[2,3]

Several plastic surgical procedures have been advocated since the first attempts at the correction of anal stricture by Dieffenbach in 1848.[4] Most of these corrective measures require extensive dissection and are different variants of V–Y, Z, or S-plasty on a handful of patients.[5-13]

Prior to Martin's mucosal advancement technique, the anal stricture was corrected by one or more incisions over the stricture, followed by a regime of anal dilations to maintain a satisfactory lumen. Martin also used one or two incisions followed by suturing the mucosa to the skin with fine catgut. Unfortunately, he did not give any details regarding follow-up of his patients and the incidence of failure following his technique.

Martin anoplasty offers a simple corrective option for the treatment of anal stricture. The mucosal advancement technique makes a tension-free anastomosis possible and precludes excessive scar formation. Inclusion of the lower free edge of the internal sphincter muscle in the anastomotic sutures not only anchors the anastomotic line within the anal canal but also prevents the often-described keyhole deformity of the anus. Sphincterotomy is omitted for fear of incontinence.

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Recommendations on supervision and working conditions of residents

TO THE EDITOR: In collaboration with the hospitals in New York State and the Joint Commission on Accreditation of Hospitals, the New York State Department of Health has restructured its role and responsibilities in quality assurance with the objective of improved patient outcome.

Another aspect of quality assurance that has recently received attention is related to the working conditions and supervision of residents in teaching hospitals. In part to respond to issues regarding inadequacy of supervision of residents raised by the New York grand jury in the investigation of the death of a young person in a teaching hospital, and because these issues are continuing concerns of the health department, the Commissioner of Health, Dr David Axelrod, appointed an advisory committee which was charged to address a variety of issues raised by the grand jury reports. (Committee members include Bertrand M. Bell, MD—Chairman, Benjamin Chu, MD, Robert L. Friedlander, MD, Alfred Gellhorn, MD, Lewis Goldfrank, MD, Paul Griner, MD, Alexander E. Kuehl, MD, Thomas Morris, MD, William Streck, MD.) The committee has transmitted a final report to the commissioner and this will be widely distributed as soon as it has been printed.

The major motivation of the committee's deliberations focused on improving the quality of the medical care of patients in hospital emergency departments and in the in-hospital environment. This focus led to an emphasis on the responsibility of attending physicians to provide mature and skilled supervision for all patients at all times. This applied to the emergency department and the in-patient services. The concern for optimal patient care also directed the committee's attention to changes in the working conditions of residents which would enhance their capacity to deliver critically important medical service and also increase the value of their educational experience. Briefly, the recommendations included the provision of ancillary services (phlebotomy teams, messenger services, transport, etc) around the clock; restriction of continuous service in the emergency department to 12 hours; implementation of a scheduled work week not to exceed an average of 80 hours per week over a four-week period; and a requirement that individual residents should not be scheduled to work as a matter of course for more than 24 consecutive hours, with one 24-hour period of nonworking time per week.

The full report with discussion of the recommendations together with testimony from national and state organizations should be consulted for detailed information.

The commissioner has accepted the report and it is being translated into the language of the Hospital Code for presentation to the Hospital Review and Planning Council. Among the recommendations for consideration by the council will be the requirement that hospitals present plans to meet the requirements by July 1988, with implementation to take place on July 1, 1989.

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Appreciation letter

TO THE EDITOR: I wish to express my sincere thanks and appreciation to the Council of the Medical Society of the State of New York for electing me a life member of the society.

Although retired, I appreciate receiving the *New York State Journal of Medicine* and *News of New York.* There is gratification in keeping abreast of the times.

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Mr Keane's tree

TO THE EDITOR: This is just a note of thanks for your beautiful article, "Mr Keane's tree," which appeared in the September issue of the *Journal.*[1] You painted a warm picture of an interesting character, with a psychological study which is clearly understood by your readers.

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1. Imperato PJ: Mr Keane's tree. *NY State J Med* 1987; 87:509–511.

Paper weight

TO THE EDITOR: "The dedicated physician, according to stereotype, is impatient with any activity that keeps him away from his patients and wants to die with his stethoscope on."[1] My husband, an internist, validates this quote, but presently his stethoscope is being weighted with restrictions, paperwork, and socialized direction.

Primary care physicians are being inundated with paperwork so that they need additional office help to handle it. In Rochester, New York, all three prepaid health care plans (Blue Choice, Preferred Care, Rochester Health Network) have a referral system. In order for a patient to see a specialist, a primary care doctor must be called. The primary care physician is responsible for notifying the patient's health plan. Currently, Blue Choice and Preferred Care permit a telephone call to transmit patient referral information; Rochester Health Network (RHN), however, has insisted on written referrals—in triplicate—signed by the primary care physician. One copy is mailed to RHN, one copy to the named specialist, and one stays in the physician's office. If, for example, six different patients are referred to six different specialists, seven different envelopes (one to RHN and one to each of the six specialists) must be addressed, requiring seven first-class postage stamps, all at the expense of the primary care physician.

Patients' charts needing completion used to be filed in the record room of Rochester General Hospital in a manner that facilitated access. Outstanding patient charts were grouped under the physicians' names, which were arranged in alphabetical order. In 1987 that system was changed so that charts are now filed under six-digit unit numbers. Physicians must now locate their patients' six-digit codes on a master file located at the main desk in the record room. The charts are filed in numerical order on several different shelves. Thus, physicians must now spend additional time to locate every chart before spending the customary few minutes needed to update the records. Although this system makes the hospital's record room more efficient, every doctor has been so inconvenienced by it that Rochester General Hospital recently decided to provide a record room assistant.

Effective July 1, 1987, prescription pads were required to be replaced when the state not only redesigned the forms but actually spelled out the point size to be used for print at the bottom of each blank. The purpose of this was to enable more people to get the benefit of generic drugs, but the program did not allow sufficient lead time for physicians to use up their old prescription pads, which often were bought in quantities to last a year. While the state wants to "hold down medical expenses," as one internist said, "I was stuck with nine months' supply of prescription blanks that are now worthless pieces of paper because they don't have the required box at the bottom."

Also new, the controlled-substance prescription blanks—in triplicate—cost about 25 cents each. Private doctors must pay this charge and cannot pass costs on to patients as fees are frozen. Perhaps anticipating physicians' anger, the state issued a warning that physicians must not stop prescribing these drugs because of this personal expense. Triplicate prescriptions are only good for one month at a time; some patients are permanently in need of controlled substances. Will doctors have to absorb the expense, keeping track, updating charts, and so forth, each month? Will patients presently seen every few months have to pay for a monthly office visit? In Rochester, about 50% or more of primary care physicians' patients are covered by prepaid health plans, so anything that raises costs like this changes doctors' profit pictures without enhancing quality of care.

Prepaid plans take doctors to task for wasting money. Yet, anytime a referral is sent in to Blue Choice, it generates a confirmation referral by computer, with one form to an envelope. One Rochester internist said, "One specific day, I received 17 separate referral forms, in 17 separate envelopes, with 17 separate stamps. Later the same week, I received 21." That means that 38 items went out in just one week to one physician. Even at the 17-cent bulk rate, that represents a considerable waste of money. About five forms make up the minimum weight for bulk rate postage. Why not send out that many with each mailing? With 1,100 doctors in the plan, 400-500 of them primary care physicians, Blue Choice could save perhaps $2,000 a week just by having its workers stuff envelopes! If the computer has been programmed to print one form to a mailing, why can't it be programmed to put all of Dr X's forms together, and send them out five at a time?

Dedicated physicians are being suffocated by paperwork. Health care plans and government policies are forcing doctors to feel "impatient with any activity" that keeps them from practicing clinical medicine in their offices.

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1. Howard RB: Doctors who don't doctor. *Postgrad Med* 1982; 72 (6):13, 16, 19.

Myocardial abscess complicating acute myocardial infarction

TO THE EDITOR: There are occasional reports in the literature of infection of cardiac mural thrombi, generally associated with a poor prognosis. We report a patient with previous myocardial infarction presenting with septicemia caused by *Bacteroides melaninogenicus*, who, based on echocardiography and radionuclide scan, was suspected of having an infected cardiac mural thrombus. During surgery, he was found to have a large myocardial abscess in addition to the infected thrombus. Following surgery, the septicemia resolved, and the patient's condition improved.

Case Report. A 39-year-old man had recently been discharged from the hospital after treatment for a large anteroseptal myocardial infarction, complicated by cardiac arrest. No intracardiac injections were required. During that hospital stay, a low-grade fever was observed. Blood cultures were negative, and the patient was treated for pericarditis with nonsteroidal anti-inflammatory drugs. After discharge, he had several episodes of chest pain, requiring readmission after ten days. On the day prior to his second admission, he had diarrhea, vomiting, fever, chills, and night sweats.

On examination, his temperature was 38.4°C; pulse rate, 84/min; blood pressure, 80/50 mmHg; and respirations, 20/min. The cardiovascular examination was remarkable for a fourth heart sound. His lungs were clear. A presumptive diagnosis of sepsis was made. After obtaining blood samples for culture, antibiotic therapy was instituted with ampicillin, 1 g intravenously every four hours, gentamicin, 80 mg intravenously every 12 hours, and clindamycin, 400 mg intravenously every eight hours.

By the second day of his hospital stay, the patient appeared clinically improved but continued to have spiking fevers in excess of 38.5°C as well as shaking chills. Cultures of urine, sputum, and stool produced no growth. Echocardiography revealed a large apical aneurysm containing a thrombus approximately 20 mm in size. There were some echolucent areas within the thrombus, but no valvular vegetations were seen.

After six days, blood cultures became positive for *Bacteroides melaninogenicus*. Antibiotic therapy was changed to penicillin, 2 million units intravenously every four hours, and metronidazole, 600 mg every six hours. A combination technetium 99 pyrophosphate and gallium citrate scan was done on the sixth hospital day. The technetium scan was consistent with recent myocardial infarction, and the gallium scan showed abnormal labelling of the left ventricular apex, the anterolateral wall, and the interventricular septum (Fig 1). The degree of gallium accumulation was much more prominent than the degree of accumulation of technetium, suggesting the probability of an infected thrombus. Because of persistent fever and gallium enhancement of the left ventricular thrombus without evidence of other source of infection, the patient was subjected to left ventricular aneurysmectomy and thrombectomy.

At operation, the entire anterior ventricular wall was noted to be scarred. The apex of

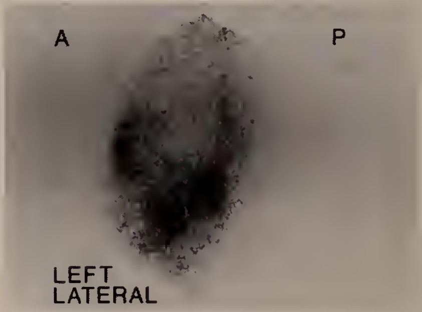

FIGURE 1. Gallium 67 citrate scanning showing abnormal gallium accumulation in the left ventricular apex, anterolateral wall, and the interventricular septum.

the thrombus was necrotic, containing pus. This necrotic area was continuous with a large abscess involving the ventricular wall and the interventricular septum. Cultures of these tissues did not reveal any definitive microorganisms in aerobic or anaerobic media. Postoperatively, antibiotic therapy was continued with penicillin, 2 million units intravenously every four hours, gentamicin, 70 mg intravenously every eight hours, and metronidazole, 500 mg intravenously every six hours for six days. The patient's temperature returned to normal, and further blood cultures were sterile.

Discussion. It is not uncommon for patients with acute myocardial infarction to develop left ventricular thrombi. There is a potential for these thrombi to become infected, progressing to myocardial abscess.[1,2] The prognosis for such patients is extremely poor, with death occurring within a period of a few days to several weeks.[3] However, there have been reports of curative surgery in two instances. Details of these cases are reported elsewhere.[2,4]

Early diagnosis of this entity enhances the success of surgical intervention. Myocardial abscesses generally escape attention because their presence is obscured by an overwhelming generalized sepsis. In fact, the majority of myocardial abscesses are discovered at autopsy. Sanson, Slodki, and Gruhn[5] suggested that a recent surgical procedure was a frequent causative factor in the infection of mural thrombi, but often no primary source is found. The infecting organism in this patient was probably *Bacteroides melaninogenicus*, since this organism was grown from two sets of blood cultures. Culture of the abscess, after surgical excision, was sterile, possibly because of previous antibiotic therapy. *Bacteroides melaninogenicus* is part of the normal oral and gut flora of man. During intubation, at the time of his cardiac arrest, the patient suffered the traumatic loss of two incisor teeth. We suspect that this might have been the source of infection. However, his gastrointestinal symptoms prior to admission suggest a possible bowel source.

Increasingly frequent reports of cardiac infection complicating myocardial infarction should encourage clinicians to consider infected infarcts or infected mural thrombi as a cause of unexplained fever or septicemia. Combined technetium and gallium scanning is useful in confirming the diagnosis. Guidelines for prevention of bacterial endocarditis in patients with certain cardiac disorders have been periodically revised.[6] However, such guidelines have not discussed antibiotic prophylaxis for patients with documented cardiac mural thrombi following acute myocardial infarction. We think it may be worthwhile to consider antibiotic prophylaxis for patients with documented mural thrombi after a recent myocardial infarction when surgical procedures or instrumentation are to be performed. Patients with older infarcts do not appear to have the same risk, because their thrombi are well organized and have healed.

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Acknowledgment. The authors thank Lori Stephenson for typing the manuscript.

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Occupant restraints

Motor vehicle injuries

In the United States, motor vehicle injuries are the number one cause of death for individuals one to 24 years old and the second leading cause of accidental death for those under the age of one (Department of Health and Human Services, 1983).

Disabling injuries (impairments beyond the day of the accident) related to motor vehicle accidents for all age groups numbered 1.7 million nationally in 1985. Costs, including wage loss, medical expense, property damage, and administrative and claim settlement costs of insurance, amounted to $50 billion. Risk factors associated with disabling injuries include alcohol use, speed, age and sex of driver, and nonuse of occupant restraints (National Safety Council, 1985).

The surgeon general has established an objective to reduce the national fatality rate for children under age 15 to 5.5 per 100,000 children by 1990. The 1985 New York State motor vehicle fatality rate for children under 15 was 4.3 per 100,000.

The morbidity and mortality rates for motor vehicle accidents in New York State in 1985 are shown in the accompanying table.

New York State occupant restraint laws

The New York State Child Restraint and Seat Belt Laws, enacted December 1984, require that:

- Children under the age of four must be secured in a federally approved safety seat when riding in passenger cars, station wagons, recreational and passenger vans, and pick-up trucks operated in the state.
- Back seat passengers aged four through nine must use a seat belt. (Depending on a child's size, a safety seat may be used.)
- All front seat drivers and passengers must wear a belt or use a child safety seat (depending on their age).
- The driver of a vehicle being operated in New York State is responsible for the protection of the children riding in that vehicle, even if the vehicle is registered elsewhere.

The penalty for violation of these laws is a fine of up to $50.

Exempt from the seat belt law are emergency vehicles, buses, taxis, liveries, tractors and trucks with a maximum gross weight of 18,000 pounds or more, and vehicle models from 1964 or earlier. Exempt from the child restraint law are emergency vehicles, buses, taxis, motorcycles, and commercial vans.

Also, people with a physically disabling condition can be exempt from the seat belt law, but must obtain and carry with them a physician's certification. A passenger under the age of four whose physical condition prevents the use of a child safety seat is also exempt with a physician's certification. However, most handicapped individuals can and should use a safety belt or seat.

Occupant restraint usage patterns

In New York State, a comparison of 1983 to 1985 occupant restraint usage rates by children up to age three showed an increase from 71% to 82%, 27% to 54% for four- to six-year-olds, and 20% to 48% for seven- to nine-year-olds (Institute for Traffic Safety Management and Research, 1986). In 1986, the New York State Department of Motor Vehicles reported that for children up to four years old, the usage rate increased from 28% before the restraint law (January 1980 to March 1982) to 91% in the first seven months after the effective date. An Insurance Institute for Highway Safety study of high school students in New York State showed 14% used seat belts before enactment of the law and 63% used them in May 1985 (the fifth month after the law).

In a recent evaluation of child passenger safety in the United States, the National Highway Traffic Safety Administration (NHTSA) found the following usage rates in the transition of safety seat and seat belt use as age increases:

under one year of age	68%
1–2 years	62%
2–3 years	51%
3–4 years	27%
4–5 years	17%
5–12 years	25%
12–19 years	12%

Observational surveys by the Governor's Traffic Safety Commission reported that seat belt usage rose from 15%

TABLE. Morbidity and Mortality Rates for Motor Vehicle Accidents, 1985*

Age	Deaths No.	Deaths Rate†	Injuries No.	Injuries Rate†
0–4	28	2.4	4,719	400
5–9	50	4.6	8,487	784
10–14	70	6.1	10,187	888
15–19	251	18.0	35,868	2,567
20–24	335	22.0	44,222	2,898
25–44	584	10.8	97,072	1,791

* New York State Department of Motor Vehicles, 1985.
† Rate per 100,000.

in the prelaw period to 69% after January 1985, but dropped to 57% by April 1985 and to 48.1% by September 1986. Self-reported use of seat belts in low-income families is less than 40% (Baldwin, Fisher, 1986).

A national survey conducted January to June 1986 by Goodell-Grivas for NHTSA showed a 47.1% seat belt usage rate among residents of 19 cities in states with a seat belt law. A comparison survey of cities without a law showed a 23.3% usage rate.

Moreover, in 1985, of the 75,724 New Yorkers involved in an accident and not using an occupant restraint, 644 people (0.9%) were killed and 5,595 (7.4%) experienced serious injury (severe lacerations, broken limbs, skull fractures, internal injuries, and unconsciousness). In comparison, of 195,960 passengers involved in an accident during 1985 while using a lap belt and harness, 197 people (0.1%) were killed and 4,688 (2.4%) experienced serious injury (NYSDMV, 1985). Therefore, the risk of death due to a motor vehicle accident is nine times greater for a passenger not using a restraint, and the risk of serious injury is more than three times greater.

Intervention strategies

Communicating injury prevention measures to health services consumers is essential. Another of the surgeon general's 1990 objectives for the nation states, "all primary health care providers should advise patients about the importance of safety belts and should include instruction about the use of child restraints to prevent motor vehicle-related injuries as part of their routine interaction with parents." Unfortunately, there are no New York State baseline data available to assess the status of this objective.

The importance of promoting occupant restraint use and instructing parents in the proper use of child safety seats cannot be overstated. For those families who use child restraints, as many as 65% use them improperly (Goodell-Grivas, Inc, 1984). As evidenced by the misuse rate of safety seats and the low level of seat belt usage, it is not sufficient for health care providers simply to tell parents they should use occupant restraints for all passengers without providing explicit instructions.

Thorough educational presentations similar to those provided by department-sponsored local child safety seat loaner programs for low-income families in 40 counties can help to promote usage. These presentations include review of the manufacturer's instructions, proper harnessing of the child in the safety seat, correct installation of the safety seat in the vehicle, and promotion of the correct use of seat belts. The parents are required to demonstrate their understanding of these procedures before a safety seat is loaned.

While this type of hands-on educational contact is not always practical for all primary health care providers to undertake, there are several key points that should be offered to parents.

1. A safety seat will protect children only if it is used the right way every time. Always harness children into the safety seat. If there is a shield, make sure that it is in place.
    - *Infant-only safety seats*—Infant safety seats should always be placed facing the rear of the car. This position protects the baby's lower body from harm in a sudden stop or crash.
    - *Convertible seats*—These seats can be used for infants and toddlers. When used for an infant, the seat must face the rear of the car. When used for a toddler, it must face forward.
2. Always use the car seat belt to hold the child safety seat in place on the car seat—even when the child is not in the car. Remember, a sudden stop or crash could cause an unbelted seat to be thrown to the front of the car and cause harm to the passengers.
3. Read and follow the manufacturer's instructions carefully.
4. Always buckle your child—and yourself—even on short trips. The rear seat is the safest for all children. A properly used seatbelt is an important safeguard for pregnant women and their unborn children.

For information on child safety loaner programs for needy families in your area, contact: New York State Department of Health, Injury Control Program, Child Safety Seat Loaner Project, Room 621, Corning Tower, Empire State Plaza, Albany, NY 12237.

The adoption information registry

Created by law in 1983, the New York State Department of Health Adoption Information Registry enables eligible adoptees to obtain nonidentifying information about their parents and the circumstances of their adoption without formally petitioning the courts to open their sealed files. The exchange of identifying information among adoptees, their biological parents, and adoptive parents is also possible through the registry.

Registry participation requires the adoptee to have been born and adopted in New York State and to be at least 21 years of age. Nonidentifying information is provided to the adoptee only and requires only the registration of the adoptee. Identifying information requires the mutual and voluntary registration of all parties to the adoption.

As shown in Table I, nearly two-thirds of the registrants are adoptees. The remaining registrants are almost evenly divided among adoptive and biological parents. Of all parent registrants, mothers represent 74% while fathers represent 26%. Mothers (53%) and fathers (47%) are nearly

TABLE I. Adoption Information Registry Participants*

Type of Registrant	Number
Adoptees	738
Adoptive mothers	100
Adoptive fathers	88
Biological mothers	183
Biological fathers	10
Total	1,119

* As of September 28, 1987.

TABLE II. Adoptee Registrants by Type of Information Requested and Provided*

Type of Information	Number	%	Non-ID Provided	ID Provided
Non-ID only	324	44	304	—
ID and non-ID	414	56	392	2
Total	738	100	696	2

* As of September 28, 1987.

equally represented among adoptive parent registrants, but mothers account for nearly 95% of the biological parent registrants.

Table II shows that 44% of the adoptee registrants are interested in nonidentifying information only. The remainder are interested in both identifying and nonidentifying information. Nonidentifying information has already been provided to 696 adoptee registrants. Data collection is in process for the remaining 42 adoptees.

Nonidentifying information consists of available data recorded at the time of the child's birth and adoption. It is collected from the records of the Department of Health, the court of adoption, and the adoption agency which may have been involved in the adoption. Depending on current availability, potential nonidentifying information may include a description of the birth parents, their race, religion, nationality, ethnic background, physical characteristics, education, occupation, interests or talents, health history and the circumstances relating to the adoption. In some cases, records have been lost or destroyed and only limited information is available.

Identifying information consists of the current names and addresses of the parties involved in the adoption. Table II shows that the registry has been able to provide identifying information to two of the 414 adoptees who have registered for this type of information. Until each corresponding person involved in an adoption voluntarily registers and gives final consent, the registry is not authorized to release identifying information.

If an adoptive parent is deceased, the adoptee may submit a certified death certificate copy in lieu of the deceased parent's registration. In the event that a required birth parent does not voluntarily register, there can be no release of identifying information.

The registry has received registrations from approximately 40 states and numerous foreign countries including Isreal, Korea, Ireland, Germany, and France. To register, an adoptee is charged $75, while adoptive and biological parents pay $20 each. Fees are waived if an individual receives public assistance.

For further information regarding the Adoption Registry, contact: New York State Department of Health, Adoption Information Registry, Corning Tower, Room 208, Empire State Plaza, Albany, NY 12237.

Women and HIV infections

Women are at risk of acquiring many types of infections, including the human immunodeficiency virus (HIV), the virus associated with AIDS. The number of women in New York State diagnosed with AIDS is substantial (1,308 women, or 11% of adult cases reported to the New York State Department of Health as of August 14, 1987). The total number of HIV-infected women is unknown—but is thought to be significantly higher.

Women can acquire HIV infection through intravenous needle sharing or by having sexual contact with an infected person. It is estimated that 80% of the women who are sexual partners of male intravenous drug users do not use intravenous drugs themselves.

The number of women who have contracted AIDS from sexual contact with a person at risk has reached 296 cases, or 22.8% of total female adult and adolescent cases reported in New York State by August 1987.

Pregnant women are able to pass HIV to their unborn children. As of August 1987, 233 pediatric AIDS cases have been reported in New York State. Of those, almost 90% were born to women with AIDS or in a known risk group for AIDS. The growing number of women infected with HIV and their potential for giving birth to infected infants demonstrates an urgent need for preventive education.

During the coming year, the Department of Health's AIDS Institute plans to increase the number and types of education and outreach activities to women, building upon the education and training programs that have been provided for family planning clinics, adolescent pregnancy programs, and prenatal care and nutrition programs across the state.

Recommended precautions for women include:

- Reduce the number of sexual partners.
- Ask your partner about his health, his sexual history, and drug use.
- Use a condom when having sex with a nonmonogamous partner.
- Use a contraceptive foam with nonoxynol-9 in addition to a condom for added protection.
- Do not engage in anal sex.
- Seek out individual HIV counseling and testing if concerned about possible exposure.

The recent New York State Department of Health memorandum directed to physicians who serve sexually active women describes the implications of HIV infection and prevention strategies for women and their children. The document recommends that confidential counseling services and testing for HIV antibody should be offered or made available to pregnant women and sexually active women in the following groups:

- those who have evidence of HIV infection;
- those who have used drugs intravenously for nonmedical purposes;
- those who are or have been sex partners of IV drug abusers, bisexual men, men with hemophilia, men who were born in countries where heterosexual transmission is thought to play a major role, or men who otherwise have evidence of HIV infection.

- those who were born in countries where heterosexual transmission is thought to play a major role.
- those with single or multiple sexual partners whose drug or sexual history is not known.

A copy of the memorandum (Public Health Memorandum #87-15) is available from: Bureau of Communicable Disease Control, New York State Department of Health, Room 651, Corning Tower, Empire State Plaza, Albany, NY 12237.

Parachute jump at Coney Island (Jeffrey Birnbaum, MD). Taken with an Asahi Pentax K-1000 400 ISO.

COLOR ATLAS OF VASCULAR SURGERY

By John S. P. Lumley, MS, MBBS. 350 pp, illustrated. Baltimore, Williams and Wilkins, 1986. $119.00 (hardcover)

Mr John S. P. Lumley of the University of London, working with a photographer, Carole Reeves, managed to produce through painstaking photographic work what should have been the definitive atlas of vascular surgery. I say should have been, because in spite of the excellence of the photographs, there are inherent limitations in the technique which at first encounter would seem to offer a potential for producing the most instructive illustrations of surgical procedures possible. The photographic technique suffers from the fact that often the critical steps in a surgical procedure do not stand out, and that the anatomical landmarks, though reasonably clear to one experienced in the operative procedures, must be terribly difficult to identify for those attempting to learn the procedures from the atlas. The volume would be infinitely more instructive were key photographs and key steps of the procedures accompanied by simple line drawings to identify landmarks and to clearly illustrate technical details which the author rightly emphasizes. As an example, on page 32, there are excellent photographs of end-to-end anastomosis between a dacron prosthesis and an artery, but the critical placement of sutures is not apparent except with prolonged study, and then questions remain. On the other hand, on pages 37 and 38, photographs which demonstrate the fashioning of a vein graft and the dacron prosthesis are incomparable, and the techniques could not be better illustrated than as shown by these photographs.

The volume is well over 300 pages long, covers the major vascular operative procedures performed by vascular surgeons, and illustrates vascular techniques, amputations, lumbar sympathectomy, and microsurgical techniques as well.

At $119 the expenditure might be worthwhile for one experienced in vascular surgery, but for those seeking an introduction to the basics of the field, perhaps line drawings alone would be more instructive.

I would urge the author to consider the addition of suitable line drawings to accompany the excellent photographs. If successful, as I am sure it would be, the result would be a definitive atlas of vascular surgery.

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ATLAS OF CHEST WALL RECONSTRUCTION

By Alan E. Seyfer, MD, Geoffrey M. Graeber, MD, and Gary G. Wind, MD. 260 pp, illustrated. Rockville, Md, Aspen Publishers, 1986. $98.00 (hardcover)

This atlas of chest wall reconstruction is the first dealing specifically with the problems faced by both plastic surgeons and thoracic surgeons. Large numbers of patients are now undergoing coronary artery bypass procedures, and the various problems encountered because of the techniques being used are discussed in detail. The text is extremely readable, easily followed, and well illustrated. There are no photographs. The entire spectrum of chest wall problems is thoroughly discussed, as are the recommendations for reconstruction based on the latest principles in plastic surgery. The book represents a well-organized concert between the specialties of plastic surgery and thoracic surgery. The sections on the preparation of the patient and anatomy are superb for plastic surgeons. Likewise, I believe thoracic surgeons will find the information on myocutaneous flaps and the discussions of the blood supply to the chest wall enlightening. A chapter on breast reconstruction has been included, I believe for the sake of completeness. However, it adds little to the text as plastic surgeons will find ample material elsewhere, and cardiothoracic surgeons, of course, would not find this of other than general interest.

The text does not adequately address the problem of internal mammary artery use for bypass surgery. This is a newer technique, and we are now seeing an increased number of patients with sternal dehiscence for whom there are now fewer choices for myocutaneous flap reconstruction due to the disruption of the choice artery from the internal mammary supply to the superior epigastric artery. I am sure that in the next edition, this problem will be addressed. This textbook is quite important for plastic surgeons who are handling these now common problems and it will give them greater facility in dealing with cardiac surgeons in treating these patients. This atlas should also encourage cardiothoracic surgeons to incorporate plastic surgeons into their teams to help meet the challenges of secondary wound closure.

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ATLAS OF ADVANCED SURGERY

Edited by Martin S. Litwin, MD. 198 pp, illustrated. New York, Yorke Medical Books, 1986. $95.00 (hardcover)

Litwin's *Atlas of Advanced Surgery* is a 198-page blend of clear black-and-white line drawings and concise descriptions of 36 selected operative procedures, all of which are relatively complex gastrointestinal operations. While many of these operations are illustrated with equal clarity in the more complete atlases, *Atlas of Advanced Surgery* has at least one major advantage: its contributors. The contributors are mostly well-known advocates or developers of these procedures, for example, the continent ileostomy by Nils G. Kock, selective splenorenal shunt by W. Dean Warren and William J. Millikan, Jr, intrahepatic cholangiojejunostomy for biliary obstruction by William P. Longmire, Collis gastroplasty by J. L. Collis, and hiatal herniorrhaphy by Ronald Belsey. The illustrations are clear as is the accompanying text. Dr Litwin notes that this is not intended to be a surgical compendium and that its contents are limited to a small number of complicated and difficult general surgical procedures meant for the practicing general surgeon. The book effectively fulfills this description.

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SAVING FACE. A DERMATOLOGIST'S GUIDE TO MAINTAINING A HEALTHIER AND YOUNGER-LOOKING FACE

By Nelson Lee Novick, MD. 258 pp, illustrated. New York, Franklin Watts, Inc, 1986. $16.95 (hardcover)

This book responds to the concern and reflects our population's awareness of the aging process with a focus on the most visible indicators of age and health, the face and scalp. Drawing on his clinical and teaching experiences, the author presents to an intended lay audience a "how to" and "what to expect" volume on maintaining and protecting a healthy face.

The reader first encounters a mixture of popular and scientific terminology on basic cutaneous anatomy and the aging process followed by two major units covering what the individual can do with self effort and what to expect from the physician. Interspersed throughout the text appear little appreciated facts and tidbits of useful trivia like the uselessness of facial muscle isometric exercises to reduce skin sag and the ability of sun to damage swimmers' skin through water transmission.

The chapters on soaps and moisturizers contain explanations and rationales which transcend and decipher some of the commercial barrage of hype. However, when the haze of advertising settles, most individuals choose soaps and moisturizers based on economics and individual preference. Contrary to popular opinion, moisturizers prevent some loss of water from the skin but do not add moisture. Although specific brand names have been inserted to aid the reader, petroleum jelly was not identified as "Vaseline," the most effective moisturizer.

While the explanation of "hypogenic" as applied to cosmetics clarifies the poorly understood issue, cosmetics remains a confusing problem. To "read carefully" cosmetic labeling, the consumer would require a *vade mecum* with lists of chemical names of cosmetic ingredients. And unless there is a specific allergen to be identified, what does one do with this information except wonder if the environment has been further polluted?

Five chapters are appropriately devoted to aspects of hair, including hair care, shaving, too much and too little hair, and the medical approach to ruling out significant disease associated with hair problems. Prices quoted for hair replacement items will not likely apply (happily) in all parts of the country or even in New York in 1987. But hair items might qualify as tax-deductible medical expenses.

Acne therapy is treated in some depth, with attention to misconceptions and aggravating factors. The figures illustrating pathogenesis lack clarity and easy correlation with the accompanying text description. Diagnosis and treatment of benign, premalignant, and malignant facial growths are discussed. Surgical techniques receive adequate attention but the consumer will have difficulty understanding and linking his/her own lesion with the techniques as described and depicted in the accompanying figures.

No discussion of the face is complete without an overview of cosmetic procedures. The author comments on the current attack on facial blemishes and defects as well as his philosophy on major cosmetic procedures and the selection of a plastic surgeon.

Finally, new therapies and approaches loom on the horizon to relieve our cutaneous ills: lasers, injectables for depressed scars and wrinkles, and medications. Already at the time of this writing the anticipated FDA approval of a new vitamin A derivative has been granted and the medication is available.

This work represents an organized approach to disorders of the face and scalp. The text is very readable but marred to some degree by spelling and grammatical errors. The many practical and "how to" activities would be more apparent if printed in bold type in lists and/or summarized following each chapter. A glossary for quick recall of medical terminology would aid the reader. The lay person might have some difficulty with the index, eg, cancer is located under skin cancer. Some of the material is already dated and the use of specific brand names to permit the patient to embark on self care does not allow for the advent of new products or deletion of the old.

Although aimed at the lay public, only a limited number of sophisticated individuals will be able to assimilate and apply this material meaningfully. An expanded audience might include medical students, residents rotating on a dermatology service, and lower level dermatology residents. Even though basic science is by design abbreviated, this volume does provide an extensive overview of face and scalp afflictions, the issues involved, and the patient-physician interaction which Dr Novick has personally experienced and relates with sound judgment and sensitivity.

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MEDICAL MAKEOVER. THE REVOLUTIONARY NO-WILLPOWER PROGRAM FOR LIFETIME HEALTH

By Robert M. Giller, MD, and Kathy Matthews. 276 pp, New York, William Morrow & Co, 1986. $16.95 (clothcover)

The stated purpose of this book is to assist people to feel good, have more energy for work and recreation, look better, and be free of afternoon fatigue, headache, and irritability. The co-authors are Robert M. Giller, MD, a specialist in preventive medicine and family practice, and Kathy Matthews, a writer. Dr Giller has practiced what he terms "Medical Makeover" for some years.

The book is directed to a diverse group of persons who consider themselves generally healthy but who nevertheless suffer from headaches, afternoon fatigue, irritability, overweight, colds, sore throat and other infections, and digestive problems; or persons with a variety of combinations of irregular living habits, including those connected with eating, sleeping, exercising, alcohol consumption, smoking, etc. The authors suggest that all such persons are candidates for an eight-week "Medical Makeover," each week represented as a separate chapter in the book.

Week One deals with caffeine in the diet: where it is found in drinks and drugs, and how to eliminate it. The authors claim this should go far in ridding a person of headache, afternoon fatigue, anxiety, and insomnia. Week Two deals with the importance of eliminating excess sugar from the diet. Week Three discusses the "New Nutrition" which stresses fruits and vegetables, complex carbohydrates, low fat and salt intakes, and a change in protein sources. The importance of a varied diet distributed among regular meals is brought out. The authors state that by spacing meals regularly, one acquires a steady source of energy throughout the day.

Week Four concentrates on vitamins and minerals in the diet and discusses their sources. They are to be used as supplements to a nutritious diet for maintaining optimum health. The minerals, chromium, zinc, copper, and iron, are listed along with their best sources.

Week Five concerns the use of alcohol. Only occasional use in small amounts is considered acceptable. Week Six stresses the importance of regular exercise for good health. Walking is recommended as one of the best and one which fits most easily into most people's lives. Swimming, skiing, and cycling are also considered good by the authors. Week Seven is concerned with stress control, a most important factor in "Medical Makeover" which influences most aspects of health and disease. The authors suggest establishing a set of priorities and then proceeding to one thing at a time to help eliminate stress. Week Eight has to do with smoking, which the authors believe to be "the single worst habit" one can have. It is noted that a small amount of weight may be gained when smoking is stopped, but that this is not much of a price to pay for the benefits realized.

This is a bare summary of the "Medical Makeover" plan. Each of the eight chapters contains many suggestions on how to recognize and deal with the problems faced in establishing a new way of living that will lead to better health. The book is written in a simple, straightforward manner with a personal approach to readers who are drawn into the text by a question-and-answer format which helps in establishing their particular situation. The authors supply an up-to-date and useful list of book and journal references.

There is a chapter which encourages the reader to continue on his/her new way of life. It also tells readers that moderation in some aspects of "Medical Makeover" is acceptable and acknowledges our humanity. As with all programs of this kind, patients should consult their physicians about it first.

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CASARETT AND DOULL'S TOXICOLOGY. THE BASIC SCIENCE OF POISONS

Third Edition. By Curtis D. Klaassen, PhD, Mary O. Amdur, PhD, and John Doull, MD. 974 pp, illustrated. New York, Macmillan Publishing Co, 1986. $49.95 (hardcover)

This multiauthored volume in its third edition continues the format, style, and objectives of the earlier editions. Its primary goal is to serve as a textbook for courses in toxicology or as an adjunct to such courses. That it continues to enjoy a good sales record appears to attest to its success in meeting that objective, but the volume can also be found on the bookshelves of many professionals who have never taught a course in toxicology. Although the text is not intended to serve as a general reference source of information about the toxicology of specific chemical compounds, it is a useful compilation of authoritative reviews about all major aspects of modern toxicology. Of course, few if any teaching programs cover all of these aspects, but the volume is organized into five units, each of which could serve as the basis for a specialty course.

The first unit concerns general principles of toxicology and consists of seven chapters. In addition to topics that are conventionally included among general principles, this section contains separate chapters on chemical carcinogens, teratogens, and genetic toxicology. The second unit is called "Systemic Toxicology;" it is concerned with chemical insults to ten mammalian organs and organ systems and includes three new chapters on toxic responses of the skin, the immune system, and the cardiovascular system. Unit 3 discusses the toxic actions of six classes of synthetic chemicals and natural products, namely pesticides, metals, solvents and vapors, radioactive materials, animal toxins, and plant toxins. A chapter on plastics in the second edition has been omitted. Unit 4 addresses various aspects of environmental toxicology, notably food additives (and contaminants) and pollutants in air, water, and soil. Unit 5 is the shortest unit; it considers applications of the science of toxicology in several health-related areas, including analytic, clinical, occupational, and regulatory toxicology.

Most of the 43 contributing authors in the volume are recognized authorities in their respective fields. Many but not all of the contributions have been significantly revised and updated. However, by continuing a policy of selecting new authors for about one-third of the chapters, the editors have insured the extensive revision of large sections of the text and the introduction of new points of view. They deserve commendation for adopting this strategem of renewal, in spite of the added editorial burdens it must impose. In general, the chapters are informative, comprehensive, and well written. Mechanisms of toxic action continue to be the central theme of most chapters. Few if any of the presentations and discussions are appropriate for undergraduate students. Only science graduates and professionals in biomedical disciplines are likely to have adequate educational backgrounds to profit from these contributions. Although the literature is now extensive and growing rapidly, this reviewer knows of no book so broadly representative of current thinking and practice in toxicology. As such, it continues to deserve the attention of all students and professionals committed to any phase of the science of toxicology.

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MEDICAL JOURNALISM. THE WRITING, DESIGN AND PRODUCTION OF LOCAL MEDICAL, PHARMACEUTICAL AND HOSPITAL PUBLICATIONS

Edited by Craig D. Burrell, MD. 250 pp. New York, Sandoz Corporation, 1986. $15.00 (hardcover)

For a number of years, the Sandoz Corporation has been conducting workshops around the country "designed to advance the cause of good medical journalism." Participants have included those involved in editing and producing some of the several thousand medical and pharmaceutical journals published throughout the United States. Sandoz also publishes a quarterly newsletter, *Medical Journalism*, from which much of the material in this book was drawn. These are very good ideas. The book is not. Its editor should have listened to his contributors.

The book does contain much worthwhile material. There is useful information on writing, editing, design, and production and photography. A lot of information is highly specific to the day-to-day responsibilities of medical editors. On the other hand, various contributors leave no doubt that there is plenty of room for innovation.

The problem is the format because of the kinds of constraints conference transcripts and medical newsletters impose. Just when one wants the material tied into some sort of cohesive whole, the article ends. In the section on writing, for example, various knowledgeable medical journalists provide solid examples of how to improve one's writing style. But each writer comes from a different perspective. After reading and re-reading these, I felt compelled to summarize each article and combine them into an edited whole. Why couldn't the book's editor have done this for me?

The book also contains a number of larger commissioned articles and others drawn from medical journals. While many are informative, others are chatty and express more the personalities of the individuals than tell us things we can do. Interspersed are lead-in comments and separate snippets of information from the editor which distract rather than unify. The book ends with an entirely gratuitous listing of the awardees in the yearly medical journalism contest sponsored by Sandoz. This section accounts for one-fifth of the book.

Perhaps the Sandoz Corporation deserves the right to pat itself on the back. It looks like everyone who has attended one of its conferences was both educated and had fun. I suppose both conference participants and the regular recipients of the newsletter are natural candidates for buying this book. Those of us who are not already tied into basic publications and regularly published magazines for editors may appreciate *Medical Journalism* for the stimulus it provides to sharpen our writing and editing skills. This book will be somewhere down the list for the library of the serious medical writer.

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FROM THE LIBRARY

THE NEW YEAR, 1958

May the New Year be a happy one for all who read these greetings! Since 1807 the Medical Society of the State of New York has seen the new years come, run their course, and retreat into the pages of history. Each year has had its triumphs for medicine and through medicine afforded more opportunity for the pursuit of happiness for the peoples of the world over a longer life span.

Now in this International Geophysical Year we see the beginnings at least of the conquest of the regions beyond the earth's atmosphere. The engineers and physicists have led the way and have proved the validity of their theoretic calculations. What may come of the successful satellite experiments for better or worse no man may yet say. But it seems certain that what one can do, another can do also, and that before long we may expect a plethora of earth satellites and moon rockets transmitting data from outer space.

Next will come the manned satellites, thus bringing the science of medicine into the picture to answer whether man can endure such acceleration and maintain life at such altitudes and temperatures without carbon dioxide suffocation, and the even more compelling question of deceleration. It seems probable that the answers may come more rapidly than has been anticipated. The prospect is a challenging one. And if the challenge be met, then what? Will greater resulting knowledge increase man's wisdom? Improve his ethics? Or merely inflate his ego already swollen to somewhat dangerous proportions?

At the level of the earth's surface the prospects seem good for a further reduction in paralytic poliomyelitis and possibly for influenza, for still further elimination of blindness due to retrolental fibroplasia, and for better understanding of the degenerative diseases to name only a few of the possible areas where productive research is paying off. Can the same progress be noted in the area of human political and social relations? Is the penetration of space now so frenetically attempted and accomplished merely a vast escape mechanism? A flight from unsolved and vexing terrestrial problems? A confession of error and defeat? An admission that risks and dangers of interplanetary space travel are not so terrible or great as the terrestrial horrors of man's inhumanity to man?

The earth satellites of 1957 have been a needed stimulus for a new look at our ideals, our educational methods, our attitude toward pure research in the scientific field, our teaching incentives, and the state of our economy and morals. We seem to have made the elementary tactical error of underestimating the strength and ability of our adversaries. This is humiliating and could well be fatal in an atomic age. It appears we must learn the hard way. The new year, 1958, may necessarily be one of agonizing reappraisal which if relentlessly pursued may—indeed must—place us again in the position of world leadership, or else. . . There is no easy road ahead to that goal. We must either put up or shut up, and that quickly.

EDITORIAL
(*NY State J Med* 1958; 58:40)

BOOKS RECEIVED

AIDS

Living with AIDS and HIV. By David Miller. 136 pp, illustrated. Dobbs Ferry, NY, Sheridan House, Inc, 1987. $14.50 (paperback)

ALLERGY

Insect Allergy. Allergic and Toxic Reactions to Insects and Other Arthropods. Second Edition. By Claude Albee Frazier, MD. 463 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1987. $42.50 (hardcover)

CARDIOLOGY

Learning Electrocardiography. A Complete Course. Third Edition. By Jules Constant, MD. 640 pp, illustrated. Boston, Little, Brown & Co, 1987. $48.00 (clothbound)

Heart Disease. Second Edition. By Earl N. Silber, MD. 1,922 pp, illustrated. New York, Macmillan Publishing Co, Inc, 1987. $120.00 (hardcover)

DIAGNOSTIC MEDICINE

A Pocket Manual of Differential Diagnosis. By Stephen N. Adler, MD, Mildred Lam, MD, and Alfred F. Connors, Jr, MD. 268 pp. Boston, Little, Brown & Co, 1982. $12.00 (paperback)

Textbook of Diagnostic Medicine. Edited by A. H. Samiy, MD. 900 pp, illustrated. Philadelphia, Lea & Febiger, 1987. $69.50 (hardcover)

HEALTH CARE

The Demographics of Physicians: Trends and Projections. 62 pp, illustrated. Chicago, American Medical Association, 1987. $18.00 (paperback)

Practice Management: Problem Solver #1. Edited by Ellen H. Bleiler. 168 pp. Oradell, NJ, Medical Economics, 1987. $14.95 (paperback)

Starting in Medical Practice. By Morton Walker, DPM, with John Parks Trowbridge, MD. 177 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $22.95 (paperback)

Financial Planning Workbook for Physicians. By Steven B. Enright, CFP. 240 pp, illustrated, Oradell, NJ, Medical Economics Books, 1987. $28.95 (paperback)

HEMATOLOGY

Sickle Cell Anemia and Thalassemia. A Primer for Health-Care Professionals. By R. G. Huntsman, MD. 223 pp, illustrated. St John's, Newfoundland, Canada, Canadian Sickle Cell Society, 1987. $10.00 (paperback)

INFECTIOUS DISEASE

Surgical Infectious Diseases. Second Edition. Edited by Richard J. Howard, MD, PHD, and Richard L. Simmons, MD. 913 pp, illustrated. East Norwalk, Conn, Appleton & Lange, 1988. $159.00 (hardcover)

Infective Disease in Primary Care. A Symptomatic Approach. By Robin Hull, MB. 234 pp, illustrated. Dobbs Ferry, NY, Sheridan House, Inc, 1987. $17.50 (paperback)

INTENSIVE CARE MEDICINE

Intensive Care Medicine. Edited by James M. Rippe, MD, Richard S. Irwin, MD, Joseph S. Alpert, MD, and James E. Dalen, MD. 1,203 pp, illustrated. Boston, Little, Brown & Co, 1985. $89.00 (clothbound)

INTERNAL MEDICINE

Internal Medicine. Second Edition. Edited by Jay H. Stein, MD. 2,300 pp, illustrated. Boston, Little, Brown & Co, 1987. $85.00 (clothbound)

Textbook of General Medicine and Primary Care. Edited by John Noble, MD. 2,320 pp, illustrated. Boston, Little, Brown & Co, 1987. $95.00 (clothbound)

Problem-Oriented Medical Diagnosis. Third Edition. By H. Harold Friedman, MD. 431 pp. Boston, Little, Brown & Co, 1983. $19.50 (spiralbound)

MISCELLANEOUS

Make the Most of a Good Thing: You! What You Need to Know About Exercise, Diet, Stress, Sexuality, Relationships and More. By Diana Shaw. 209 pp, illustrated. Boston, Little, Brown & Co, 1987. $6.95 (paperback)

Your Gut Feelings. A Complete Guide to Living Better with Intestinal Problems. By Henry D. Janowitz, MD. 204 pp, illustrated. New York, Oxford University Press, 1987. $15.95 (hardcover)

Becoming a Doctor. A Journey of Initiation in Medical School. By Melvin Konner, MD. 390 pp. New York, Viking Penguin, Inc, 1987. $19.95 (hardcover)

Physician's Guide to the Tax Reform Act of 1986. By Donald L. DeMuth, MBA, CPA, and Daniel C. Miller, MBA, CPA. 152 pp. Oradell, NJ, Medical Economics Books, 1987. $19.95 (paperback)

Heart Attack Prevention. A Practical Approach to Reducing Your Risk of Heart Attack. By Isadore Rosenfeld. New York, Bantam Audio, $7.95 (audio cassette)

We Are Not Alone. Learning to Live with Chronic Illness. By Sefra Kobrin Pitzele. 336 pp, illustrated. New York, Workman Publishing Co, Inc, 1986. $8.95 (paperback)

Doctor Dock. Teaching and Learning Medicine at the Turn of the Century. By Dr Horace W. Davenport. 342 pp, illustrated. New Brunswick, NJ, Rutgers University Press, 1987. $35.00 (cloth)

Dying. Facing the Facts. Second Edition. Edited by Hannelore Wass, PHD, Felix M. Berardo, PHD, and Robert A. Neimeyer, PHD. 472 pp. New York, Hemisphere Publishing Corp, 1988. $49.95 (cloth)

Living with Lung Cancer. A Guide for Patients and Their Families. Second Edition. By Barbara G. Cox, MA, EDD, and David T. Carr, MD. 153 pp, illustrated. Gainesville, Fla, Triad Publishing Co, 1987. $7.95 (paperback)

NUTRITION

Women's Work, Families, and Health. The Balancing Diet. Edited by Kristen M. Swanson-Kauffman, RN. 98 pp, illustrated. New York, Hemisphere Publishing Corp, 1987. $24.50 (hardcover)

OBSTETRICS-GYNECOLOGY

Handbook of Office and Ambulatory Gynecologic Surgery. Edited by Philip D. Darney, MD. 226 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $24.95 (paperback)

Obstetrics and Gynecology Review. By Harrison H. Sheld, MD. 249 pp. New York, Macmillan Publishing Co, 1987. $25.00 (paperback)

Gynecology: Principles and Practice. Edited by Zev Rosenwaks, MD, Fred Benjamin, MD, and Martin L. Stone, MD. 668 pp, illustrated. New York, Macmillan Publishing Co, Inc, 1987. $75.00 (hardcover)

Miracle Babies and Other Happy Endings for Couples with Fertility Problems. How Modern Medical Advances Can Help Couples Conceive. By Mark Perloe, MD, and Linda Gail Christie. 275 pp, illustrated. New York, Penguin Books, 1986. $7.95 (paperback)

ONCOLOGY

Cancer of the Head and Neck. By Stephan Ariyan, MD. 827 pp, illustrated. St Louis, Mo, C.V. Mosby Co, 1987. $125.00 (hardcover)

Primary Care of Cancer. Recommendations for Screening, Diagnosis and Management. By Edward A. Mortimer, Jr, MD. 195 pp. Cleveland, Ohio, Case Western Reserve University School of Medicine, 1987. $14.00 (paperback)

ORTHOPEDIC SURGERY

Evaluating Orthopedic Disability. A Commonsense Approach. Second Edition. 104 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $21.95 (paperback)

PATHOLOGY

Essentials of General Pathology. By John R. Warren, MD, Dante G. Scarpelli, MD, Janardan K. Reddy, MD, and Yashpal S. Kanwar, MD. 259 pp, illustrated. New York, Macmillan Publishing Co, Inc, 1987. $29.95 (paperback)

PEDIATRICS

Your Child's Health. A Pediatric Guide for Parents. By Barton D. Schmitt, MD. 562 pp. New York, Bantam Books, 1987. $12.95 (paperback)

PHYSIOLOGY

Arterial System Dynamics. By John K-J Li. 119 pp, illustrated. New York, New York University Press, 1987. $30.00 (hardcover)

PREVENTIVE MEDICINE

Evaluating Preventive Care. Report on a Workshop. By Louise B. Russell. 107 pp. Washington, DC, Brookings Books, 1987. $22.95 (cloth)

Modern Prevention. The New Medicine. By Isadore Rosenfeld, MD. 431 pp. New York, Bantam Books, 1986. $10.95 (paperback)

PSYCHIATRY

Family Art Psychotherapy. A Clinical Guide and Casebook. By Helen B. Landgarten, MA, ATR. 320 pp, illustrated. New York, Brunner/Mazel Publishers, 1987. $27.50 (hardcover)

The Treatment of Alcoholism. By Edgar P. Nace, MD. 304 pp, illustrated. New York, Brunner/Mazel Publishers, 1987. $30.00 (hardcover)

The Facts About Drugs and Alcohol. By Mark S. Gold, MD. 120 pp. New York, Bantam Books, 1986. $3.50 (paperback)

The Self in the System. Expanding the Limits of Family Therapy. By Michael P. Nichols, PHD. 326 pp. New York, Brunner/Mazel Publishers, 1987. $30.00 (hardcover)

Balance in Motion. Ivan Boszormenyi-Nagy and His Vision of Individual and Family Therapy. By Ammy Van Heusden and Elsemarie Van Den Erenbeemt. 140 pp. New York, Brunner/Mazel Publishers, 1987. $22.50 (hardcover)

Handbook of Measurements for Marriage and Family Therapy. By Norman Fredman, PHD, and Robert Sherman, EDD. 236 pp. New York, Brunner/Mazel Publishers, 1987. $25.00 (hardcover)

Psychiatry and the Mental Health Professionals. New Roles for Changing Times. Formulated by the Committee on Governmental Agencies, Group for the Advancement of Psychiatry. 216 pp. New York, Brunner/Mazel Publishers, 1987. $13.95 (paperback)

Depression Prevention. Research Directions. Edited by Ricardo Munoz. 301 pp, illustrated. New York, Hemisphere Publishing Corp, 1987. $49.95 (hardcover)

SURGERY

Principles of Surgical Technique. The Art of Surgery. Second Edition. By Gary G. Wind, MD, and Norman M. Rich, MD. 280 pp, illustrated. Baltimore, Md, Urban & Schwarzenberg, Inc, 1987. $34.50 (paperback)

TRAUMATOLOGY

Evaluation and Management of Trauma. Edited by Norman E. McSwain, Jr, MD, and Morris D. Kerstein, MD. 470 pp, illustrated. E. Norwalk, Conn, Appleton-Century-Crofts, 1987. $59.95 (hardcover)

UROLOGY

Advances in Urologic Oncology. Volume I. General Perspectives. Edited by Richard D. Williams, MD, 222 pp, illustrated. New York, Macmillan Publishing Co, 1987. $37.50 (hardcover)

Physical activity levels and risk of coronary heart disease

In the course of the Multiple Risk Factor Intervention Trial (MRFIT), the investigators introduced a quantitative leisure-time physical activity (LTPA) questionnaire. Their goal was to determine whether there was a relationship between LTPA and coronary heart disease (CHD) and overall mortality. The MRFIT was a "randomized, multicenter primary prevention trial designed to determine whether multifactor intervention would result in a significant reduction in CHD mortality in middle-aged men who at the time of entry were in the upper 10% to 15% of a risk score distribution derived from Framingham Heart Study data but who had no clinical evidence of CHD." The authors observed that "CHD and overall mortality are inversely related to regular physical exertion, even in men at high risk for CHD" (*JAMA* 1987; 258:2388–2395).

As part of the MRFIT, male volunteers were randomly assigned to either a special intervention (SI) group or a usual care (UC) group. The treatment protocol for the SI group included a dietary program designed to reduce cholesterol levels, a smoking cessation program, and drug therapy to combat hypertension. For this study of LTPA and CHD, the data for the SI and UC groups were pooled, because no significant difference in mortality from CHD was found during the six- to eight-year follow-up period of the MRFIT.

The investigators used the Minnesota questionnaire, which contains 18 major activity groups and 62 individual physical activities, to obtain measurements of LTPA at baseline and at the first, fourth, and sixth annual examinations of the participants. The subjects recorded how often and for how long they performed each activity. The sum of the activities, which are subclassified by intensity as heavy, moderate, and light, comprises the LTPA.

Statistical analysis revealed that "the six-year probability of CHD mortality was inversely related to LTPA level ($P < .001$), primarily caused by differences in levels of cigarette smoking." The duration of LTPA ranged from an average of 16 minutes for the least active group to 134 minutes for the most active men. Only 4.7% of the subjects engaged in an hour or more per week of heavy LTPA—"the recommended quality and quantity of exercise for developing and maintaining physical fitness."

The largest decrease in mortality rates occurred in association with a moderate increase in mainly light- and moderate-intensity LTPA over sedentary levels. Thirty to 69 minutes of such exercise each day was optimal for reducing CHD mortality. The authors concluded that "it is encouraging that predominantly low- to moderate-intensity LTPA [performed for a reasonable period of time each day] appears to be sufficient to reduce premature mortality, since there is the potential risk of sudden cardiac death during heavy exercise in people with coronary risk factors."

Passive smoking and the MRFIT

Although the health risks of smoking cigarettes have been recognized for many years, only recently have the risks to nonsmokers from environmental tobacco smoke been proclaimed. Defined as passive smoking, exposure to another person's tobacco smoke may increase one's risk of developing diseases such as cancer, chronic obstructive pulmonary disease, and, perhaps, heart disease. Svendsen et al (*Am J Epidemiol* 1987; 126:783–795) studied the effects of passive smoking on men in the Multiple Risk Factor Intervention Trial (MRFIT; for a brief description of MRFIT see the previous news brief).

Passive smoking results from inhaling primarily sidestream smoke, which escapes from the lit end of a cigarette, cigar, or pipe, as opposed to mainstream smoke, which the smoker exhales. Sidestream smoke contains more free nicotine and more carbon monoxide than mainstream smoke, as well as several highly carcinogenic components.

At the beginning of the MRFIT, each participant provided a detailed history of his own smoking habits, as well as those of his wives, family members, and coworkers. The investigators then limited their study to evaluating the effects of passive smoking on those men who did not smoke prior to entry into the trial. The results are further broken down between nonsmokers at the time of the trial and men who had never smoked. The smoking status of wives was used as a measure of passive smoking exposure for the men.

Data collected on the men included annual serum thiocyanate levels, which are elevated in smokers due to the cyanide in tobacco smoke, expired carbon monoxide levels (at the third and sixth annual examinations, and the results of annual pulmonary function tests, including the forced expiratory volume in one second (FEV_1).

The mean thiocyanate levels for the two groups were similar. The average expired air carbon monoxide was significantly ($p = 0.001$) higher for men whose wives smoked, as compared to men whose wives did not smoke, although the increase was relatively small, and, the authors conclude, this finding "in and of itself is of relatively little biologic significance." In addition, men whose wives smoked had significantly lower levels of pulmonary function at baseline, based on the measurement of maximum FEV_1.

"Our findings," observe the authors, "support the hypothesis that passive smoking is associated with an increase in morbidity and mortality among nonsmokers. The excess total and coronary heart disease mortality and morbidity among MRFIT men who were exposed to environmental tobacco smoke," they continue, "is further evidence of a potential serious health risk for a large segment of the nonsmoking population."

NEWS BRIEFS are compiled and written by Vicki Glaser.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Victor T. Arnao, MD, Margaretville. Died September 23, 1987; age 59. Dr Arnao was a 1958 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. He was a member of the Delaware County Medical Society and the Medical Society of the State of New York.

Anthony Ricardo Bucalo, MD, Bellerose. Died August 9, 1987; age 65. He was a 1951 graduate of Facolte de Medicine de l'Universite de Lausanne, Lausanne, Switzerland. Dr Bucalo was a member of the American Geriatrics Society, the Aerospace Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Milford Newton Childs, MD, Buffalo. Died September 6, 1987; age 74. He was a 1940 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. Dr Childs was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Sidney Pearl Elpern, MD, Bronx. Died October 11, 1987; age 81. Dr Elpern was a 1930 graduate of New York University School of Medicine, New York. Dr Elpern was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the Bronx County Medical Society, and the Medical Society of the State of New York.

Sidney S. Gaynor, MD, Westport, Indiana. Died October 21, 1987; age 82. Dr Gaynor was a 1930 graduate of New York University School of Medicine, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Chester H. Golding, Jr, MD, Wappingers Falls. Died September 17, 1987; age 69. Dr Golding was a 1943 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a member of the Dutchess County Medical Society and the Medical Society of the State of New York.

Louis Green, MD, Jackson Heights. Died September 19, 1987; age 81. He was a 1937 graduate of Medizinische Fakultaet der Universitaet Basel, Basel, Switzerland. Dr Green was a member of the American Academy of Family Practice, the Bronx County Medical Society, and the Medical Society of the State of New York.

William Cowgill Harrison, MD, Binghamton. Died October 7, 1987; age 76. Dr Harrison was a 1935 graduate of Hahnemann Medical College of Philadelphia, Pennsylvania. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Vincent Aloysius Hawro, MD, Buffalo. Died September 6, 1987; age 83. Dr Hawro was a 1928 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Charles E. R. Hopkins, MD, Jamaica. Died September 16, 1987; age 77. He was a 1936 graduate of Georgetown University School of Medicine, Washington. Dr Hopkins was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. His memberships included the Pan American Association of Ophthalmology, the Pan American Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Leo Karron, MD, Queens Village. Died October 6, 1987; age 75. Dr Karron was a 1942 graduate of Middlesex University School of Medicine, Waltham. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Eugene Andrew Kaskiw, MD, Rome. Died October 7, 1987; age 76. Dr Kaskiw was a 1937 graduate of St Louis University School of Medicine, St Louis, Missouri. He was a member of the Medical Society of the County of Oneida and the Medical Society of the State of New York.

Conrad G. Lattes, MD, New York. Died October 4, 1987; age 50. He was a 1963 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Lattes was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the New York State Surgical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Harry Minassian, MD, Cambridge. Died August 30, 1987; age 31. He was a 1981 graduate of Albany Medical College of Union University, Albany. Dr Minassian was a Diplomate of the American Board of Internal Medicine and a member of the Medical Society of the County of Washington and the Medical Society of the State of New York.

Jacob D. Nahum, MD, Forest Hills. Died October 2, 1987; age 80. Dr Nahum was a 1933 graduate of Friedrich-Wilhelms-Universitaet Medizinische Fakultat, Berlin, Germany. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Lewis Charles Park, MD, Forest Hills. Died September 14, 1987; age 79. He was a 1934 graduate of the University of Tennessee Center for Health Sciences, Memphis. Dr Park was a member of the New York State Society of Internal Medicine, the Clinical Society, the New York Diabetes Association, the Academy of Medicine, the American Society of Internal Medicine, the Association for Advancement of Psychotherapy, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Patrick Francis Pender, MD, Brentwood, Tennessee. Died September 17, 1987; age 81. Dr Pender was a 1933 graduate of Harvard Medical School, Boston. His memberships included the Medical Society of the County of Oneida and the Medical Society of the State of New York.

Leonard S. Rakow, MD, Bronx. Died September 19, 1987; age 78. He was a 1933 graduate of New York University College of Medicine, New York. Dr Rakow was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the Bronx County Medical Society and the Medical Society of the State of New York.

John C. Robin, Buffalo. Died September 2, 1987; age 36. Mr Robin was attending the State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the Medical Society of the State of New York.

James Rudel, MD, Elmhurst. Died October 2, 1987; age 83. Dr Rudel was a 1931 graduate of Charles Univerzita Fakulta of Pediatric-General Medicine, Praha, Czechoslovakia. He was a Diplomate of the American Board of Radiology and a member of the New York Roentgen Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Raymond Francis Smith, MD, Garden City. Died September 19, 1987; age 80. Dr Smith was a 1933 graduate of Jefferson Medical College of Thomas Jefferson University, Philadelphia. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the Pan American Medical Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Nathan Solomon, MD, Maplewood, New Jersey. Died February 6, 1987; age 88. Dr Solomon was a 1925 graduate of New York Medical College, New York. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Jerome Joseph Spitzer, MD, Wantagh. Died September 27, 1987; age 66. He was a 1944 graduate of Tulane University School of Medicine, New Orleans. Dr Spitzer was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Nassau County Medical Society and the Medical Society of the State of New York.

Alfred Morrell Stanley, MD, Adams Center. Died September 3, 1987; age 87. Dr Stanley was a 1922 graduate of the University of Toronto Faculty of Medicine, Toronto, Canada. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the Academy of Medicine, the American Psychiatric Association, the Medical Society of the County of Jefferson, and the Medical Society of the State of New York.

Emery Szanto, MD, Bronx. Died October 15, 1987; age 75. He was a 1939 graduate of Orvosi Fakultas Tudomanyegyetem, Budapest, Hungary. Dr Szanto was a Fellow of the American College of Chest Physicians and a member of the American Thoracic Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Henry A. Wahn, MD, Scotia. Died September 26, 1987; age 83. Dr Wahn was a 1929 graduate of Cornell University Medical College, New York. He was a Fellow of the American College of Surgeons and a member of the Bronx County Medical Society and the Medical Society of the State of New York.

David Weisselberger, MD, New York. Died September 30, 1987; age 81. Dr Weisselberger was a 1935 graduate of Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the American Academy of Child Psychiatry, the Pan American Medical Association, the Academy of Psychoanalysis, the American Society of Adolescent Psychiatry, the American Association of Psychoanalytic Physicians, the New York Medical Society, and the Medical Society of the State of New York.

COURTESY DAVID S. ARSENAULT

Medical Society of the State of New York

ANNUAL CME ASSEMBLY

Newer Approaches to Habit Abuse & Dependency

April 22–24, 1988

and the

Annual Meeting of the House of Delegates

April 21–24, 1988

The New York Hilton
Avenue of the Americas at 53rd/54th Streets
New York City

HIGHLIGHTS . . .

- 25 Sessions
- Panel Discussions
- Technical Exhibits
- Two Receptions
- President's Reception & Dinner Dance

CLIP & MAIL

Director of Front Office Operations
The New York Hilton
1335 Avenue of the Americas
New York, N.Y. 10019

Phone: 212-586-7000

for hotel use only

NAME ______________________ TITLE ______________

FIRM ______________________ PHONE ______________

STREET ______________________________________

CITY/STATE/ZIP ______________________________

REMARKS ______________________________________

(please print)

NOTE: Reservations must be received no later than March 30, 1988 and will be held only until 6 p.m. on day of arrival unless guaranteed.

ARRIVAL DATE ______________________

(check-in time is 3 p.m.)

☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.

DEPARTURE DATE ______________________

(checkout time is 1 p.m.)

HAVE A CREDIT CARD? To take advantage of this credit card PAYMENT GUARANTEED reservation, please complete the information below.

☐ MasterCard
☐ Visa
☐ American Express
☐ Diners Club
☐ Carte Blanche

Expiration Date ______________

____
Credit Card No.

Signature ______________________________

MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
April 21–24, 1988
Annual CME Assembly
April 22–24, 1988

Circle Preferred rate:

Singles: $135 $150 $160
Doubles/Twins: $160 $175 $185
Executive Tower: $205 Single/Double
Suites: (Parlor & 1) $365 $400
Suites: (Parlor & 2) $465 $500

Rates subject to 8¼% N.Y. State sales tax; $2.00 per room per night, and 5% N.Y.C. occupancy taxes. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

GENERAL INFORMATION

Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. Bring your family to New York City to enjoy the many sights.

All physicians, members of the allied professions, and their guests are invited to attend the Medical Society of the State of New York's Annual CME Assembly to be held April 22–24, 1988, at the New York Hilton. The two and one-half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. Over 125 outstanding speakers, including a number from out-of-state, will present papers at 25 sessions.

The theme of the CME Sessions will be "Newer Approaches to Habit Abuse and Dependency". A few of the subjects to be presented will be, Human Abuse Through the Ages: "From the Womb to the Tomb"; Managing Life — Style Abuses of the G.I. Tract; Wake Up! Sleep Apnea Can Kill You; AIDS and Self-Induced Skin Diseases. For details, see the Preliminary Program which appears in the January 1988 issue of the *New York State Journal of Medicine*. Our program will offer the clinician timely approaches in diagnosis and treatment of diseases in a wide range of medical specialties.

Here are comments from some of the attendees at the 1987 CME Assembly: "The best program I have attended in 25 years; "The speakers were stimulating and gave scholarly presentations", "The subjects were timely and practical". "The sessions were well organized and informative".

Professional representatives from more than 75 companies will be in the Rhinelander Gallery Exhibit Hall on Friday and Saturday to discuss their products and services. The Technical exhibits will include drug products, office management systems, medical and surgical suppliers, health insurance plans, pension plans and more.

SPECIAL EVENTS

Thursday, April 21

The Annual Dinner Dance, in honor of our President, Samuel M. Gelfand, M.D., will be held in the Trianon Ballroom of the New York Hilton. The reception will be held in the Mercury Ballroom starting at 7:00 p.m. All attendees are welcome. Tickets are $75.00 per person and should be ordered in advance from the Medical Society.

THE WOOLWORTH BUILDING
— *Courtesy David S. Arsenault*

Friday, April 22 and Saturday, April 23

A reception for physicians, their guests, the allied professions and exhibitors will be held both days in the Exhibit Hall between 5:00 p.m. and 6:00 p.m. A free drawing for a color TV set will take place at both receptions.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than March 30, 1988. After that date, rooms are on an availability basis only. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

SIGHTSEEING

A partial check list of the sights New York City has to offer

- ☐ American Museum of Natural History and Naturemax Theatre
- ☐ Chinatown
- ☐ Citicorp Center
- ☐ Frick Collection of paintings; sculpture; etc.
- ☐ Gray Line Sightseeing— conducted tours via Motorcoach
- ☐ Greenwich Village
- ☐ Hayden Planetarium
- ☐ Lincoln Center for the Performing Arts
- ☐ Metropolitan Museum of Art
- ☐ Museum of Modern Art
- ☐ New York Stock Exchange
- ☐ Radio City Entertainment Center
- ☐ Rockefeller Center
- ☐ South Street Seaport
- ☐ Staten Island Ferry
- ☐ Theatre Museum
- ☐ Trump Tower
- ☐ United Nations Headquarters
- ☐ World Trade Center
- ☐ WW II Aircraft Carrier Intrepid

1988
PRELIMINARY PROGRAM
Annual CME Assembly
April 22–24, 1988

Annual Meeting of the
House of Delegates
April 21–24, 1988

The New York Hilton
New York City

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REGISTRATION & MEETING HOURS

CME Assembly

Registration is open to MSSNY members, spouses, invited guests, non-member physicians, and members of the allied professions. There is a $25 registration fee for MSSNY active members and members of other state medical societies. There is no registration fee for residents/interns, speakers, medical students, medical assistants and spouses. There is a registration fee of $50 for non-member physicians, and a $10 fee for the allied professions.

An advance registration form will be found in this section. For those who register in advance, your identification badge will be held at the CME Registration Desk located in the Rhinelander Gallery Exhibit Hall on the second floor of the hotel. For those who have not registered in advance, registration will take place on Friday, April 22, and Saturday, April 23, from 8:00 a.m. to 5:00 p.m., and on Sunday, April 24, from 8:00 a.m. to 12:30 p.m.

All registrants are required to show identification at the registration desk before a badge is issued. Identification badges are required for admittance to all CME Meetings and exhibits.

CME Meetings will be held on Friday, April 22, and Saturday, April 23, from 8:30 a.m. to 5:00 p.m., and on Sunday, April 24, from 8:30 a.m. to 1:00 p.m.

Delegates

Registration for delegates will take place on the second and third floor Promenades as follows: Wednesday, April 20 from 12 Noon to 6:00 p.m.: Thursday, April 21, Friday, April 22, and Saturday, April 23, from 8:30 a.m. to 5:30 p.m.; and Sunday, April 24, from 8:30 a.m. to 12 Noon.

The 182nd Annual Meeting of the House of Delegates will be called to order at 1:00 p.m. on Thursday, April 21, in the East Ballroom of The New York Hilton. The schedule follows: Thursday, April 21, from 1:00 p.m. to 5:00 p.m.; Saturday, April 23, from 9:00 a.m. to 5:30 p.m.; and on Sunday, April 24, from 9:00 a.m. to 1:00 p.m.

AUXILIARY TO MSSNY

The Auxiliary House of Delegates meeting will take place on Friday, April 22, from 8:30 a.m. to 5:00 p.m, and on Saturday, April 23, from 8:00 a.m. to 11:00 a.m. in the Mercury Ballroom of The New York Hilton.

CME CREDITS

The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. The Medical Society of the State of New York designates all CME activities in this CME Assembly as meeting the criteria for Category 1.

Each program is acceptable for prescribed or elective credits by the American Academy of Family Physicians (AAFP) as designated. The Medical Society's CME credit forms will be issued at the entrance to each meeting room at the opening of each session.

EXHIBITS

Technical exhibits will be located in the Rhinelander Gallery on the second floor of The New York Hilton. Exhibits will be on view Friday, April 22 and Saturday, April 23 from 9:00 a.m. to 6:00 p.m.

PHYSICIAN MESSAGE CENTER

Messages for the physicians attending the CME sessions may be left at the MSSNY Information Desk located in the Exhibit Hall on the second floor. Messages for Delegates may be left at the Delegates Information Desk on the third floor, West Promenade. The New York Hilton telephone number is 212-586-7000.

COFFEE BREAKS

Coffee, sponsored by the Medical Society of the State of New York, will be served in the Exhibit Hall on the second floor daily from 9:00 a.m. to 5:00 p.m. All registered attendees are welcome.

DINNER DANCE

On Thursday, April 21, the Annual Dinner Dance will be held in the Trianon Ballroom, preceded by a reception in the Mercury Ballroom at 7:00 p.m. Tickets should be ordered in advance from the Medical Society of the State of New York. Subscription is $75.00 per person. See the order form in this section.

RECEPTION

A reception, sponsored by the MSSNY, will be held on Friday, April 22 and Saturday, April 23 from 5:00 p.m. to 6:00 p.m. in the Rhinelander Gallery Exhibit Hall located on the second floor of the hotel. All physicians, their spouses, the allied professions and exhibitors are welcome. A free drawing for a color TV will be held at both receptions.

1988 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City

Newer Approaches to Habit Abuse and Dependency

FRIDAY, APRIL 22

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Plenary Session: Human Abuses Through the Ages: "From the Womb to the Tomb," *Sutton South/Regent Parlor*
2:00 p.m. - 5:00 p.m.	Women's Medical Society/Occupational Health, *Gramercy B*
	Cardiovascular Diseases, *Sutton South*
	Physical Medicine & Rehabilitation/Family Practice/Orthepedics, *Gramercy A*
	Emergency Medicine/Critical Care, *Murray Hill B*
	Pathology/Pediatrics, *Bryant Suite*
	General Surgery/Radiology, *Regent Parlor*
	Neurosurgery, *Murray Hill A*
	Medical Liability Mutual Insurance Co., *Rendezvous Trianon*

SATURDAY, APRIL 23

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Internal Medicine (All Day), *Regent Parlor*
	Ophthalmology (All Day), *Sutton South*
	Hand Surgery/Neurology (All Day), *Sutton Parlor North*
	Psychiatry (All Day), *Gramercy B*
	Urology/Radiology, *Nassau A*
	Dermatology, *Murray Hill A*
2:00 p.m. - 3:00 p.m.	Medical Technology
2:00 p.m. - 5:00 p.m.	Internal Medicine, *Regent Parlor*
	Ophthalmology, *Sutton South*
	Hand Surgery/Neurology, *Sutton Parlor North*
	Psychiatry, *Gramercy B*
	Chest Diseases/Otolaryngology, *Murray Hill B*
3:00 p.m. - 5:00 p.m.	Computers in Medicine, *Nassau A*

SUNDAY, APRIL 24

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Allergy & Immunology, *Gramercy B*
	Obstetrics and Gynecology, *Murray Hill A*
	Gastroenterology/Colon and Rectal Surgery, *Nassau A*
	Orthopedics/Physical Medicine & Rehabilitation/Anesthesiology, *Regent Parlor*
	Sleep Disorders—Apnoea, *Nassau B*

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians.

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Technical Exhibits

1988 ANNUAL CME ASSEMBLY

SCIENTIFIC PROGRAM COMMITTEE

STEPHEN NORDLICHT, M.D., New York City, Chairman
PETER B. FARNSWORTH, M.D., Lake Success, Director, Division of Scientific Activities

ASSOCIATE CHAIRMEN

SAMUEL CYTRYN, M.D., Levittown
JOHN DAVIS, M.D., Cooperstown
DAVID HIRSH, M.D., Bronx
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MARILYN KRITCHMAN, M.D., Great Neck
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A. W. MARTIN MARINO, JR., M.D., Brooklyn
MAXWELL SPRING, M.D., Bronx
SIDNEY WEINSTEIN, M.D., Rochester

MEMBERS REPRESENTING SPECIALTY SOCIETIES

Alcoholism

ANNE GELLER, M.D., New York
American Medical Society on Alcoholism—New York Chapter

Allergy & Immunology

YALAMANCHILI A. K. RAO, M.D., Brooklyn

Anesthesiology

KENNETH FREESE, M.D., East Meadow
New York State Society of Anesthesiologists

Cardiovascular Diseases

MOHAMMAD ZAHIR, M.D., Brooklyn
New York Cardiological Society

Chest Diseases

WILLIAM H. BECKER, M.D., Brooklyn
American College of Chest Physicians

Dermatology

LOWELL GOLDSMITH, M.D., Rochester
New York Society of Dermatology

Emergency Medicine

LORRAINE GIORDANO, M.D., Jamaica
New York Chapter of American College of Emergency Physicians

Family and General Practice

MARK KROTOWSKI, M.D., Brooklyn
New York State Academy of Family Physicians

Gastroenterology/Colon & Rectal Surgery

JOHN DAVIS, M.D., Cooperstown
A. W. Martin Marino, Jr., M.D., Brooklyn

Hand Surgery

THOMAS PALMIERI, M.D., New Hyde Park
New York State Society for Surgery of the Hand

Internal Medicine

SIDNEY WEINSTEIN, M.D., Rochester
New York State Society of Internal Medicine

Neurology

RICHARD SATRAN, M.D., Rochester

Neurosurgery

KALMON POST, M.D., New York City

Obstetrics and Gynecology

JOHN PARENTE, M.D., Bronx
American College of Obstetricians and Gynecologists

Occupational & Environmental Health including Workers' Compensation

RICHARD L. KLEIN, M.D., New York City

Ophthalmology

SAMUEL PACKER, M.D., Manhasset
New York State Ophthalmological Society

Orthopedic Surgery

DAVID HIRSH, M.D., Scarsdale
New York Society of Orthopedic Surgery

Otolaryngology

STEVEN PARNES, M.D., Albany
New York State Society of Otolaryngology—Head and Neck Surgery

Pathology

HENRY SIMPKINS, M.D., Staten Island
New York State Society of Pathology

Pediatrics

MARVIN BLUMBERG, M.D., Jamaica
American Academy of Pediatrics, District 11

Physical Medicine and Rehabilitation

SAMUEL SVERDLIK, M.D., New York City
New York Society of Physical Medicine and Rehabilitation

Psychiatry

RICHARD B. DROOZ, M.D., New York City
New York State Psychiatric Association

Radiology

RITA GIROLAMO, M.D., Valhalla
New York State Chapter of the American College of Radiology

Surgery

LESLIE WISE, M.D., New Hyde Park
New York State Society of Surgeons

Urology

PETER J. PUCHNER, M.D., New York City
New York State Urological Society

Women's Medical Society of New York State

LESLIE KOHMAN, M.D., Syracuse
New York State Women's Medical Society

FRIDAY, APRIL 22, 1988

Advances In The Application Of Medical Technology 1

8:30 A.M.–9:30 A.M.
MURRAY HILL B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 1 Hour, Category 1; AAFP, 1 Hour, Prescribed

WILLIAM K. WEISSMAN, M.D., New York City, *Moderator*

President, Theorex Corporation; Adjunct Research Professor, New Jersey Institute of Technology

Objective: To give some examples of mathematical models and computer programs that may be useful in medical research or clinical medicine.

8:30 A.M.–9:30 A.M. **Mathematical And Computational Methods In The Service Of Medicine**

CHARLES PESKIN, Ph.D., New York City

Professor of Mathematics, Courant Institute of Mathematical Sciences, New York University

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Plenary Session
Committee on Bioethical Issues

9:30 A.M.–1:00 P.M.
SUTTON SOUTH/REGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

HUMAN ABUSE THROUGH THE AGES: "FROM THE WOMB TO THE TOMB"

FRED ROSNER, M.D., Jamaica, *Moderator*

Director, Department of Medicine, Queens Hospital Center; Professor of Medicine, Health Sciences Center, State University of New York at Stony Brook

Objective: (1) To present the moral and ethical issues relating to abuse of the fetus, the child, the adolescent, the spouse and the elderly. (2) To enable physicians to recognize, gain an understanding of, and treat or refer for treatment patients with these conditions or members of their families.

9:30 A.M. **Introduction**

FRED ROSNER, M.D., Jamaica

Director, Department of Medicine, Queens Hospital Center; Professor of Medicine, Health Sciences Center, State University of New York at Stony Brook

9:40 A.M. **Fetal Abuse**

HERMAN M. RISEMBERG, M.D., Albany

Professor of Pediatrics (Neonatology), Albany Medical College

10:00 A.M. **Child Abuse**

VINCENT J. FONTANA, M.D., New York City

Medical Director, New York Foundling Hospital; Chairman, Mayor's Task Force on Child Abuse and Neglect of New York City

10:20 A.M. **Adolescent Abuse**

REVEREND BRUCE RITTER, O.F.M. Conv., New York City

President and Founder of Covenant House, New York City

10:40 A.M. **Question and Answer Period**

11:00 A.M. **Coffee Break to View Exhibits**

11:20 A.M. **Spouse Abuse**

LAURENCE LOEB, M.D., Hartsdale, New York

Clinical Associate Professor of Psychiatry, Cornell University Medical College; Senior Consultant in Child Psychiatry and the Law, New York Hospital (Westchester Division)

11:40 A.M. **Elderly Abuse**

ERIC J. CASSELL, M.D., New York City

Clinical Professor of Public Health, Cornell University Medical College; Attending Physician, New York Hospital—Cornell Medical Center, New York City

12:00 P.M. **Wrap-Up**

FRED ROSNER, M.D., Jamaica

(continued)

Women's Medical Society of New York State/Occupational And Environmental Health
Joint Meeting With
New York Occupational Medical Association, Inc.

2:00 P.M.–5:00 P.M.
GRAMERCY B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

PATIENTS, PHYSICIANS, SOCIETY AND SMOKING

LESLIE J. KOHMAN, M.D., Syracuse, *Moderator*
Assistant Professor of Surgery, Women's Medical Society of New York State; President, Women's Medical Society, of New York State

RICHARD L. KLEIN, M.D., New York City, *Moderator*
Director of Employee Health Services, Bristol-Myers Company; Clinical Instructor of Medicine, Cornell University Medical College

Objective: To understand the societal and psychological factors influencing smoking behavior. To learn how health care providers can counteract these influences and to understand how society can act to develop a smoke-free environment.

2:00 P.M. **Butt-Out—The Science of Quitting Smoking**
ALAN LIPSCHITZ, M.D., New York City
Assistant Professor, Department of Psychiatry, New York Medical College; Assistant Director, Outpatient Psychiatry Department, Metropolitan Hospital

2:30 P.M. **A Smoke-Free Society By the Year 2000: What We Can Do To Help**
NANCY RIGOTTI, M.D., Cambridge, Massachusetts
Associate Director of the Institute for the Study of Smoking Behavior and Policy; Instructor in Medicine, Harvard Medical School

3:00 P.M. **Coffee Break to View Exhibits**

3:20 P.M. **Tobacco Advertising And Women**
JEAN KILBOURNE, Ed.D., West Newton, Massachusetts
Internationally known media critic, lecturer and writer; Associate of the Stone Center for Developmental Studies at Wellesley College; Board of Directors of the National Council on Alcoholism

3:50 P.M. **The Politics Of Smoking In Public Places**
RICHARD S. HAMBURG, M.P.A., New York City
Director, Government Affairs, New York Heart Association

4:20 P.M. **Panel Discussion**

Cardiovascular Diseases
Joint Meeting with
New York Cardiological Society

2:00 P.M.–5:00 P.M.
SUTTON PARLOR SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

THE HEART AND HABIT ABUSE

MARY ALLEN ENGLE, M.D., New York City, *Moderator*
Director of Pediatric Cardiology, New York Hospital; Professor of Pediatric Cardiology, Cornell Medical College

Objective: Physicians will gain information enabling them to recognize and manage the effects of habit abuse on the heart.

2:00 P.M. **The Heart and Ethanol**
LAWRENCE A. GOULD, M.D., Brooklyn
Acting Director of Medicine; Chief of Cardiology, Methodist Hospital of Brooklyn

2:40 P.M. **The Heart and Cocaine**
ALAN F. LYON, M.D., Brooklyn
Director of Medicine, Brookdale Hospital; Professor of Medicine, SUNY Health Science Center of Brooklyn

3:20 P.M. **Coffee Break to View Exhibits**

3:40 P.M. **Sedentary Living and The Heart**
RICHARD STEIN, M.D., Brooklyn
Chief of Cardiology; Associate Professor of Medicine, SUNY Health Science Center of Brooklyn

4:20 P.M. **Endocarditis and Drug Abuse**
RANDOLPH CHASE, JR., M.D., New York City
Associate Professor of Medicine, New York University; Director of Microbiology, University Hospital

(continued)

Emergency Medicine/Critical Care
Joint Meeting with
New York Chapter of American College of Emergency Physicians

2:00 P.M.–5:00 P.M.
MURRAY HILL B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

TOXIDROMES, RELATED ILLNESSES AND ETHICAL ISSUES OF THE POISONED PATIENT

LORRAINE M. GIORDANO, M.D., FACEP, Jamaica, *Moderator*

Chairperson, Department of Emergency Medicine, Jamaica Hospital, Jamaica, N.Y.

Objective: This program will review common toxidromes, common substance abuse poisoning, respiratory problems of the drug overdose patient, alcohol-related illnesses such as AIDS occurring in the emergency room. Physicians attending this program will be able to recognize these problems and manage them appropriately.

2:00 P.M. **An Approach To The Poisoned Patient—Common Toxidromes And Their Management**

DIANE SAUTER, M.D., New York City

Associate Medical Director, N.Y.C. Poison Control Center; Attending Physician, Bellevue Hospital, New York City

2:40 P.M. **Acute Respiratory Problems In The Drug Overdose Patient**

CARLOS R. ORTIZ, M.D., Rochester

Associate Professor of Medicine, University of Rochester School of Medicine and Dentistry; Medical Director, Intensive Care Unit, The Genessee Hospital, Rochester

3:20 P.M. **Coffee Break to View Exhibits**

3:40 P.M. **Alcohol Related Illnesses Presented to Emergency Department—Presentations, Approaches, And Therapeutic Options**

TIMOTHY SHOEN, M.D., Saratoga Springs

Attending Physician, Emergency Department, Saratoga Hospital; Staff Physician, Saratoga County Alcoholism Services

4:20 P.M. **Ethical Issues Relating To Substance Abuse Patients In The Emergency Department**

Joint Presentation:

NEAL FLOMENBAUM, M.D., FACEP, New York City

Director of Emergency Medicine, Long Island College Hospital; Assistant Professor Of Clinical Medicine, New York University Medical School and

KEVIN PORTER, Esq., New York City

Bower and Gardner

Pathology/Pediatrics
Joint Meeting With
American Academy of Pediatrics—District 11/New York State Society Of Pathologists

2:00 P.M.–5:00 P.M.
BRYANT SUITE

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

INTERDISCIPLINARY MANAGEMENT OF YOUTHFUL SUBSTANCE ABUSE

MARVIN L. BLUMBERG, M.D., Jamaica, *Moderator*

Chairman, Department of Pediatrics, The Jamaica Hospital; Associate Professor of Clinical Pediatrics, State University of New York at Stony Brook

Objective: To make physicians aware of the signs and symptoms of drug and alcohol abuse, the pre-disposing causes, the physiological and emotional effects and the medical and psychiatric treatment.

2:00 P.M. **The Pediatrician's Role In The Management Of Drug Abuse**

MICHAEL NUSSBAUM, M.D., New Hyde Park

Staff Physician, Division of Adolescent Medicine, Schneider Children's Hospital, New Hyde Park; Assistant Professor of Pediatrics, State University of New York at Stony Brook

2:30 P.M. **Psychiatric Aspects Of Drug And Alcohol Abuse In Youth**

HAROLD S. KOPLEWICZ, M.D., New Hyde Park

Chief of Child and Adolescent Psychiatry, Schneider Children's Hospital, New Hyde Park; Assistant Attending in Psychiatry, New York Psychiatric Hospital

(continued)

3:00 P.M. **Coffee Break to View Exhibits**

3:20 P.M. **Adolescents And Drug Abuse**

RAYMOND GAMBINO, M.D., Englewood Cliffs, New Jersey

Chief Medical Officer—Met Path Labs; Adjunct Professor of Pathology, Columbia University

3:50 P.M. **Concepts And Conundrums Of HIV Infection In Children**

KEITH KRASINSKI, M.D., New York City

Assistant Professor of Pediatrics, New York University Medical Center

4:20 P.M. **Questions and Answers**

General Surgery/Radiology
Joint Meeting With
NYS Society of Surgeons/NYS Chapter of the American College of Radiology

2:00 P.M.–5:00 P.M.
REGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

NEW TECHNOLOGIES IN GENERAL SURGERY AND RADIOLOGY

ERIC MUNOZ, M.D., M.B.A., F.A.C.S., New Hyde Park, *Moderator*

Head of Research Division, and Assistant Professor of Surgery Technology Assessment and Medicine, Long Island Jewish Medical Center, New Hyde Park

Objective: Physicians will gain information on new and changing technologies in surgery and radiology and their application in practice.

2:00 P.M. **Technology Assessment and Medicine**

ERIC MUNOZ, M.D., M.B.A., F.A.C.S., New Hyde Park

Head of Research Division, and Assistant Professor of Surgery Technology Assessment and Medicine, Long Island Jewish Medical Center, New Hyde Park

2:15 P.M. **New Technologies and General Surgery**

LESLIE WISE, M.D., F.A.C.S., New Hyde Park

Chairman, Department of Surgery, Long Island Jewish Medical Center; Professor of Surgery, State University of New York

2:40 P.M. **Technology and Surgical Endoscopy**

HOUSTON JOHNSON, M.D., New Hyde Park

Assistant Professor of Surgery, State University of New York, (Stony Brook); Attending Surgeon, Long Island Jewish Medical Center, New Hyde Park

3:05 P.M. **Coffee Break to View Exhibits**

3:25 P.M. **Technology and Vascular Surgery**

JON R. COHEN, M.D., New Hyde Park

Assistant Professor of Surgery, State University of New York, (Stony Brook); Attending Vascular Surgeon, Long Island Jewish Medical Center, New Hyde Park

3:55 P.M. **Nuclear Magnetic Resonance (NMR) of the Spine**

DENISE LESLIE, M.D., Valhalla

Assistant Professor of Radiology, New York Medical College

4:25 P.M. **Nuclear Magnetic Resonance (NMR) of the Vascular and Musculoskeletal System**

ANDREW SCHECHTER, M.D., Valhalla

Assistant Professor of Radiology; Director of Magnetic Resonance Imaging.

Physical Medicine and Rehabilitation/Family Practice/Orthopedic Surgery
Joint Meeting with
The New York Society of Physical Medicine and Rehabilitation/New York State Academy of Family Physicians/New York Society of Orthopedic Surgery

2:00 P.M.–5:00 P.M.
GRAMERCY A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

EVALUATION AND MANAGEMENT OF ADOLESCENTS FOR PARTICIPATION IN ATHLETIC ACTIVITIES

AUDREY RANDOLPH, M.D., Valhalla, N.Y., *Moderator*

Department of Rehabilitation Medicine, Westchester County Medical Center Westchester, N.Y.

Objective: To define the parameters of the cardiopulmonary musculoskeletal systems that should be met, to insure that children engaging in athletics are not at risk.

(continued)

2:00 P.M. **Family Practitioner**

Richard B. Birrer, M.D., Danville, PA.

Chairman, Department Family Practice, Geisinger Medical Center, Danville, PA.; Associate Professor of Family Practice, SUNY Health Science Center, Brooklyn, New York

2:30 P.M. **Cardiopulmonary Evaluation**

Filippo A. Balboni, M.D., Roslyn

Director, Pediatric Cardiology, St. Francis Hospital, Roslyn; Associate Professor of Clinical Pediatrics, State University of New York at Stonybrook

2:55 P.M. **Physiatric Evaluation**

David Ernstoff, M.D., Flushing, N.Y.

Director, Rehabilitation Medicine/Sports Medicine Program, Booth Memorial Medical Center, Flushing; Assistant Clinical Professor of Rehabilitation Medicine, New York University College of Medicine, New York City

3:20 P.M. **Coffee Break To View Exhibits**

3:40 P.M. **Orthopedic Evaluation**

David Mensche, M.D., New York City

Attending Orthopedics, Hospital for Joint Disease, New York City; Assistant Director, Sports Medicine Program, Booth Memorial Medical Center, Flushing, New York

4:10 P.M. **Scoliosis In The Adolescent Athlete**

Stanley Hoppenfeld, M.D., New York City

Chairman, Department Family Practice Geisinger Medical Center, Danville, PA.; Associate Professor of Family Practice, SUNY Health Science Center, Brooklyn, New York

4:55 P.M. **QUESTION AND ANSWER PERIOD**

Neurosurgery
Joint Meeting with
New York State Neurosurgical Society

2:00 P.M.–5:00 P.M.
MURRAY HILL A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

NEUROSURGICAL UPDATE

Kalmon D. Post, M.D., New York City, *Moderator*

Professor of Neurological Surgery, College of Physicians and Surgeons, Columbia University, New York City

Objective: Review in depth the modern concepts and treatment of Aneurysms

2:00 P.M. **Introduction**

Kalmon D. Post, M.D., New York City

Professor of Neurological Surgery, College of Physicians and Surgeons, Columbia University, New York City

2:10 P.M. **Management Of Subarachnoid Hemorrhage**

Robert A. Solomon, M.D., New York City

Assistant Professor of Neurological Surgery, College of Physicians and Surgeons, Columbia University, New York City

2:55 P.M. **Complications of Aneurysm Surgery**

Nicholas Hopkins, M.D., Buffalo

Clinical Associate Professor of Neurological Surgery, State University of New York at Buffalo

3:40 P.M.–4:00 P.M. **Coffee Break To View Exhibits**

4:00 P.M. **Open Papers**

Medical Liability Mutual Insurance Company
Joint Meeting With The
Medical Society of the State of New York

2:00 P.M.–5:00 P.M.
RENDEZVOUS TRIANON

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

MEDICAL LIABILITY; PATIENT-PHYSICIAN COMMUNICATIONS WITH CASE PRESENTATIONS

Ralph M. Schwartz, M.D., New York City, *Moderator*

Associate Professor, Clinical Obstetrics and Gynecology, State University of New York Downstate Medical Center; Chairman, Patient Safety Committee, Medical Liability Mutual Insurance Company

Objective: The purpose of this seminar is to provide physicians in attendance with information about professional liability in the practice of medicine. It will deal with cases regarding patient-physician communications.

(continued)

2:00 P.M. **Communications As It Relates to Physicians' Liability**

DAVID H. SMITH, Ph.D., Tampa, Florida

Professor and Chief of Human Values Division, Department of Comprehensive Medicine, University of Southern Florida, College of Medicine

3:00 P.M. **Coffee Break to View Exhibits**

3:20 P.M. **Case Presentations:**

Legal Implications-Defense

EDWARD J. AMSLER, Esq., New York City

Assistant Secretary, Medical Liability Mutual Insurance Co.; Partner, Fager and Amsler

and

PAUL RHEINGOLD, Esq.

Partner, Rheingold and Golomb, New York City

4:05 P.M. **Physicians Panel**

SATURDAY, APRIL 23, 1988

Advances in the Application of Medical Technology #2

8:30 A.M.–9:30 A.M.
MURRAY HILL B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 1 Hour, Category 1; AAFP, 1 Hour, Prescribed

WILLIAM K. WEISSMAN, M.D., New York City, *Moderator*

President, Theorex, Corporation; Adjunct Research Professor, New Jersey Institute of Technology

Objective: LASER: A device that produces light by a process of Light Amplification by the Stimulated Emission of Radiation.

ELECTRO-OPTICS: Describes the behavior of light in the presence of electric and magnetic fields in materials. The physician will gain an understanding of these fundamental physical principles, as applied to human use with an aim to expanding their clinical utility.

8:30 A.M.–9:30 A.M. **Lasers and Electro-optics: Principles And Medical Applications**

MALVIN CARL TEICH, Ph.D., New York City

Professor of Engineering Science, Columbia University Department of Electrical Engineering and Applied Physics

Hand Surgery/Neurology
Joint Meeting With
The New York Society for Surgery of the Hand/New York Neurological Society

9:30 A.M.–5:00 P.M.
SUTTON PARLOR NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 6 Hours, Prescribed

NEUROLOGICAL DISORDERS OF THE UPPER EXTREMITY

THOMAS J. PALMIERI, M.D. New Hyde Park, *Moderator*

Staff Surgeon, North Shore University Hospital, Manhasset, New York; Physician-in-charge Hand Surgery, Long Island Jewish-Hillside Medical Center, New Hyde Park; Assistant Professor of Clinical Surgery, State University of New York at Stony Brook

Objective: To provide the internist, neurologist, primary care physician, orthopedic, plastic and hand surgeon, as well as therapists, nurses, and paramedical personnel with an update on the dilemmas and controversies in treating patients with neurological disorders of the upper extremity, and in particular, the hand and wrist. Included will be the etiology, diagnosis, and pathophysiology of the various neuropathies affecting the upper extremity, as well as the medical and surgical treatments which are currently available.

9:30 A.M. **Clinical Examination, Evaluation and Differential Diagnosis of Upper Extremity Neuropathies (Including Syndromes of Nerve Loss)**

CHARLES P. MELONE, JR., M.D., New York City

Associate Professor of Orthopedic Surgery, New York University Medical Center; Director of Hand Surgery, Cabrini Medical Center, New York City

10:00 A.M. **Electrophysiological Testing of Peripheral Nerve Disorders**

CHARULATA BADLANI, M.D., Bayside

Consultant Rehabilitation Medicine at Long Island Jewish Medical Center, New Hyde Park; Consultant Rehabilitation Medicine, Syosset Hospital, Syosset

10:30 A.M. **Cervical Compression and Dorsal and Ventral Root Disease (Manifesting Itself in the Upper Extremity)**

EDWARD WEILAND, M.D., Great Neck

Staff Neurologist, North Shore University Hospital, Manhasset

11:00 A.M. **Coffee Break to View Exhibits**

11:15 A.M. **Obstetrical Palsy (Pathophysiology, Etiology and Prognosis)**

MARVIN KLEIN, M.D., Great Neck

Former Chief, Department of Pediatric Neurology, Long Island Jewish Medical Center, New Hyde Park

(continued)

11:45 A.M. **Inflammatory Polyradiculoneuropathies**

PETER LICHTENFELD, M.D., Jericho

Adjunct Assistant Professor of Clinical Neurology, Cornell University Medical College, New York; Clinical Associate Professor of Neurology School of Medicine, State University of New York at Stony Brook

12:15 P.M. **Metabolic, Hereditary, Toxic, Nutritional and Idiopathic Neuropathies of the Upper Extremity**

DAVID BIDDLE, M.D., FAAN, New Hyde Park

Assistant Professor of Medicine Neurology, State University of New York at Stony Brook

12:45 P.M.-1:45 P.M. **Lunch Break**

1:45 P.M. **Cerebral Palsy and The Spastic Upper Extremity**

STEVEN M. GREEN, M.D., New York City

Clinical Associate Professor of Orthopedic Surgery at Mt. Sinai Medical School, New York; Assistant Chief of Hand Services, Mt. Sinai Hospital and Hospital For Joint Diseases, New York City

2:15 P.M. **The Pathology Of Compression Neuropathies Of The Upper Extremity**

THOMAS J.PALMIERI, M.D., New Hyde Park

Physician-in-charge Hand Surgery, Long Island Jewish-Hillside Medical Center; Assistant Professor of Clinical Surgery, State University of New York at Stony Brook

2:45 P.M. **The Diagnosis and Surgical Repair of Peripheral Nerve Injuries**

JOEL B. GRAD, M.D., New York City

Assistant Professor, Hand Surgery Service, New York University Medical Center; Director, Hand Surgery Service, Department of Orthopedic Surgery, St. Vincent's Hospital, New York City

3:15 P.M. **Coffee Break to View Exhibits**

3:30 P.M. **Tendon Transfers For Peripheral Nerve Palsies (Including Biomechanics of The Transfer)**

MELVIN ROSENWASSER, M.D., New York City

Assistant Professor, Department of Orthopedic Surgery, Columbia University College of Physicians and Surgeons, New York City

4:00 P.M. **The Management of Chronic Pain and Reflex Sympathetic Dystrophy**

CHARLES M. FERMON, M.D., New York City

Attending Anesthesiologist, Long Island Jewish Medical Center; Residency Program Director of Anesthesiology, Long Island Jewish Medical Center, New Hyde Park

4:30 P.M. **The Role of the Hand Therapist in Rehabilitation of the Paralyzed Hand and Upper Extremity**

WENDY R. BURNETT, O.T.R., New York City

Director, New York Hand Surgery and Rehabilitation, P.C.; President, New York Society for Hand Therapy

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Psychiatry
Joint Meeting With
New York State Psychiatric Association

9:30 A.M.-5:00 P.M.
GRAMERCY B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 6 Hours, Prescribed

MODERN PSYCHIATRY IN THE TREATMENT OF SUBSTANCE ABUSE AND DEPENDENCY

RICHARD B. DROOZ, M.D., New York City, *Moderator*

Associate Professor of Psychiatry (Ret.), State University of New York, Downstate Medical Center, Brooklyn; Attending Psychiatrist (Ret.), Kings County Hospital Center, Brooklyn.

Objective: To provide a comprehensive and timely view of "Habit, Abuse and Dependency," and of the modern psychiatric techniques of treatment of these conditions in both ambulatory and hospital settings.

9:30 A.M. **Introduction**

RICHARD B. DROOZ, M.D., New York City, *Moderator*

Associate Professor of Psychiatry (Ret.), State University of New York, Downstate Medical Center, Brooklyn; Attending Psychiatrist (Ret.), Kings County Hospital Center, Brooklyn

9:40 A.M. **The Epidemiology Of Substance Abuse**

DENISE B. KANDEL, Ph.D., New York City

Professor of Public Health in Psychiatry, College of Physicians and Surgeons, Columbia University, New York; Research Scientist, New York State Psychiatric Institute, New York

10:25 A.M. **The Differential Diagnosis Of The Mentally Ill Chemical Abuser**

RICHARD N. ROSENTHAL, M.D., New York City

Assistant Professor of Psychiatry, Mt. Sinai College of Medicine, New York; Physician-in-Charge, Psychiatric Substance Abuse Services, Beth Israel Medical Center, New York

11:10 A.M. **Coffee Break to View Exhibits**

(continued)

11:30 A.M. **The Dynamic And Technical Aspects Of Anorexia and Bulimia**

IRA L. MINTZ, M.D., Englewood, New Jersey

Supervising Child Psychoanalyst, Columbia University Center for Psychoanalytic Training and Research, New York; Associate Professor of Psychiatry, New Jersey College of Medicine, Jersey City, New Jersey

12:15 P.M. **The Airline Pilot Alcoholism Program: Success And Implications For Other Professions**

BARTON PAKULL, M.D., Washington, D.C.

Chief Psychiatrist, Federal Aviation Administration, Washington, D.C.

1:00 P.M.–2:00 P.M. **Lunch Break**

2:00 P.M. **1888–1988: Cocaine And Freud Revisited**

RONALD J. DOUGHERTY, M.D., Brewerton

Administrative Service Chief, Chemical Abuse Recovery Service Unit, Benjamin Rush Center, Syracuse; Medical Director, Pelion, Inc.

2:40 P.M. **Anorexia Nervosa: The Destructive Pursuit of Thinness**

STEPHEN NORDLICHT, M.D., New York City

Consulting Psychiatrist, Memorial Sloan-Kettering Center, Cornell University Medical Center, New York; Chairman, Scientific Program Committee, Medical Society of the State of New York, Lake Success

3:20 P.M. **Coffee Break to View Exhibits**

3:40 P.M.
1. **Comprehensive Treatment: Teens With Drug Depression, Sexual And Eating Disorders**
2. **The Hotline: 8-0-0--C-O-C-A-I-N-E**

J. CALVIN CHATLOS, M.D., Summit, New Jersey

Director, Adolescent Substance Abuse Program, Fair Oaks Hospital, Summit, New Jersey; Director, Adolescent Services, "8-0-0–C-O-C-A-I-N-E," Fair Oaks Hospital, Summit, New Jersey

4:20 P.M. **The Epidemiology Of The Interrelationship Of Chemical Dependency And Acquired Immune Deficiency Syndrome**

DANIEL K. FLAVIN, M.D., New York City

Assistant Clinical Professor of Psychiatry, New York Medical College, Valhalla; Chief, Outpatient Alcoholism Treatment Program, St. Vincent's Hospital and Medical Center, New York

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Ophthalmology
Joint Meeting with
New York State Ophthalmological Society

9:30 A.M.–5:00 P.M.
SUTTON PARLOR SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 6 Hours, Prescribed

CONTACT LENS USE AND ABUSE: PREVENTION AND MANAGEMENT OF COMPLICATIONS

ERIC D. DONNENFELD, M.D., Rockville Centre, *Moderator*

Associate Director Cornea Service, North Shore University Hospital, Manhasset; Co-Director External Disease Service, Manhattan Eye, Ear and Throat Hospital, New York City

Objective: A comprehensive program dealing with the prevention and management of contact lens complications. Objectives include: Selection of an appropriate contact lens and contact lens cleaning regimen, the early recognition of patients at risk for developing contact lens complications and the treatment of these complications. The course will update new therapies and is geared to the general practicing Ophthalmologist.

9:30 A.M. ERIC D. DONNENFELD, M.D., Associate Director Cornea Service, North Shore University Hospital, Manhasset; Co-Director External Disease Service, Manhattan Eye, Ear and Throat Hospital, New York City

9:40 A.M. **Basic Contact Lens Design and Fitting**

SID MANDELBAUM, M.D., New York City

Assistant Attending Physician, Manhattan Eye, Ear and Throat Hospital, New York City

10:10 A.M. **Evaluation of Contact Lens Cleaning and Disinfection Systems**

MICHAEL STARR, M.D., New York City

Co-Chairman External Disease Department, Manhattan Eye, Ear and Throat Hospital, New York City; Associate Clinical Professor, New York Hospital, New York City

10:40 A.M. **Coffee Break to View Exhibits**

11:00 A.M. **Prefitting Evaluation of the Contact Lens Patient**

HENRY D. PERRY, M.D., New York City

Director Cornea Service, North Shore University Hospital, Manhasset; Associate Professor, Cornell University Medical Center, New York City

11:30 A.M. **Question and Answer Period**

12:00 P.M. **Giant Papillary Conjunctivitis Update**

IRA J. UDELL, M.D., New Hyde Park

Physician in Charge, Division of Corneal and External Disease, Long Island Jewish Medical Center, New Hyde Park

(continued)

12:30 P.M. **Preservative Hypersensitivity Reactions**

KEN KENYON, M.D., Boston, Massachusettts

Director of Cornea and External Disease Service, Associate Chief of Ophthalmology, Massachusetts Eye and Ear Infirmary

1:00 P.M.–2:00 P.M. **Lunch Break**

2:00 P.M. **Contact Lens Keratopathy and Contact Lens Related Corneal Neovascularization**

ERIC DONNENFELD, M.D., Rockville Centre

Associate Director Cornea Service, North Shore University Hospital, Manhasset; Co-Director External Disease Service, Manhattan Eye, Ear and Throat Hospital, New York City

2:30 P.M. **Question and Answer Period**

2:55 P.M. **Changing Trends In Contact Lens Related Corneal Ulcers**

PETER LAIBSON, M.D. Philadelphia, Pennsylvania

Professor of Ophthalmology, Thomas Jefferson University Hospital; Director Cornea Service, Wills Eye Hospital

3:25 P.M. **Coffee Break To View Exhibits**

3:45 P.M. **Treatment of Contact Lens Related Corneal Ulcers**

JULES BAUM, M.D., Boston, Massachusetts

Director of Cornea and External Disease Department, Professor of Ophthalmology, New England Medical Center and Tufts University School of Medicine, Boston, Massachusetts

4:15 P.M. **Acanthamoeba Keratitis**

CYNTHIA PARLATO, M.D., Utica

Associate Professor of Ophthalmology, Upstate Medical Center at Syracuse

4:45 P.M. **Question and Answer Period**

Internal Medicine
Joint Meeting With
New York State Society of Internal Medicine

9:30 A.M.–5:00 P.M.
REGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 6 Hours, Prescribed

INFECTIOUS DISEASE: FOCUS ON PREVENTION

SIDNEY WEINSTEIN, M.D., Rochester, *Moderator*

Clinical Associate Professor of Medicine, University of Rochester School of Medicine

Objective: This meeting is designed for primary care and all other physicians interested in the prevention of infectious disease. A broad range of problems in the area of infectious disease will be examined.

9:30 A.M. **Rapid Diagnosis Of Infectious Diseases**

LEONARD J. LASCOLEA, JR., Ph.D., Buffalo

Director, Clinical Microbiology Laboratories, Children's Hospital, of Buffalo; Associate Professor of Pediatrics and Microbiology, Children's Hospital of Buffalo

10:00 A.M. **Avoiding Infection While Traveling**

RICHARD HORNICK, M.D., Orlando, Florida

Vice President for Medical Education, Orlando Regional Medical Center

10:30 A.M. **Coffee Break**

10:50 A.M. **Antibiotic Prophylaxis In Primary Care**

BARRY BRAUSE, M.D., New York City

Clinical Associate Professor of Medicine, Cornell University Medical College; Associate Attending Physician, New York Hospital and Hospital for Special Surgery

11:20 A.M. **Potentials For Immunotherapy of Gram Negative Sepsis**

WILLIAM MCCABE, M.D.

Professor of Medicine and Microbiology, Boston University School of Medicine; Director of Infectious Diseases, Boston University

11:50 A.M. **Prevention Of Infectious Disease By Environmental Control**

KAREN BELL, M.D.

Deputy Director, Monroe County Health Department

12:20 P.M. **Immunologic and Chemotherapeutic Approaches In The Prevention of Human Immuno Deficiency Virus (HIV)**

OSCAR LASKIN, M.D.

Assistant Professor of Medicine and Pharmacology, Cornell University Medical College

1:00 P.M.–2:00 P.M. **Lunch Break**

(continued)

2:00 P.M. **Sexually Transmitted Diseases: Primary and Secondary Prevention**

MICHAEL F. REIN, M.D., Charlottesville, Virginia

Associate Professor of Medicine, University of Virginia School of Medicine; Editor *Sexually Transmitted Diseases*

2:30 P.M. **Hospital Acquired Infections: Who's At Risk and Who Isn't**

WILLIAM VALENTI, M.D., Rochester

Associate Professor of Medicine, University of Rochester School of Medicine

3:00 P.M. **Prevention of Respiratory Infection in Adults: Value of Vaccine and Antiviral Prophylaxis**

ROBERT BETTS, M.D., Rochester

Professor of Medicine, University of Rochester School of Medicine

3:30 P.M. **Coffee Break To View Exhibits**

3:50 P.M. **To Be Announced**

4:20 P.M. **To Be Announced**

Urology/Radiology
Joint Meeting With
New York State Urological Society/New York State Chapter—American College Of Radiology

9:30 A.M.-1:00 P.M.
NASSAU A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

EVALUATION AND TREATMENT OF RENAL MASSES

PETER J. PUCHNER, M.D., New York City, *Moderator*

Associate Professor of Clinical Urology, Columbia University, College of Physicians and Surgeons; Associate Attending Urologist, Columbia Presbyterian Medical Center

Objective: This joint symposium will explore clinical concepts in the evaluation of renal masses. An interdisciplinary panel will present current modalities for accurate diagnosis and treatment of benign and malignant renal tumors.

9:30 A.M. **Changing Clinical Presentation of Renal Masses**

PETER J. PUCHNER, M.D., New York City

Associate Professor of Clinical Urology, Columbia University, College of Physicians and Surgeons; Associate Attending Urologist, Columbia Presbyterian Medical Center

9:45 A.M. **Pathology Of Renal Masses**

KATHLEEN M. O'TOOLE, M.D., New York City

Assistant Professor of Pathology, Columbia University, College of Physicians and Surgeons

10:05 A.M. **Sensitivity And Specificity Of Imaging For Renal Tumors**

JEFFREY NEWHOUSE, M.D., New York City

Associate Professor of Radiology, Columbia University, College of Physicians and Surgeons; Associate Attending Radiologist, Columbia Presbyterian Medical Center

10:20 A.M. **Imaging Differential Diagnosis Of Renal Masses**

E. S. AMIS, JR., M.D., New York City

Associate Professor of Radiology and Chief, Uroradiology Section Columbia University, College of Physicians and Surgeons; Associate Attending Radiologist, Columbia Presbyterian Medical Center

10:40 A.M. **Magnetic Resonance Imaging And Other Modalities in Staging Renal Carcinoma**

JEFFREY NEWHOUSE, M.D., New York City

Associate Professor of Radiology, Columbia University, College of Physicians and Surgeons; Associate Attending Radiologist, Columbia Presbyterian Medical Center

11:00 A.M. **Coffee Break to View Exhibits**

11:20 A.M. **Clinical Staging Of Renal Tumors; Treatment of Benign and Low-Stage Renal Tumors**

RICHARD J. MACCHIA, M.D., Brooklyn

Associate Professor of Urology, State University of New York, Health Science Center; Chairman of Department of Urology, State University of New York, Health Science Center

11:45 A.M. **Treatment of Advanced Renal Carcinoma—Overall Prognosis By Stage Of Disease**

IHOR SAWCZUK, M.D., New York City

Assistant Professor of Urology, Columbia University, College of Physicians and Surgeons; Assistant Attending Urologist, Columbia Presbyterian Medical Center

12:10 P.M. **Diagnosis and Treatment Of Renal Tumors In Children**

KEVIN BURBIGE, M.D., New York City

Assistant Professor of Urology, Columbia University, College of Physicians and Surgeons; Associate Director of Pediatric Urology, Columbia University, College of Physicians and Surgeons

12:30 P.M. **Summary**

Questions and Answers

(continued)

Dermatology Joint Meeting with New York State Society of Dermatology

9:30 A.M.–1:00 P.M.
MURRAY HILL A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

AIDS and SELF-INDUCED SKIN DISEASE

LOWELL A. GOLDSMITH, M.D., Rochester, New York, *Moderator*

Professor and Chairman, Department of Dermatology, University of Rochester Medical Center.

Objective: Physicians will gain updated information on AIDS-induced and self-induced skin diseases, enabling them to recognize and manage these disorders.

9:30 A.M. **AIDS And Its Cutaneous Manifestations**

ALVIN E. FRIEDMAN-KEIN, M.D., New York City

Professor of Dermatology, New York University Medical Center

10:10 A.M. **Histopathology and Histogenesis of Kaposi's Sarcoma**

A. BERNARD ACKERMAN, M.D., New York City

Professor of Dermatology and Pathology, New York University Medical Center

10:50 A.M. **Coffee Break to View Exhibits**

11:10 A.M. **Cutaneous Signs of Child Abuse and What To Do About Them**

ALFRED T. LANE, M.D., Rochester

Assistant Professor of Dermatology and Pediatrics, University of Rochester School of Medicine and Dentistry

11:45 A.M. **Diagnosis and Management of Factitial Disease**

MILTON VIEDERMAN, M.D., New York City

Professor of Clinical Psychiatry, Cornell University Medical School; Director of Consultation Services, New York Hospital

12:20 P.M. **Dermatological and Psychogenic Aspects of Factitial Skin Ulcers**

LOUIS N. VOGEL, M.D., New York City

Clinical Instructor in Dermatology, New York University

12:50 P.M. **Question and Answer Period**

Advances In The Application of Medical Technology #3

2:00 P.M.–3:00 P.M.
NASSAU A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 1 Hour, Category 1; AAFP, 1 Hour, Prescribed

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WILLIAM K. WEISSMAN, M.D., New York City, *Moderator*

President, Theorex Corporation; Adjunct Research Professor, New Jersey Institute of Technology

Objective: To demonstrate that the data handling, arrhythmetic and analytical capabilities of the most recent personal computers, when associated with information networks, remote data bases and other computers, serve as powerful tools for use by the physician.

2:00 P.M. **Implications of Computing To The Practice Of Medicine**

FRANK S. BECKMAN, Ph.D.

Professor and Executive Officer, Ph.D. Program in Computer Science, Graduate School and University Center, City University of New York

(continued)

Chest Diseases/Otolaryngology
Joint Meeting With
American College of Chest Physicians, New York State Chapter and New York State Society of Otolaryngology—Head and Neck Surgery

2:00 P.M.-5:00 P.M.
MURRAY HILL B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

PULMONARY PROBLEMS IN SUBSTANCE ABUSE

WILLIAM A. BECKER, M.D., Bloomfield, New Jersey, *Moderator*

Chest Diseases Director Emeritus, Methodist Hospital of Brooklyn; Internal Medicine Clinical Professor Emeritus, SUNY Health Science Center, Brooklyn

ROY B. SESSIONS, M.D., New York City, *Moderator*, Otolaryngology

Associate Attending, Surgery Head and Neck, Memorial Sloan Kettering Center, New York City

Objective: To review the physiology and treatment of substance abuse in pulmonary and infectious diseases.

2:00 P.M. **The impact of Smokeless Tobacco on Society**

ROY B. SESSIONS, M.D., New York City

Associate Attending, Surgery Head and Neck, Memorial Sloan Kettering Center, New York

2:40 P.M. **THE 37th HOWARD LILIENTHAL MEMORIAL LECTURE: Physiologic Pulmonary Problems in Substance Abuse**

ROBERTA M. GOLDRING, M.D., New York City

Professor of Medicine, New York University School of Medicine, New York City; Director of Pulmonary Function Laboratory, Bellevue Hospital, New York City

3:25 P.M. **Coffee Break to View Exhibits**

3:45 P.M. **Tuberculosis in the Substance Abuse Patient: Diagnosis, Treatment and Toxicity**

CHARLES FELTON, M.D., New York City

Clinical Professor of Medicine; Acting Director of Medicine, Harlem Hospital Center, New York City

4:20 P.M. **Pulmonary Problems in AIDS in Substance Abuse Patients**

STUART GARAY, M.D., New York City

Assistant Professor of Medicine, New York University, Bellevue Medical Center

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Computers In Medicine

3:00 P.M.-5:00 P.M.
NASSAU A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 2 Hours, Category 1; AAFP, 2 Hours, Prescribed

PERCEPTION AND COMMUNICATIONS

WILLIAM K. WEISSMAN, M.D., New York City, *Moderator*

President, Theorex Corporation; Adjunct Research Professor, New Jersey Institute of Technology

Objective: To illustrate some representative computerized projects in speech and vision current in selected research laboratories. The outcome of these and similar activities will probably lead to advanced clinical applications.

3:00 P.M. **Speech Recognition**

Jay Wilpon, M.S. in E.E., member of technical staff at AT&T, Bell Laboratories, Murray Hill, New Jersey

4:00 P.M. Towards An Organizing Principle For A Perceptual System

Ralph Linsker, M.D., Ph.D., IBM Research Division, TJW Research Center Yorktown Heights, New York

(continued)

SUNDAY, APRIL 24, 1988

Advances In The Application Of Medical Technology #4

8:30 A.M.–9:30 A.M.
MURRAY HILL B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 1 Hour, Category 1; AAFP, 1 Hour, Prescribed

GUEST TECHNOLOGY SPEAKERS; MEDICAL SOCIETY OF THE STATE OF NEW YORK ASSEMBLY '88

WILLIAM K. WEISSMAN, M.D., New York City, *Moderator*

President, Theorex Corporation; Adjunct Research Professor, New Jersey Institute of Technology

Objective: All living organisms depend upon polymeric materials for their structure and chemical functions. Modern polymer science attempts to create macromolecules and related materials that complement and reinforce naturally occurring substances. This talk describes relationships between these synthetic polymer structures and their applications in medicine.

8:30 A.M.–9:30 A.M. **Polymer Science In New Medicine**

CARL C. GRYTE, Ph.D., New York City

Associate Professor, Department of Chemical Engineering and Applied Chemistry, Columbia University School of Engineering and Applied Science

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Obstetrics And Gynecology
Joint Meeting With
American College of Obstetricians and Gynecologists—District 11

9:30 A.M.–1:00 P.M.
MURRAY HILL A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

SUBSTANCE ABUSE AND OBSTETRICS AND GYNECOLOGY

JOHN T. PARENTE, M.D., *Moderator*, Bronx

Director, Department of Obstetrics and Gynecology, The Bronx-Lebanon Hospital Center; Associate Professor, Obstetrics and Gynecology, The Albert Einstein College of Medicine, Bronx

Objective: To educate physicians regarding the problems of drug abuse in pregnancy and the effect on the neonate.

9:30 A.M. **Introduction**

JOHN T. PARENTE, M.D., Bronx

Director, Department of Obstetrics and Gynecology, The Bronx-Lebanon Hospital Center; Associate Professor, Obstetrics and Gynecology, The Albert Einstein College of Medicine, Bronx

9:40 A.M. **A Sequential Drug Abuse Study of Obstetrics/Gynecology In-patients At The Bronx-Lebanon Hospital Center**

BENJAMIN GAINES, M.D., Bronx

Chief Resident, Obstetrics and Gynecology, The Bronx-Lebanon Hospital Center, Bronx

10:15 A.M. **Effect Of Maternal Cocaine Abuse On The Newborn**

JING J. YOON, M.D., Bronx

Associate Professor, Department of Pediatrics, Albert Einstein College of Medicine, Bronx; Chief, Division of Neonatology, Department of Pediatrics, The Bronx-Lebanon Hospital Center, Bronx

10:50 A.M. **Coffee Break to View Exhibits**

11:10 A.M. **Effect Of Maternal Marijuana Abuse On The Newborn**

LAWRENCE NOBLE, M.D., Bronx

Assistant Professor, Department of Pediatrics, Albert Einstein College of Medicine, Bronx; Neonatologist, Division of Neonatology, Department of Pediatrics, The Bronx-Lebanon Hospital Center, Bronx

11:45 A.M. **Conclusion**

JOHN T. PARENTE, M.D., Bronx

Director, Department of Obstetrics and Gynecology, The Bronx-Lebanon Hospital Center

12:15 P.M. **Questions and Answers**

(continued)

Gastroenterology/Colon And Rectal Surgery

9:30 A.M.–1:00 P.M.
NASSAU A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 hours, Prescribed

MANAGING LIFE-STYLE ABUSES OF THE G.I. TRACT

JOHN S. DAVIS, M.D., Cooperstown, *Moderator*

Director of Medical Education, The Mary Imogene Bassett Hospital, Cooperstown; Associate Professor of Clinical Medicine, College of Physicians and Surgeons of Columbia University, New York City

Objective: A Symposium, including the Albert F. R. Andresen Memorial Lecture devoted to recognition and management of dysfunction and disease stemming from life-style injury of the G.I. tract and liver. Infectious and nutritional concepts will be stressed. Participants will evolve a working understanding of the recognition and treatment of a variety of serious and distressingly common disorders originating in the G.I. tract.

9:30 A.M. **Introduction**

JOHN S. DAVIS, M.D., Cooperstown

Director of Medical Education, The Mary Imogene Bassett Hospital, Cooperstown; Associate Professor of Clinical Medicine, College of Physicians and Surgeons of Columbia University, New York City

9:40 A.M. **Infectious Complications in the G.I. Tract From Life-Style Abuse**

DONALD P. KOTLER, M.D., New York City

Associate Professor of Clinical Medicine, College of Physicians and Surgeons, Columbia University, New York City; Division of Gastroenterology, St. Luke's Roosevelt Hospital Center, New York City

10:10 A.M. **Questions and Answers**

10:20 A.M. **Coffee Break to View Exhibits**

10:40 A.M. **Life-Style Abuse of The Liver: Hepatitis B and D**

DAVID J. CLAIN, M.D., New York City

Chief of Gastroenterology, Harlem Hospital Center, New York City; Associate Professor of Clinical Medicine, College of Physicians and Surgeons, Columbia University, New York City

11:10 A.M. **Questions and Answers**

11:20 A.M. **ALBERT F. R. ANDRESEN MEMORIAL LECTURE: Questionable Nutritional Practices: Ignoring the Rules of Efficacy and Safety**

VICTOR HERBERT, M.D., J.D., Bronx

Professor of Medicine, Mount Sinai School of Medicine, New York City; Chief, Hematology and Nutrition Laboratory, Veteran's Administration Medical Center, Bronx

12:20 P.M. **Questions and Answers**

Special Session On Sleep Disorders

9:30 A.M.–1:00 P.M.
NASSAU B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

WAKE UP! SLEEP APNEA CAN KILL YOU—A MULTIDISCIPLINARY APPROACH TO DIAGNOSIS AND TREATMENT OF SLEEP APNEA

MAXWELL SPRING, M.D., F.A.C.P., Bronx, *Moderator*

Clinical Associate Professor of Medicine, University of Medicine and Dentistry of New Jersey (UMD), New Jersey Medical School; Associate Chairman of Scientific Program Committee of the Medical Society Of The State Of New York

Objective: To familiarize the physician with the sleep apnea syndrome—etiology, diagnoses, complications and treatment, both medical and surgical.

9:30 A.M. **Introduction**

MAXWELL SPRING, M.D., F.A.C.P., Bronx

Clinical Associate Professor of Medicine, University of Medicine and Dentistry of New Jersey (UMD) New Jersey Medical School; Associate Chairman of Scientific Program Committee of the Medical Society of the State of New York

9:40 A.M. **Clinical Manifestations And Diagnosis**

MICHAEL J. THORPY, M.D., New York City

Assistant Professor of Neurology, Albert Einstein College of Medicine; Director, Sleep-Wake Disorders, Department of Neurology Montefiore Medical Center, Bronx

10:10 A.M. **Cardiologic Complications Of Sleep Apnea**

BERNARD BURACK, M.D., New York City

Assistant Clinical Professor, Department of Medicine, Albert Einstein College of Medicine; Attending Cardiologist, Cardiac Consultant, Sleep-Wake Disorders Unit, Montefiore Medical Center, Bronx

10:40 A.M. **Approach To The Surgical Management In Children**

AARON SHER, M.D., Albany

Clinical Associate Professor of Surgery (Oto) Albany Medical College; Associate Professor of Otorhinolaryngology, Albert Einstein College of Medicine, Bronx

(continued)

11:00 A.M. **Coffee Break to View Exhibits**

11:20 A.M. **Approach To The Surgical Management In Adults**

DANUTA L. ROZYCKI, M.D., Bronx

Associate Professor, Department of Otorhinolaryngology, Albert Einstein College of Medicine, Bronx; Director, Department of Otorhinolaryngology, Montefiore Medical Center, Bronx

12:00 P.M. **Panel Discussion**

Orthopaedic Surgery/Physical Medicine and Rehabilitation/Anesthesiology Joint Meeting with The New York Society of Orthopedic Surgery; The New York Society of Physical Medicine and Rehabilitation; The New York State Society of Anesthesiologists

9:30 A.M.–1:00 P.M.
REGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1;
AAFP, 3 Hours, Prescribed

RECREATIONAL ATHLETES–PROBLEMS AND TREATMENT

DAVID M. HIRSH, M.D., Bronx, *Moderator*

Associate Clinical Professor, Department of Orthopaedic Surgery, Jack D. Weiler Hospital of the Albert Einstein College of Medicine

Objective: To provide guidelines for systematic evaluation of the skeletal injuries and their management for non-orthopaedic physicians.

9:30 A.M. **Running Injuries In The Non-Professional–A Guide To Diagnosis and Treatment**

I. MARTIN LEVY, M.D., Bronx

Attending Orthopaedic Surgery, Jack D. Weiler Hospital of the Albert Einstein College of Medicine

10:15 A.M. **The Diagnosis and Management of Reflex Sympathetic Dystrophies**

DANILO ODIAMAR, M.D., East Meadow

Director, Pain Management Center, Nassau County Medical Center

11:00 A.M. **Coffee Break To View Exhibits**

11:20 A.M. **On-Field Management of Sports Related Injuries**

BRIAN ERNSDORF, M.D., Philadelphia, PA

Attending physician at Morse Rehabilitation Center

12:05 P.M. **Questions and Answers**

1988 ANNUAL CME ASSEMBLY

TECHNICAL EXHIBITS

Friday, April 22/Saturday, April 23

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Needham Heights, Massachusetts

Data Medic Corp.
Hauppauge, New York

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New York, New York

Encyclopaedia Brittanica, USA
Chicago, Illinois

Excelmed Billing Corp.
Bronx, New York

Execu-Flow Systems, Inc.
Cranford, New Jersey

GLAXO, Inc.
Cherry Hill, New Jersey

Gibraltar Securities Co.
Florham Park, New Jersey

Greater New York Doctor's Shopper
Brooklyn, New York

Group Health, Inc.
New York, New York

Harris-Lanier
New York, New York

Hoechst-Roussel Pharmaceuticals
Somerville, New Jersey

Hospital Recruiters
Port Jefferson Station, New York

International Preferred Assets
Coppell, Texas

Jamison Business Systems, Inc.
Smithtown, New York

Medcomp Systems, Ltd.
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Medical Billing Specialists, Inc.
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Bedford Hills, New York

Medical Business Services/Division of Empire Blue Cross and Blue Shield
New York, New York

Medical Record Association of New York State
Albany, New York

Merck Sharp & Dohme
West Point, Pennsylvania

Merrill Lynch, Pierce, Fenner and Smith
Garden City, New York

Millin Associates, Inc.
Cedarhurst, New York

Mutual Association of Professional Services (MAPS)
New York, New York

National Telebill Corp.
Norwell, Massachusetts

National Westminster Bank USA
West Hempstead, New York

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Albany, New York

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Palisades Pharmaceuticals, Inc.
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Medical Society of the
State of New York

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in honor of
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Thursday
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Trianon Ballroom
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Cocktail Hour 7 P.M.	Superlative Dinner

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Please make reservation(s)
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Check enclosed for $_________

PLEASE NOTE: Reservations and ticket sales will close at 12 Noon on Thursday April 21, one hour before the House Convenes.

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■

Note: Exhibitors who are not listed here will appear in the final program book which will be distributed at the CME Assembly registration desk during the meetings. Booth numbers will appear in the final program book.

United Nations Headquarters Overlooking the East River
Photo courtesy of David S. Arsenault

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1988 Annual CME Assembly, April 22-24
The New York Hilton, New York City

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BRIEF SUMMARY

CONTRAINDICATIONS
There are no known contraindications to the use of sucralfate.

PRECAUTIONS
Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that the simultaneous administration of CARAFATE with tetracycline, phenytoin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. The clinical significance of these animal studies is yet to be defined.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No evidence of drug-related tumorigenicity was found in chronic oral toxicity studies of 24 months' duration conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies have not been conducted.

Pregnancy: Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients, adverse effects were reported in 121 (4.7%). Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

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CARAFATE (sucralfate) 1-gm pink tablets are supplied in bottles of 100 and in Unit Dose Identification Paks of 100. The tablets are embossed with MARION/1712. Issued 3/84

References:
1. Korman MG, Shaw RG, Hansky J, et al: *Gastroenterology* 80:1451-1453, 1981.
2. Korman MG, Hansky J, Merrett AC, et al: *Dig Dis Sci* 27:712-715, 1982.
3. Brandstaetter G, Kratochvil P: *Am J Med* 79(suppl 2C):36-38, 1985.
4. Marks IN, Wright JP, Gilinsky NH, et al: *J Clin Gastroenterol* 8:419-423, 1986.
5. Lam SK, Hui WM, Lau WY, et al: *Gastroenterology* 92:1193-1201, 1987.

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(*continued from p 6A*)

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Feb 21–24. **Seventh Annual UCSD Imaging and Intervention Conference.** 25 Cat 1 Credits. Hotel Inter-Continental, San Diego. *Feb 29–Mar 4.* **Radiology: Imaging and Intervention.** Hotel Inter-Continental, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

COLORADO

Feb 6–13. **13th Annual Vail Symposium in Intensive Care.** Marriott's Mark Resort, Vail. Contact: Professional Seminars/University of Miami, PO Box 012318, Miami, FL 33101. Tel: (305) 547-6411.

Feb 14–19. **14th Stanford-Research Institute, Palo Alto Medical Foundation, Winter Course in Infectious Diseases.** Continental Inn, Aspen. *Feb 15–19.* **Ninth Annual Conference on Problems in Gastroenterology: A Clinical and Pathological Approach.** Keystone. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

Feb 21–25. **Eighth Annual Medical Ski Conference.** Keystone Lodge. Contact: Marge Adey or Brenda Ram, Center for Continuing Education, University of Nebraska Medical Center, 42nd and Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

DISTRICT OF COLUMBIA

Feb 28–Mar 4. **Annual Meeting of the United States and Canadian Academy of Pathology.** Washington Hilton. Contact: Dr Nathan Kaufman, Secretary-Treasurer, United States and Canadian Academy of Pathology, Inc, Building C, Suite B, 3515 Wheeler Rd, Augusta, GA 30909. Tel: (404) 733-7550.

FLORIDA

Feb 1–4. **Eighth Annual Postgraduate Course—Uroradiology '88.** 28 Cat 1 Credits. Stouffer Orlando Resort at Sea World, Orlando. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (405) 641-9773.

Feb 11–13. **Perspectives in Allergy and Rheumatology.** Sarasota. Contact: American College of Physicians, PO Box 777-R-0510, Philadelphia, PA 19175.

IDAHO

Feb 20–27. **Effective Management of Common Sports Injuries.** 24 Credits AAFP. Sun Valley. Contact: Kathy Rairigh, EPIC Expeditions, PO Box 209, Sun Valley, ID 83353. Tel: (208) 788-4995.

KENTUCKY

Feb 21–26. **19th Family Medicine Review—Session 1.** Hyatt Regency Hotel, Lexington. Contact: Joy Greene, Continuing Medical Education, 132 College of Medicine Office Bldg, University of Kentucky, Lexington, KY 40536-0086. Tel: (606) 233-5161.

LOUISIANA

Feb 11–13. **Mardi Gras Endocrine Update.** New Orleans. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

TEXAS

Feb 5–7. **Hematology for Non-Hematologists: Laboratory and Practice.** Galveston. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

Feb 26–28. **Rhinoplasty: An Educational Symposium.** 21 Cat 1 Credits. The University of Texas Health Science Center at Dallas. Contact: Ann Parchem, Continuing Education Division, Division of Plastic and Reconstructive Surgery, The University of Texas Health Science Center at Dallas, 5323 Harry Hines Blvd, Dallas, TX 75235.

UTAH

Feb 6–13. **Effective Diagnosis and Treatment of Infectious Diseases.** Park City. 24 Credits by AAFP. Contact: Kathy Rairigh, EPIC Expeditions, PO Box 209, Sun Valley, ID 83353. Tel: (208) 788-4995.

Feb 27–Mar 5. **Fourth Annual Office Based Sports Medicine.** Snowbird. Contact: University of California, Ex-

(*continued on p 42A*)

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References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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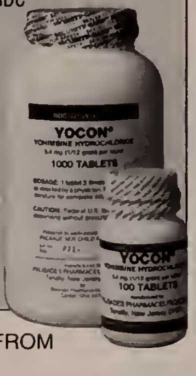

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(*continued from p 40A*)

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VERMONT

Feb 1–5. **What's New in Internal Medicine.** Sugarbush Inn, Warren. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

Feb 9–12. **1988 Clinical Update in Obstetrics and Gynecology.** Contact: Office of CME, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

AROUND THE WORLD

CANADA

Feb 8–11. **Banff Sport Medicine '88.** Banff Springs Hotel. Contact: Banff Sport Medicine '88, c/o Dr James Clapperton, 121A-14th St NW, Calgary, Alberta, Canada T2N 1Z6.

FRANCE

Feb 19–21. **Second International Congress on Neo-Adjuvant Chemotherapy.** Contact: Service d'Oncologie Medicale, Professeur Claude Jacquillat, 47 Boulevard de l'Hopital, 75651 Paris Cedex 13—France.

MEXICO

Feb 15–19. **Clinical Heart Disease.** Hyatt Regency Hotel, Cancun. American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

Feb 22–27. **Seventh Annual Comprehensive Update on Neuroradiology, Body Imaging, and Mammography.** 30 Cat 1 Credits. Acapulco Princess Hotel, Acapulco. Contact: Office of Continuing Education, SUNY Health Science Center at Syracuse, 750 East Adams St, Syracuse, NY 13210.

PUERTO RICO

Feb 14–18. **13th W. Franklin Keim Memorial Seminar on Head and Back Tumors.** Cerromar Beach Hotel, Dorado. Contact: Ki Han, MD, Newark Eye and Ear Infirmary, 15 South 9th St, Newark, NJ 07107. Tel: (201) 368-8129. □

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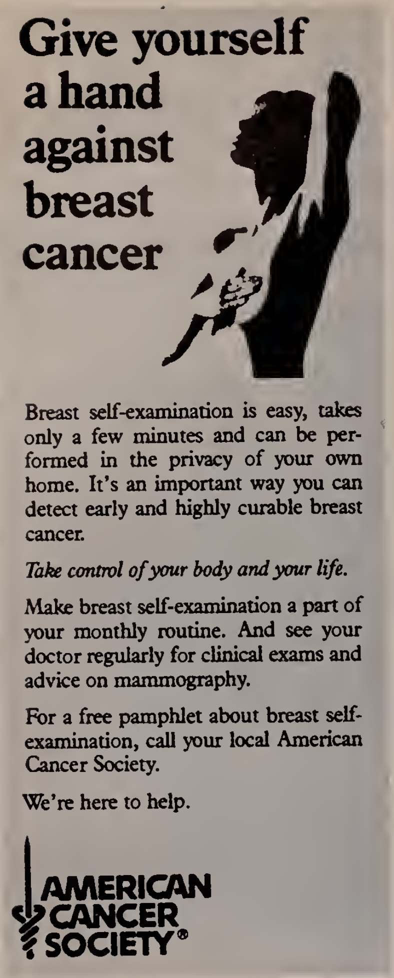

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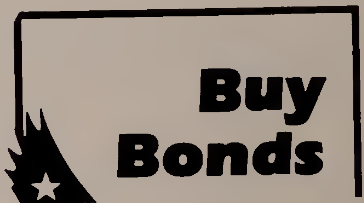

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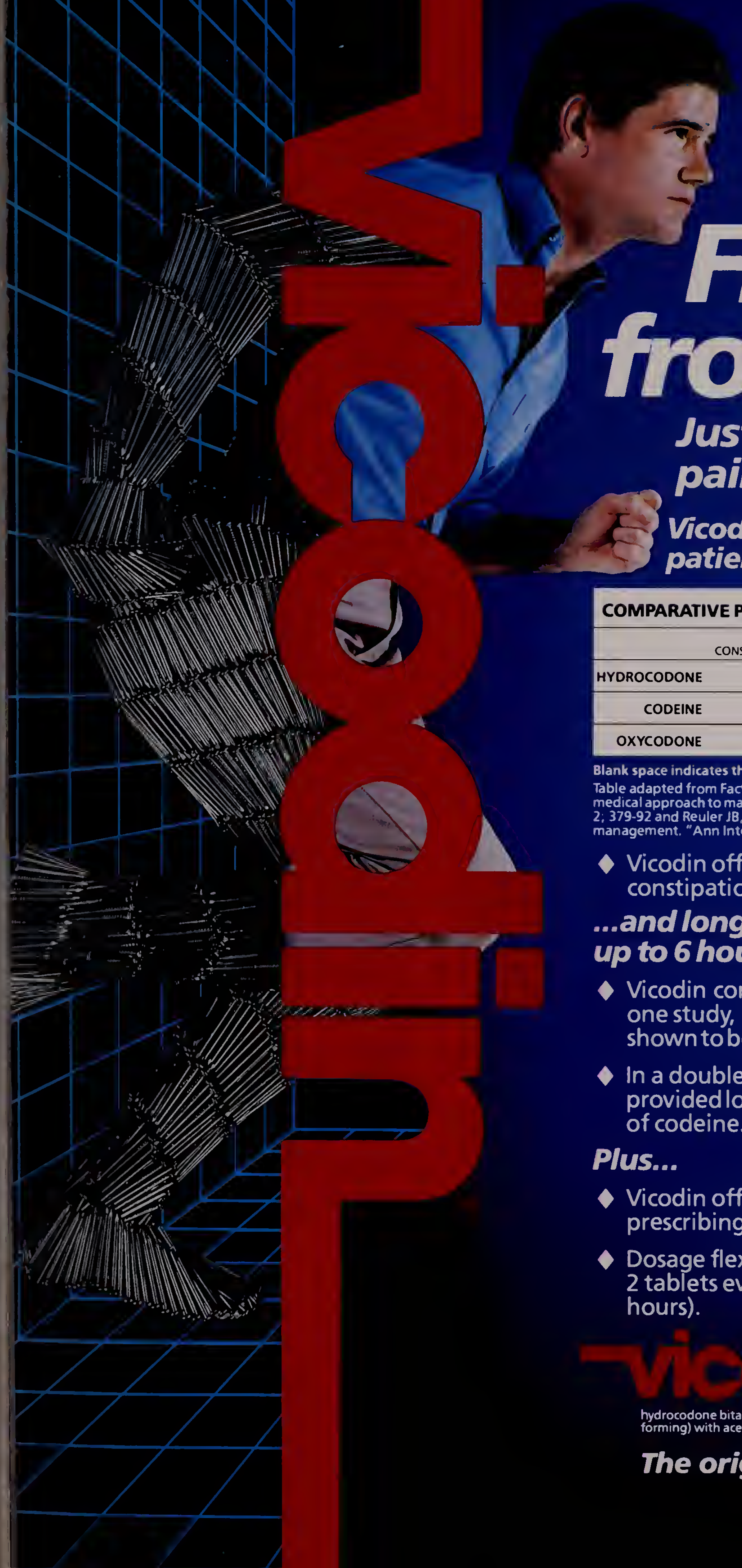

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1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med,* 141:293-300, 1981.

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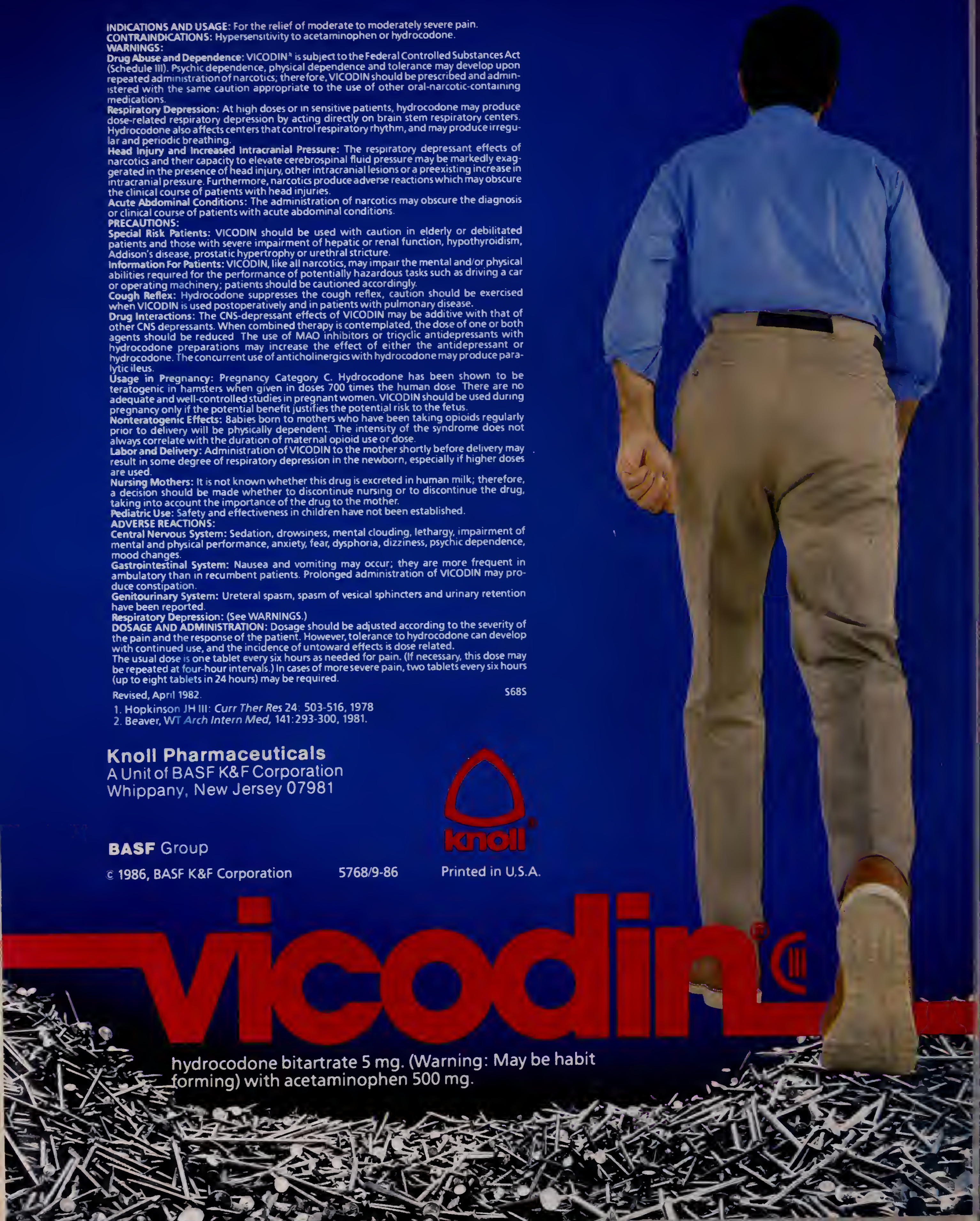

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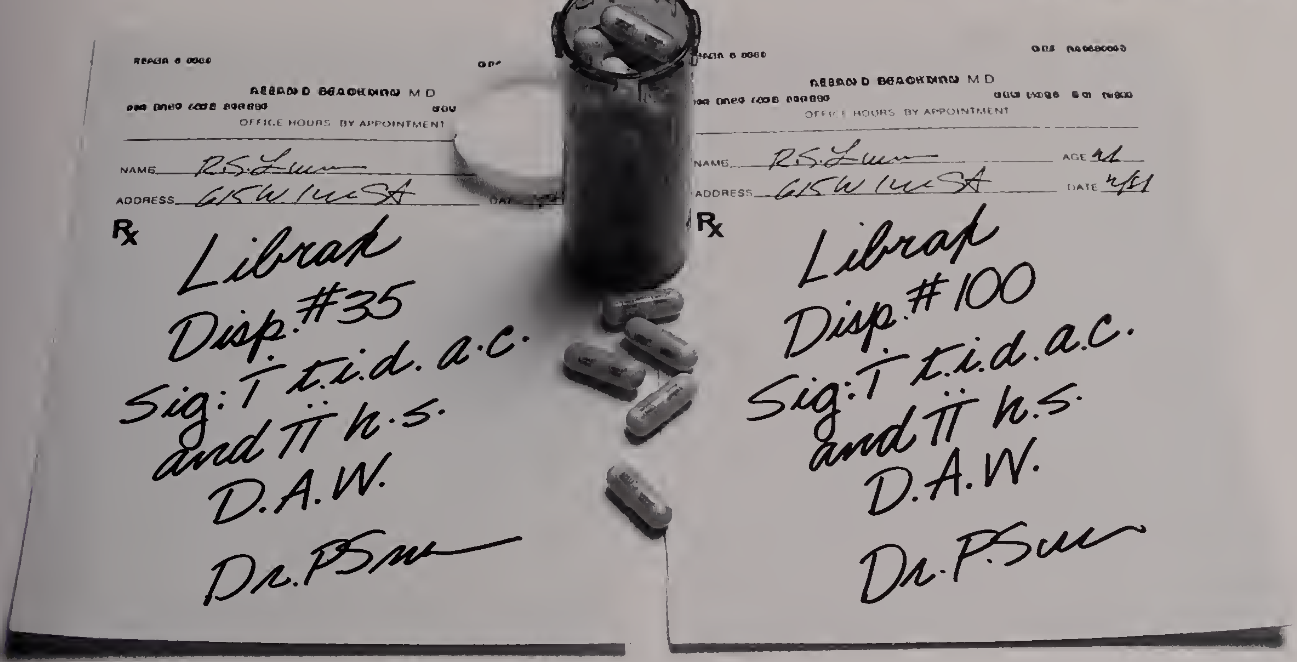

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Disp. #35
Sig: ī t.i.d. a.c.
and īī h.s.
D.A.W.
Dr. P.Sm

OFFICE HOURS BY APPOINTMENT

NAME R.S. Lum AGE 41

ADDRESS 615 W 1..St DATE 4/81

℞ Librax
Disp. #100
Sig: ī t.i.d. a.c.
and īī h.s.
D.A.W.
Dr. P.Sm

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.
Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

P.I. 0186

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LIBRAX®

Each capsule contains 5 mg chlordiazepoxide HCl
and 2.5 mg clidinium bromide

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of peptic ulcer and the irritable bowel syndrome.

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FEBRUARY 1988 • Volume 88, Number 2

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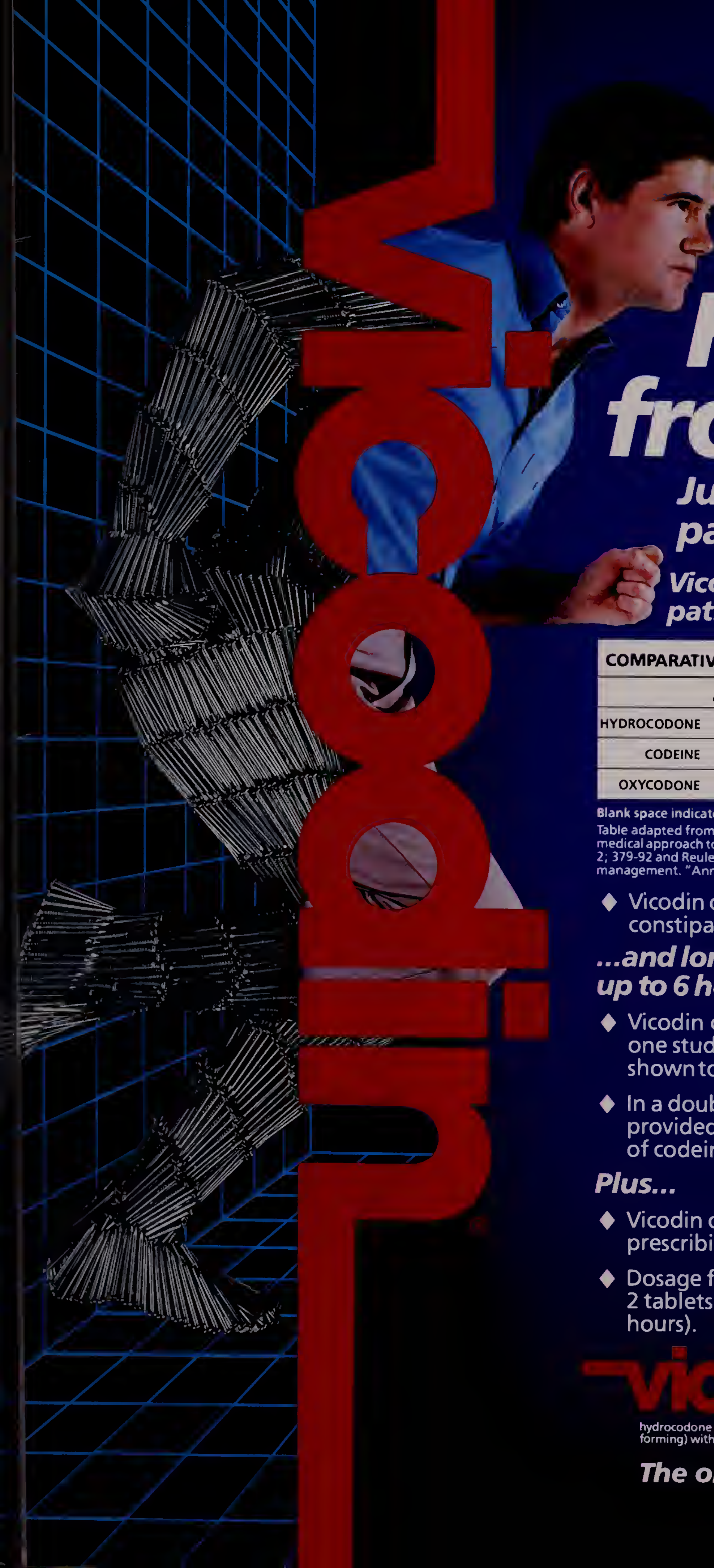

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CODEINE	X	X	X	X	X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975; 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

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PRECAUTIONS:
Special Risk Patients: VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Information For Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
Cough Reflex: Hydrocodone suppresses the cough reflex; caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants. When combined therapy is contemplated, the dose of one or both agents should be reduced. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic Effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
Labor and Delivery: Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used
Nursing Mothers: It is not known whether this drug is excreted in human milk; therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS:
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes
Gastrointestinal System: Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients. Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.
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Revised, April 1982 5685

1 Hopkinson JH III *Curr Ther Res* 24 503-516, 1978
2 Beaver, WT *Arch Intern Med*, 141.293-300, 1981.

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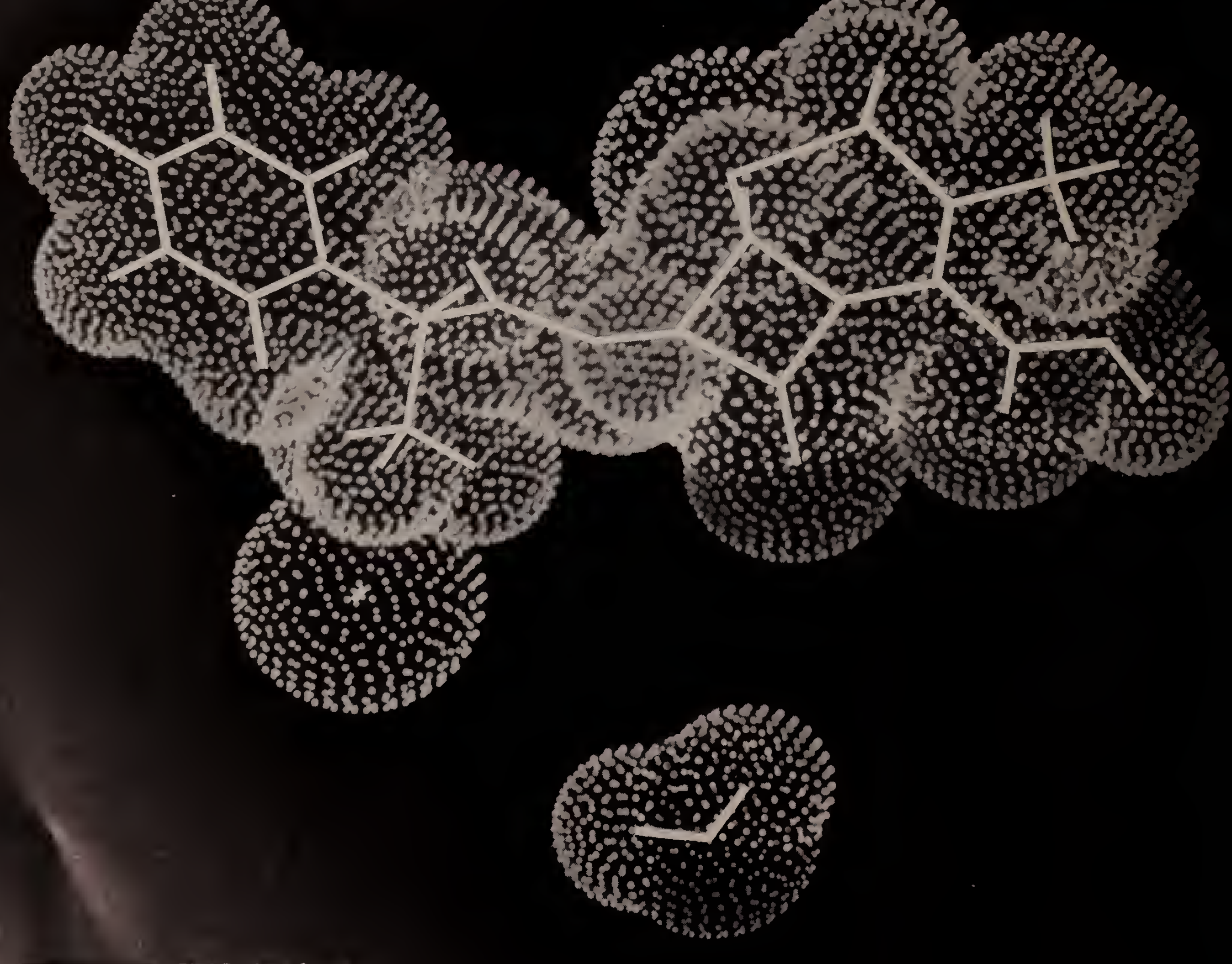

Computer-generated molecular structure of cephalexin hydrochloride monohydrate

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☐ uncomplicated cystitis[†]
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[*] Due to susceptible strains of *Staphylococcus aureus* and/or β-hemolytic streptococci.
Due to susceptible strains of *Escherichia coli*, *Proteus mirabilis*, and *Klebsiella* sp
[‡] Due to susceptible strains of group A β-hemolytic streptococci.

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(cephalexin hydrochloride monohydrate)

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Indications and Usage:

Respiratory tract infections caused by susceptible strains of *Streptococcus pneumoniae* and group A β-hemolytic streptococci.

Skin and skin structure infections caused by susceptible strains of *Staphylococcus aureus* and/or β-hemolytic streptococci.

Bone infections caused by susceptible strains of *S aureus* and/or *Proteus mirabilis.*

Genitourinary tract infections, including acute prostatitis, caused by susceptible strains of *Escherichia coli, P mirabilis,* and *Klebsiella* sp.

Contraindication: Known allergy to cephalosporins.

Warnings: KEFTAB SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:

- Discontinue Keftab in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Keftab should be administered cautiously in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy and lactation. Cephalexin is excreted in mother's milk. Exercise caution in prescribing Keftab for these patients.
- Safety and effectiveness in children have not been established.

Adverse Reactions:

- *Gastrointestinal,* including diarrhea and, rarely, nausea and vomiting. Transient hepatitis and cholestatic jaundice have been reported rarely.
- *Hypersensitivity* in the form of rash, urticaria, angioedema, and, rarely, erythema multiforme, Stevens-Johnson syndrome, or toxic epidermal necrolysis.
- *Anaphylaxis* has been reported.
- *Other reactions* have included genital/anal pruritus, genital moniliasis, vaginitis/vaginal discharge, dizziness, fatigue, headache, eosinophilia, neutropenia, and thrombocytopenia; reversible interstitial nephritis has been reported rarely.
- Cephalosporins have been implicated in triggering seizures, particularly in patients with renal impairment.
- *Abnormalities in laboratory test results* included slight elevations in aspartate aminotransferase (AST, SGOT) and alanine aminotransferase (ALT, SGPT). False-positive reactions for glucose in the urine may occur with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

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Before prescribing, see complete prescribing information in SK&F LAB CO. literature or PDR. The following is a brief summary.

Contraindications: *There are no known contraindications to the use of 'Tagamet'.*

Precautions: *While a weak antiandrogenic effect has been demonstrated in animals, 'Tagamet' has been shown to have no effect on spermatogenesis, sperm count, motility, morphology or in vitro fertilizing capacity in humans.*

In a 24-month toxicity study in rats at dose levels approximately 9 to 56 times the recommended human dose, benign Leydig cell tumors were seen. These were common in both the treated and control groups, and the incidence became significantly higher only in the aged rats receiving 'Tagamet'.

Rare instances of cardiac arrhythmias and hypotension have been reported following the rapid administration of 'Tagamet' HCl (brand of cimetidine hydrochloride) Injection by intravenous bolus.

Symptomatic response to 'Tagamet' therapy does not preclude the presence of a gastric malignancy. There have been rare reports of transient healing of gastric ulcers despite subsequently documented malignancy.

Reversible confusional states have been reported on occasion, predominantly in severely ill patients.

'Tagamet' has been reported to reduce the hepatic metabolism of warfarin-type anticoagulants, phenytoin, propranolol, chlordiazepoxide, diazepam, lidocaine, theophylline and metronidazole. Clinically significant effects have been reported with the warfarin anticoagulants; therefore, close monitoring of prothrombin time is recommended, and adjustment of the anticoagulant dose may be necessary when 'Tagamet' is administered concomitantly. Interaction with phenytoin, lidocaine and theophylline has also been reported to produce adverse clinical effects.

However, a crossover study in healthy subjects receiving either 'Tagamet' 300 mg. q.i.d. or 800 mg. h.s. concomitantly with a 300 mg. b.i.d. dosage of theophylline (Theo-Dur®, Key Pharmaceuticals, Inc.), demonstrated less alteration in steady-state theophylline peak serum levels with the 800 mg. h.s. regimen, particularly in subjects aged 54 years and older. Data beyond ten days are not available. (Note: All patients receiving theophylline should be monitored appropriately, regardless of concomitant drug therapy.)

Lack of experience to date precludes recommending 'Tagamet' for use in pregnant patients, women of childbearing potential, nursing mothers or children under 16 unless anticipated benefits outweigh potential risks; generally, nursing should not be undertaken in patients taking the drug since cimetidine is secreted in human milk.

Adverse Reactions: *Diarrhea, dizziness, somnolence, headache, rash. Reversible arthralgia, myalgia and exacerbation of joint symptoms in patients with preexisting arthritis have been reported. Reversible confusional states (e.g., mental confusion, agitation, psychosis, depression, anxiety, hallucinations, disorientation), predominantly in severely ill patients, have been reported. Gynecomastia and reversible impotence in patients with pathological hypersecretory disorders receiving 'Tagamet', particularly in high doses, for at least 12 months, have been reported. Reversible alopecia has been reported very rarely. Decreased white blood cell counts in 'Tagamet'-treated patients (approximately 1 per 100,000 patients), including agranulocytosis (approximately 3 per million patients), have been reported, including a few reports of recurrence on rechallenge. Most of these reports were in patients who had serious concomitant illnesses and received drugs and/or treatment known to produce neutropenia. Thrombocytopenia (approximately 3 per million patients) and a few cases of aplastic anemia have also been reported. Increased serum transaminase and creatinine, as well as rare cases of fever, interstitial nephritis, urinary retention, pancreatitis and allergic reactions, including hypersensitivity vasculitis, have been reported. Reversible adverse hepatic effects, cholestatic or mixed cholestatic-hepatocellular in nature, have been reported rarely. Because of the predominance of cholestatic features, severe parenchymal injury is considered highly unlikely. A single case of biopsy-proven periportal hepatic fibrosis in a patient receiving 'Tagamet' has been reported.*

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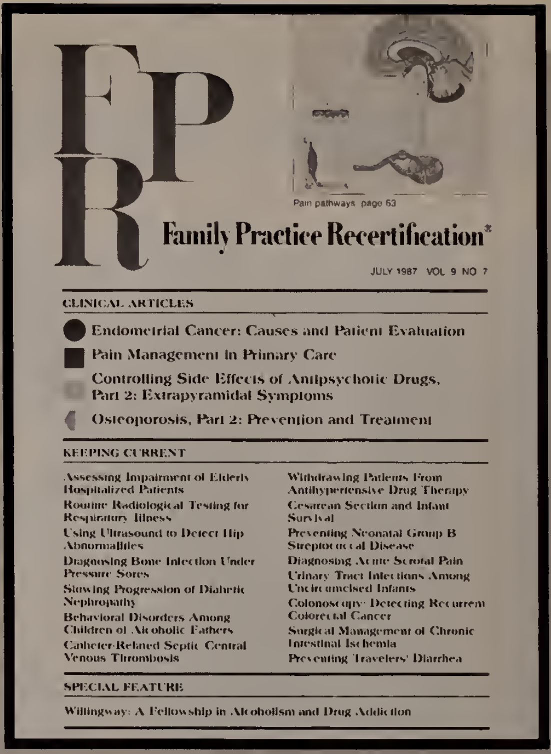

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Endometrial Cancer: Causes and Patient Evaluation

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Controlling Side Effects of Antipsychotic Drugs, Part 2: Extrapyramidal Symptoms

Osteoporosis, Part 2: Prevention and Treatment

KEEPING CURRENT

Assessing Impairment of Elderly Hospitalized Patients

Routine Radiological Testing for Respiratory Illness

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Diagnosing Bone Infection Under Pressure Sores

Slowing Progression of Diabetic Nephropathy

Behavioral Disorders Among Children of Alcoholic Fathers

Catheter-Related Septic Central Venous Thrombosis

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Diagnosing Acute Scrotal Pain

Urinary Tract Infections Among Uncircumcised Infants

Colonoscopy: Detecting Recurrent Colorectal Cancer

Surgical Management of Chronic Intestinal Ischemia

Preventing Travelers' Diarrhea

SPECIAL FEATURE

Willingway: A Fellowship in Alcoholism and Drug Addiction

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

MARCH 1988

AROUND THE STATE

ALBANY

Mar 30–31. **New Ideas and Frontiers in Gastroenterology.** Albany Hilton Hotel and Albany Medical College. Contact: Office of CME, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

MANHATTAN

Mar 3–5. **The 15th Annual Conference on Dyslexia.** The New York Penta Hotel. Contact: Samuel M. Fleisher, EdD, The Orton Dyslexia Society, Inc, 80 Fifth Ave, Room 903, New York, NY 10011. Tel: (212) 691-1930.

Mar 5. **Retinal Detachment Surgery.** *Mar 11–12.* **Fourth Annual Live Ophthalmic Plastic Surgery Workshop: Lacrimal, Eyelid, Orbit and Pediatric Surgery.** Contact: Martha Klapp, Manhattan Eye, Ear & Throat Hospital, 210 East 64th St, New York, NY 10021. Tel: (212) 605-3762.

Mar 13–18. **Anesthesiology Comprehensive Review VIII.** 46 Cat 1 Credits. New York Hilton Hotel. *Mar 14–18.* **Seminar in Advanced Rheumatology.** 28¼ Cat 1 Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical Center, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Mar 21–25. **Basic Electromyography and Electrodiagnosis and Evoked Potentials.** 31 Cat 1 Credits. Mayflower Hotel. Contact: Course Coordinator, Daoud B. Karam, MD, New York Medical College, Dept. of Rehabilitation Medicine, Lincoln Medical and Mental Health Center, 234 East 149th St, Bronx, NY 10451. Tel: (212) 579-5426.

QUEENS

Mar 23. **Update: Tuberculosis/AIDS in the Eighties.** Academy of Medicine of Queens County. Contact: Mary Nee, Secretary to the Continuing Medical Education Committee, Academy of Medicine of Queens County, 112-25 Queens Blvd, Forest Hills, NY 11375. Tel: (718) 268-7300.

SYRACUSE

Mar 4. **Noninvasive Evaluation of Coronary Artery Disease—A Clinical Perspective.** 4 Cat 1 Credits. Sheraton University Inn. Contact: Office of Continuing Education, SUNY Health Science Center at Syracuse, 750 East Adams St, Syracuse, NY 13210. Tel: (315) 473-4606.

AROUND THE NATION

CALIFORNIA

Mar 6–11. **Ninth Annual Mammoth Mountain Emergency Medicine Ski**

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JOURNAL OF MEDICINE
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COMMENTARIES

AIDS and the origin of species

Any sudden catastrophe forces us initially to view its effects in the short term—the immediate threat to ourselves and to loved ones, and the damage to society. But a few moments' reflection on the more profound implications of a calamity may allow us to profit from the lessons that it teaches.

The current epidemic of acquired immunodeficiency syndrome (AIDS) does not as yet compare in its effects with the European epidemics of bubonic and pneumonic plague in the 14th and 17th centuries, or the pandemic of influenza in 1918, or the continuing endemic of malaria in many countries. But the insidious, unrelenting, lethal characteristics of AIDS have laid an icy hand upon society. Accusations of moral misbehavior against the victims of the current AIDS epidemic tend only to divert attention from the useful lessons we can learn from consideration of other aspects of the problem.

In all likelihood the abrupt appearance of this illness would have been no surprise to Charles Darwin. A rereading of his *Origin of Species*[1] suggests that not only would he have anticipated such an event for a number of speculative reasons which I will mention, but he probably would have suggested that a parallel condition must have occurred in the extremely remote past, long before records of such events could have been kept.

Darwin concluded that each successive generation of a species produces variants of the original organism, usually over the very long term and with small increments of change, but sometimes with abruptly evident effects. Those variants which are better adapted to their environment than their forebears or their contemporaries tend to survive; those less well adapted tend to vanish. He believed that without variation within its many species, life itself would not have survived the widely differing and frequently changing conditions on this planet. In Darwin's view, even those variants that best fit a particular environment would disappear if the environment itself were to change significantly. Further, even those variants that are well suited to a particular environment are often subject to very narrow limits of behavior, which the organism ignores at its peril.

In explaining the biological interactions between living organisms, Darwin offers a complex scenario which appears to fit many aspects of AIDS.

The sexual form of reproduction appears to allow the offspring within a species to have access to a diverse gene pool and to a wide opportunity for variation. The act of sexual intercourse involves the transmission of cells and secretions from one sexual partner to the other; hence it must involve some modification of the recipient's immune system in order to avoid antibody production that would prevent effective insemination.

It is possible that currently existing sexual species are those variants which happened to evolve a controlled or reduced immune reaction in the lower genital tracts of the females, whereby the inseminating cells (spermatozoa) are not attacked by antibody production, despite repeated inseminations. At the same time that spermatozoa are left relatively intact, microorganisms invading the female genital tract generally are dealt with effectively by usual host-defenses. The immunologic mechanisms no doubt evolved side-by-side with the common genital-to-genital patterns of direct sexual contact between male and female partners.

During embryonic development, the cloaca differentiates to form the urogenital sinus and the rectum-anus. Postnatally, both vagina and rectum-anus must adjust to colonization by different groupings of microorganisms. The columnar epithelium of the lower colon and rectum adjusts immunologically to the presence of some 400 or more different bacterial species, and to other microorganisms.[2] By contrast, the vaginal squamous epithelium, the cervix, and no doubt the endometrium adjust to different groupings of organisms.[3-5] As long as sexual intercourse occurs in the usual fashion, the receptive site can deal appropriately with the entry of many foreign proteins, spermatozoa, and microorganisms.

When sexual contact does not follow the usual pattern, a variety of effects may result. In the case of oral-genital sexual contact, the immunologic defenses of the squamous epithelium of the upper respiratory and alimentary tracts might be effective in preventing invasion by unanticipated microorganisms, since from the moment of birth these points of entry have adjusted to a wide spectrum of microorganisms and foreign proteins. In addition, the swallowing process quickly invokes the destructive effects of gastric acidity and proteolytic enzymes.

By contrast, when genital-anal penetration occurs, spermatozoa together with an unaccustomed set of microorganisms now come in contact with columnar intestinal mucosa that may not yet have evolved adequate immunologic barriers against them.[6] The result may be easier invasion of microorganisms into the mucosal cells and thence into the internal milieu of the recipient sexual partner. In most cases, the latter's systemic immunologic defenses then generate adequate responses.

Let us vary this scenario by adding the factor of numerous and promiscuous genital-anal intrusions, during either homosexual or heterosexual contacts.[7] In other settings one can take into account the factor of excessive frequency and promiscuity of sexual contacts, as with prostitutes; or the more obvious transmission of contaminated materials by the intravenous route. Darwin in all likelihood would have suggested that sooner or later, in one or more locations on this Earth, these situations would give a special environmental advantage to one or more variants within species of microorganisms. The inevitable results would be ready invasion of the host by the variant or variants, unchecked proliferation within the host, and a variety of destructive consequences, including widespread geographic dissemination. The AIDS virus appears to be such a variant, able to penetrate colonic cells and ultimately to spread to many different types of cells in the human body. It invades the vulnerable T-cell, often though not always destroying its capacity to defend the host, and leaves the host a prey for the invasion of still other microorganisms.

When this coincidence of circumstances involving unusual host exposure to an especially capable invader occurs, death of the host is inevitable, unless effective therapeutic interventions can be developed. This pattern of destructive invasion, in the course of which the invader kills the susceptible host and as a consequence destroys itself, is common to many epidemics; it suggests that the course of the AIDS "epidemic" may be self-limited over time, though at what ultimate cost in human lives and personal distress cannot be imagined.

Darwin might have suggested a corollary hypothesis. From the early beginning of the process of evolution, the more complex evolving life-forms must have been attacked by a broad spectrum of microorganisms. Those variants of these complex life-forms that developed the ability to cope effectively with such microorganisms were the ones that survived. One mechanism of coping which these variants developed could have been the immune system as we know it. Darwin very likely would have suggested that the apparently unusual organisms that we identify as secondary invaders in AIDS today—*Pneumocystis carinii*, *Mycobacterium avium-intracellulare*, *Candida*, cytomegalovirus, cryptosporidium, cryptococcus, toxoplasma, and others—are the same or similar organisms which millions of years ago were destructive to our early biological ancestors, until the latter developed variants with effective immunologic means of coping with them. Now that the HIV virus can destroy that same segment of the immune system which originally dealt with these organisms, we are again witness to the invasions and destructiveness of these same ancient predators.

We have much to learn about the process that leads to the production of variants within a species. Such a process brings with it many advantages for survival but also limitations on behavior. Of necessity, we must treat with respect those biological patterns which Nature has found most effective and most efficient for our survival in the present environment of this planet. This is not a moralistic precept; Darwin himself was attacked as a destroyer of human moral systems, even though it is likely that many moral codes originated, in part at least, in a wise accommodation to the requirements of Nature.

At some risk, we may by scientific effort alter Nature's patterns. Until we do, those of us who do not adapt appropriately to current patterns face the threat of extinction, to be replaced by life-forms whose biological conduct conforms to requirements worked out slowly and painfully by Nature over the last half-billion years.

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The morality of hospital stewardship*

As we totter toward our graves, some of us—perhaps from nostalgia—recall our roots. These may include the notion of stewardship, the idea that we are morally responsible custodians of resources and opportunities for which, on Judgment Day, we shall account. Is the world better because we lived in it? Did we add some small fragment to human knowledge? Were we kind and helpful? Were we generous with our knowledge, time, and skills? Did we, as Micah advised, do justice and love mercy?

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* Presented at the 1987 Annual CME Assembly of the Medical Society of the State of New York, March 13, 1987.

Physicians share a tradition that even if sometimes forgotten imposes very high moral demands. Ancient physicians were useful and well paid, but despite their considerable attainments, remained technicians. Early medieval religious writers, Christian and Jewish, emphasized our social obligations, and we gradually emerged as a responsible, learned, and merciful profession. We are inheritors of this considerable moral capital, and should we squander or alienate it, our claim to be a self-governing profession must inevitably collapse. Once again we will be prosperous technicians, hirelings rather than stewards.

Right or wrong, some of us suspect that what may seem to be economic problems are really ethical problems. This means that those of us who work in hospitals must be fair and thoughtful stewards of resources, which will always be limited, provided in trust by those who built these hospitals for our use. Nor need it surprise us that fairness and wisdom are as scarce in hospitals as elsewhere.

In honesty, we must say that butchers and grocers feel no imperative to feed poor people just because they are hungry; innkeepers and landlords to house them merely because they lack shelter; shoemakers, tailors, and haberdashers to clothe them simply because they are naked; nor do university professors teach without being paid. Yet physicians and hospital managers—generations ago—worked things out so that those in need get what we can do for them whether or not payment is available. The poor may and do starve, freeze, or die in the streets ignorant, and few voices protest. Let one sick child be turned away from the emergency room of a proprietary hospital and four hours later television reports it nationwide. Such a thing is news because it is so unusual. We remain judged by our own standards, as a profession should be and as ours is.

Physicians my age and older recall an informal covenant between hospitals, physicians, and society: Charge those who can pay enough so that those who cannot, or will not, pay can be cared for. In those less complex days, physicians and surgeons collected perhaps half of what they billed, yet most of them recall with pleasure and satisfaction their unreimbursed clinic, ward, teaching, and committee work. This was how it was and always had been; our dues as physicians included giving away a certain amount of professional care. The poor were taken care of but neither hospital nor physicians got paid. Physicians then earned two or three times and surgeons three or four times average industrial wages, they lived comfortably but not extravagantly, and their incomes were not resented. The system was paternalistic and maternalistic, informal and a cottage industry, but it was economical, efficient, had very strong grassroots support, and did not threaten to bankrupt the republic.

Then came the Forward Thinkers of the 1960s! Health care became a right and charity demeaning unless purified by passage from the taxpayers' pockets through a bureaucracy, preferably federal. Organized medicine demanded, and got, first dollar reimbursement and—in the beginning—"customary and reasonable fees"—under Medicare and Medicaid. But the Forward Thinkers forgot that everything is more complicated than most people think, and never understood or perhaps were unwilling to acknowledge just how much physicians and hospitals had always done for free, that hospitals' and physicians' charges had always included this "Robin Hood" factor, and that paying customary and usual fees in full would merely increase costs without increasing services.

For the first time in American history, there was enough money in the medical and hospital system to corrupt it. Anyone who could get his hands on a license to practice medicine could, if he wanted, earn a very handsome income. In our zeal to become entrepreneurs, we forgot that our survival as an independent profession demanded that we be responsible stewards of a very austere tradition. Many of us faltered, some of us failed.

A profession or guild—and we are probably more guild than profession, relying as we do on long apprenticeships called residencies—is by its nature autonomous. It controls training and admission to itself; it disciplines itself; and it monitors the quality and costs of what it does.

A climate of mutual distrust seems to be developing in hospitals: we versus them. *We* have to be very careful because *they* are out to get us, *they* are invading *our* turf. Physicians now murmur among themselves that they are losing, or have lost, control of their patients and that they are unable to influence adminstrative and policy decisions that adversely affect patient care. Some suspect that physicians abdicated this control, and that lay administrators only filled a power vacuum. But power is not easily surrendered once acquired, not in Washington, not in city hall, not in hospitals.

We are responsible for the qualifications and quality of those whom we license and allow to practice medicine. Our legal system tenderly protects sinners, but current medical discipline is a joke. It may be legally hazardous to write an adverse letter or recommendation about anyone. We may have to challenge this. Most physicians who come to media attention because of outrageously improper or incompetent professional conduct do so after years of doubtful professional behavior, well known to an entire hospital staff but never quite enough to document during the lawsuits that would inevitably follow staff disciplinary action. Only when some patients have been harmed can formal action be taken. Is a license to practice medicine, or hospital staff membership, a right or a privilege? Has society destroyed our capability to disçipline ourselves and our members short of a trial? Is this responsible?

We are, as medical staffs, required to oversee the quality of our own practice. As inheritors of an ancient guild tradition, perhaps we should rebel when various pressure groups demand that we share independent practice and, of course, independent billing privileges with nonphysician parapractitioners. What are we to do with the proliferation of others—acupuncturists, midwives, naturopaths, holistic counselors, nurse practitioners—who want to practice medicine without all the bother of medical school? We might meditate upon the possibility that the very popularity of fringe practitioners suggests certain inadequacies in our care of patients or our fee structures, and therefore failures on our parts. Care of patients is learned from role models, and we may have become too busy to be role models.

We are responsible for the quality of the care that we give, and we must maintain this responsibility to our patients. We can best accomplish this by scrupulous standards of practice and documentation, and by approaching medical staff committee assignments with a vengeance. Committee work is time consuming and unpaid. It is also an excellent way to show voluntary support for hospital and professional standards.

Voluntarism seems to be declining nationwide, and it is not clear that for-profit corporations or government can replace it. People generally do not give money for what they take already to be sufficiently supported from tax funds. Voluntary contributions and bequests to hospitals are drying up, but a more subtle and probably more dangerous part of this decline in voluntarism is waning community interest and involvement in hospitals.

Only since World War II have hospitals depended on patient fees for most of their income. At the moment, the future seems most secure for those hospitals that do least what good hospitals have generally tried to do best: treat sick people whether or not they can pay, solve diagnostic and therapeutic problems, teach, investigate, and write.

Hospital administrators, including physician administrators, in their more thoughtful moments are uneasy as to whether an industrial or commercial management model can or should be imposed upon so complex and traditional an entity as a hospital. Physicians are sure that it cannot and should not, but fail to be good stewards when they do not try to make a very imperfect system work. This failure only partially explains the hordes of administrative assistants involved in medical matters which they never quite understand, largely because physicians fail to get involved. Hospital administrators aim to give good patient care in a financially responsible way, because if the hospital goes broke it will close and be unable to care for anybody. As physicians, we once controlled professional matters in hospitals. We no longer do so. We have let amateurs take over our own administrative duties, leaving us free to make more money, but in so doing, we have gotten farther and farther from policy definition and decision making. Hospital administrators are generally conscientious and decent people who are attempting to do the impossible to manage a community social resource as a business. Abundant sensitivity, intelligence, good will, and knowledge cannot square the circle. Their tasks will be made less impossible by a constructive and responsible medical staff.

The schools and hospitals that trained us and the hospitals in which we work were built at considerable sacrifice, and not by us.

The *Newark Star Ledger*, on February 12, 1987, under the headline, "Medical Aid Program a 'Boondoggle,' " announced that New Jersey would have spent, during 1987, $10 million to deliver $1 million worth of services to the needy. The idea was excellent. The paperwork, regulations, eligibility requirements, and a staff of 200 to serve 1,800 beneficiaries was ineffective, and the state wisely abolished it. Somebody has to keep an eye on hospital and health program administrative overhead—administrators certainly won't—and who better for this cheerful task than physicians? We can do it only if, like honest stewards, our hands are clean.

What to do? Certain problems in the responsible use of hospital resources will remain insoluble. But for the deteriorating relationship between hospital medical staffs and their hospital administrations, answers are not simple—realistic answers to real problems seldom are—but might begin with recollection of physicians' traditional responsibility to be advocates for their patients, particularly in matters about which we know vastly more than patients can possibly know. Not very long ago, we decided a great deal about what went on in hospitals—only recently have assistant vice-presidents been able to veto the professional decisions of clinical directors or medical staff committees, to fail to inform these physicians of the vetoed decisions, and to expect to remain on the payroll much beyond noon of the next day! We can reaffirm and reestablish our stewardship over patient care. This may include confrontation, which we dislike, and may call for coalition formation, at which we are past masters. We would do well to accomplish this as an organized medical staff, not with individual trustees who are our patients or golf partners. The time, thought, and energy required will be considerable. Our motives must be, and must clearly be seen to be, independent of our incomes. Organized medicine currently lacks public confidence and support because it is perceived to be almost wholly concerned with the labor union aspects of our profession. If we can persuade our patients that we have their interests at heart, we will prevail in our determination to define medical policy and to monitor the quality of care, education, and medical practice. Our friends, the administrators, can then do what they have been trained to do—manage things—and we shall prove good and faithful stewards.

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Gastric ulcers

Gastric ulcers share common pathologic features with duodenal ulcers, but are epidemiologically quite different. There has been an increased incidence of duodenal ulcers in this century, related to urbanization. The incidence of gastric ulcers, however, is more closely related to the age and dietary changes of the population.[1] Prior to the 1950s, the incidence of duodenal ulcers increased steadily, while that of gastric ulcers declined, especially among the young. In the past 40 years, gastric ulcers in the Western world have not appreciably declined in incidence, except in countries in which residents have experienced a major change in diet.[2] Many factors contribute to the formation of gastric ulcers, including diet, drugs, and smoking. In the absence of poorly preserved or smoked foods, the underlying cause is commonly a decline in mucosal integrity associated with an age-related decrease in the stomach's acid-producing capacity, resulting in back-diffusion of hydrogen ions and cell destruction.[3]

According to national government vital statistics, during the 1970s there were about 240 peptic (duodenal and gastric combined) ulcers noted per 10,000 hospital discharges. In the 1980s, the rate for gastric ulceration has hovered at 55 per 10,000,[4,5] without evidence of decline.[6] Another study found that the hospital admission rate for duodenal ulcers in the 1980s has declined by 43%, and that of gastric ulcers by only 9%.[7]

Both duodenal and gastric pathologic processes may present as gastritis, chronic asymptomatic or symptomatic ulcers, bleeding, perforation, or cancer. Non-emergent cases are best diagnosed by endoscopy,[8] though barium studies may be helpful in following benign lesions.[9] Medical therapy is the treatment of choice for eight to 15 weeks before one resorts to surgery because of nonhealing.[10] Even after a gastric ulcer has healed, the mucosa is more permeable to acid.[11] This defect is almost certainly irreversible, and only resection of the vulnerable mucosa and/or a substantial decline in the stomach lumen's average acid concentration can reliably prevent recurrence. Lesser measures, such as discontinuation of offending medications and cessation of smoking, probably will tip the balance against ulceration in many patients. However, the underlying imbalance between acid production and mucosal atrophy can only be directly addressed by antacid therapy of some type. The relative long-term risk-benefit ratios of surgical procedures, histamine$_2$ blockers, and the new omeprazole family of potent hydrogen pump inhibitors are quite uncertain.[12] A report of carcinogenicity of the latter is particularly worrisome.[13]

A different approach to gastric ulcer therapy is cytoprotection—augmenting the resistance of the mucosa. Some agents, such as sucralfate, create mucosal shields against the lumenal acid.[14] Experimental prostaglandins increase the mucosa's resistance to attack by a number of agents,[15] decrease acid secretion,[16] and have been shown to be equivalent to ranitidine[17] and cimetidine[18] in the healing of gastric ulcers. These agents are highly promising, but their long-term effects are unknown, and they are not available in the United States. Certainly, however, the time seems near when virtually all gastric ulcers will be amenable to medical therapy, except those that present with a major surgical complication.

In this issue of the *Journal*, Cordone et al[19] present a well known but apparently rare complication of such an ulcer: posterior perforation with erosion of the splenic artery, resulting in massive hemorrhage. This is clearly an uncommon presentation. At City Hospital Center at Elmhurst, the collective surgical memory of 12 years recalls only one such case, out of approximately 300 operations for gastric ulcer. Surgical therapy has two aims, to treat the acute ulcer, usually by resection, and to decrease the acid-producing capacity of the residual stomach. Since most ulcers are conveniently located on the lesser curvature, an antrectomy which includes the ulcer, with or without a vagotomy, is effective.[20-22] After the operation, during which the vulnerable antrum has been excised, "normal" remaining gastric wall heals well. The stomach now has a greatly reduced average acid content, so the surgical morbidity and recurrence rates are both low. However, there are several difficulties: some ulcers are not easily excised; some patients are too ill for a major resection; and some patients have troublesome post-operative sequelae. Nevertheless, those procedures, such as vagotomy and pyloroplasty, that do not include gastric resection, have a higher rate of continued problems such as bleeding and recurrence.[23,24] Highly selective vagotomy with resection of the ulcer alone has been used, but recurrence rates will probably be high.[25] With improved medical therapy available to treat or prevent recurrence, long-term results may make it the procedure of choice. However, patients must be able to tolerate the higher risks, costs, and inconvenience associated with diligent medical follow-up.

In the case described by Cordone and colleagues,[19] a fatal complication occurred after ten days of post-operative medical therapy for severe anterior and posterior ulcer disease. The additional tendency of gastric ulcers to form, bleed, and perforate following surgery is well documented. In this patient, complications developed despite the partial protection offered by the use of cimetidine. The message is that the presence of gastric ulceration indicates a severe defect in the mucosa's resistance to acid, and that the utmost surveillance is mandatory to avoid complications.

The authors describe a method for controlling the aorta through the gastrohepatic omentum with an aneurysm clamp.[19] Additionally, the left triangular ligament may be incised and the left lobe of the liver reflected to facilitate exposure of this area.[26] This maneuver should be reserved for short-term use in hypotensive patients to gain time for fluid resuscitation, since it deprives the liver and kidneys of blood. Also, the splenic artery itself can be located here by following the left gastric artery back to the celiac, and can be ligated individually to control gastric-ulcer-related hemorrhage.

The inflammatory process is fundamentally a chronic one. Thus, both the healing time of an ulcer left in situ and the time to recurrence in susceptible but grossly normal mucosa are generally lengthy. What constitutes the best surgical therapy is difficult to determine because the methods of medical therapy will change during the long follow-up period that will accompany any study.

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A seroepidemiologic profile of persons seeking anonymous HIV testing at alternate sites in upstate New York

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ABSTRACT. Alternate sites for human immunodeficiency virus (HIV) counseling and testing were established in New York State in the late summer of 1985. In a six-month period at the beginning of 1986, 14.4% of individuals who received HIV test results were seropositive. Questionnaire data were obtained from 1,635 persons for development of an epidemiologic profile of attendees: most were white (83%), males (72%), born in the United States (94%), well (80%), who sought testing because they had risk factors (73%) or had had sex with persons at risk (26%). Higher rates of HIV seropositivity were found among blacks (26%), Hispanics (30%), males (16%), and those with a known risk factor (18%), and among those with symptoms (21%), than among those without these characteristics. Factors associated with HIV seropositivity are described.

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In his report to the nation, Surgeon General C. Everett Koop stated that "AIDS is a life-threatening disease and a major public health issue. Its impact on our society is and will continue to be devastating. By the end of 1991, an estimated 270,000 cases of AIDS will have occurred with 179,000 deaths within the decade since the disease was first recognized."[1] In that same report, Dr Koop also said that in some cases, it may be appropriate for individuals with histories of high-risk behavior to obtain a blood test for antibodies to the AIDS virus.

In April 1985, the Centers for Disease Control (CDC) established 55 cooperative agreements with state and local health departments to defray start-up costs of alternate testing sites. The purpose of the alternate sites is to make HIV antibody testing available (outside the blood banking system) to individuals wishing to learn their antibody status. Further, the nation's blood supply would benefit as the potential for false-negative donations would be reduced. By the end of 1985, 874 sites had been established in 53 project areas. During the first eight months, 17.3% of 55,500 individuals tested across the country were repeatedly reactive on enzyme-linked immunoassay (ELISA) tests for HIV.[2] While there has been a substantial commitment to the alternate site effort across the country, only a limited number of published reports are available documenting the activities at the sites.

The state of West Virginia established 11 alternate test sites at local health departments throughout the state. As of mid-July 1986, 414 persons had been tested for HIV at one of the sites. The positivity rate for the state sites was 8.5%. Men were twice as likely as women to test positive, with 8.5% of the men and 4.1% of the women being positive.[3]

Through March 1986, alternate sites in the Minneapolis-St Paul areas tested 2,812 blood samples for HIV—13.2% were positive.[4] For a six-month period ending in November 1986, the state of Alabama reported 18% positive of 729 people tested at alternate sites.[5]

During the early years of the AIDS epidemic, New York State accounted for half of all cases recorded nationally. As the disease has spread across the country, New York State has contributed a decreasing proportion of the cases reported to the Centers for Disease Control. As of October 12, 1987, New York State had 28.5% of the cumulative national AIDS incidence, reporting 12,012 cases since the beginning of the epidemic.[6]

This report describes epidemiologic variables and the HIV serologic status of those who attended alternate sites in New York State exclusive of New York City for the first six months of 1986.

METHODS

Shortly after the Food and Drug Administration approved an ELISA test for antibodies to HTLV-III, and with the assistance of a CDC cooperative agreement, the New York State Department of Health began developing a program to establish alternate testing sites throughout upstate New York (ie, New York State exclusive of New York City). Anonymous testing for AIDS antibodies became available to those wishing to know if they had been exposed to the human immunodeficiency virus (HIV). Protocols for counseling individuals at pre- and post-test sessions were developed, staff hired and trained, locations established, and announcement of the availability of the testing sites completed in a matter of months. The first counseling site opened in late summer 1985. To obtain testing, potential clients called a hotline number, spoke with a staff member, and scheduled a pre-test counseling appointment. If, based on the discussion at the pre-test counseling session, the client elected to have the test, a blood sample was obtained and a post-test appointment was scheduled a few weeks later.

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In upstate New York, for a six-month period starting in January 1986, a ten-item self-administered questionnaire was utilized at all the counseling sites (seven main alternate sites and seven satellite clinics). The main sites were located in Buffalo, Rochester, Syracuse, Albany, New Rochelle, Mineola, and Farmingdale. The satellite clinics were in outlying areas surrounding the cities or towns listed above. Clients who received antibody testing were asked to complete the voluntary and anonymous questionnaire. Questions focused on basic demographics, as well as risk-group status, reasons for wanting to be tested, self-perceived health status, and sexual contacts with males and/or females since 1978. Questionnaires were completed and forwarded to the central office at the time the blood sample was drawn. All blood samples were examined initially using an ELISA test marketed by Electronucleonics; those specimens that were repeatedly reactive were confirmed using a Western Blot kit manufactured by Organo Biotechnika. Laboratory results were available two to four weeks after the specimen was submitted. As a result it was necessary to link, via computer, the questionnaire data file with the laboratory result file.

RESULTS

During the first six months of 1986, more than 3,100 people scheduled appointments at alternate testing sites. During that same time period, 2,127 clients electing to have the test completed the two-step counseling process and received their antibody test results. Of these, 14.4% were seropositive. Questionnaires were completed by 1,754 (82.6%) of those receiving test results. Of these, 1,635 (93.2%) were linked to laboratory report forms. A comparison of the 120 forms without laboratory data and the 1,635 complete records revealed no statistical differences except on place of birth (5% more of the group without laboratory data were born in the US) and perceived risk group membership (9% more of the incomplete records were self-classified as risk group members).

Information on the 1,635 individuals attending the alternate sites for whom complete information was available showed attendees to be primarily native-born Americans (94%). Among those born outside the continental United States, Puerto Rico and West Germany were the most frequently mentioned locations, each with 0.6% of the total. Figure 1 presents the age distribution and seropositivity data on the client group. The age distribution of persons attending alternate sites is approximately the same as persons meeting the AIDS case definition. Seropositivity varied by age, with the 25–44-year-old group having the highest rates of seropositivity.

Seropositivity rates by race and gender are presented in Table I. Eighty-three percent of those seeking testing were white, 10% were black, and 4% were Hispanic. The positivity rates for blacks and Hispanics were more than double the rate for whites ($p < 0.001$). Each minority group differed significantly from whites but not from each other. Independent of race, males were 2.4 times more likely to be positive than females ($p < 0.001$). The differences in positivity rates by gender were most pronounced among whites, where the rate was more than four times greater in males than females ($p < 0.001$). Among blacks and Hispanics the rate among males was only about 1.5 times greater than that of females (the difference was not statistically significant). As of March 1986 (the mid-point of the study period), cumulative AIDS incidence figures for the state excluding New York City were 90.7% male and 9.3% female; 45.5% were white, 30.4% black, and 23.1% Hispanic.[7]

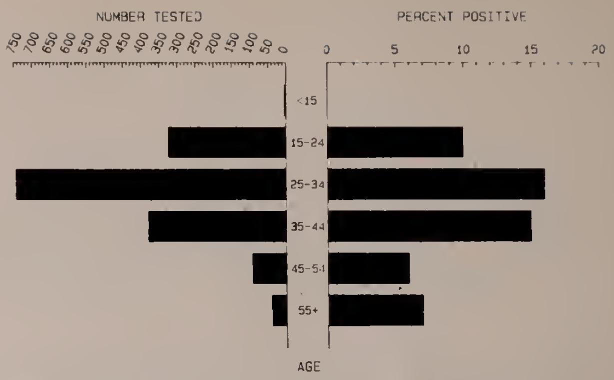

FIGURE 1. Number tested and seropositivity by age, upstate New York, January–June 1986 (42 respondents did not report their age; 19% were positive).

Seventy-three percent of clients reported having a risk factor for AIDS, and 17.5% of these were seropositive. Among the clients who did not believe they had a risk factor, 3.3% were positive. Among those indicating a risk factor, the factors cited in order of frequency were homosexuality (40.0%), intravenous drug use (21.8%), bisexuality (20.2%), blood transfusions (5.0%), and hemophilia (0.3%). Among males not reporting themselves as either homosexual or bisexual, 3.3% admitted to having had sex with a male since January 1978.

With regard to the reason for wanting to be tested, the most frequently cited reasons for testing for both men and women were risk-group membership (54.8%) or sexual contact with a risk-group member (39.1%). Family planning was listed as a reason for being tested among 6.4% of males and 8.3% of females. There were 23 women who indicated that they were pregnant at the time of testing. Two (8.7%) of them were seropositive. In response to the question concerning risk-group status, 11 of these women reported intravenous drug abuse and seven indicated a past history of sexual contact with a person who had AIDS. The racial distribution of pregnant females approximated the racial distribution of the population of alternate site attendees.

TABLE I. HIV Seropositivity by Race and Gender, Upstate New York, January–June 1986

Race	Male		Female		Gender Unknown			
	Tested	% Pos	Tested	% Pos	Tested	% Pos	Total	% Pos
White (Non-Hispanic)	1,022	13.5	303	3.0	36	25.0	1,361	11.5
Black (Non-Hispanic)	84	28.6	63	20.6	12	41.7	159	26.4
	(RR = 2.1, $p < 0.001$)		(RR = 6.9, $p < 0.001$)		(RR = 1.7, p, NS[†])		(RR = 2.3, $p < 0.001$)	
Hispanic	51	33.3	18	22.2	1	0	70	30.0
	(RR = 2.5, $p < 0.001$)		(RR = 7.4, $p < 0.001$)		(NA[‡])		(RR = 2.6, $p < 0.001$)	
Other/unknown	27	18.5	13	0	5	40.0	45	15.6
	(RR = 1.4, p, NS)		(NA)		(RR = 1.6, p, NS)		(RR = 1.4, p, NS)	
Total	1,184	15.5	397	6.5	54	29.6	1,635	13.8

[*] RR = relative risks—are gender specific using whites as the standard.
[†] NS = not significant ($p > 0.05$).
[‡] NA = not applicable.
Note: P values for the chi-square test are with continuity correction or Fisher's Exact Test. P values are not adjusted for multiple testing.

The overwhelming majority of clients attending the alternate sites reported their health status as "well" (80%). The seropositivity rate among those reported as "well" was 11.9%. One person indicated a diagnosis of AIDS, two a diagnosis of AIDS-related complex (ARC), and 38 did not report their health status. Two hundred ninety-four reported that they had one or more symptoms consistent with HIV infection, and their overall seropositivity rate was 20.7%. The reported symptoms, in order of frequency of report, were swollen glands (41.2%), cough (35.2%), sweats (34.7%), weight loss (27.6%), diarrhea (24.5%), and fever (19.7%). Seropositivity was highest in those reporting swollen glands (30.6%) and lowest among persons reporting diarrhea (11.1%).

Respondents were given the opportunity to report membership in as many risk groups as they felt appropriate. Table II lists seropositivity rates for males who listed a single risk behavior as well as some dual risk categories. Among those listing a single factor, intravenous drug abuse was associated with the highest rate of seropositivity (30.6%), followed by homosexuality (22.5%). The numbers of individuals indicating multiple risk groups were notably lower; the combination of homosexual contact and intravenous drug abuse produced a seropositivity rate in excess of 33%, but the absolute number of individuals was only six.

Analysis of the question concerning the reason for testing was performed for those respondents who listed a single risk behavior of either homosexuality, bisexuality, or intravenous drug use. Among the 383 males who listed only homosexuality as a risk group, 46.8% indicated risk-group membership as their only reason for pursuing testing. An additional 26.4% indicated risk-group membership plus sexual contact with a risk-group member. The response pattern for persons listing their only risk behavior as bisexuality was 46.4% for risk-group membership and 19% for sexual contact with a risk-group member (N = 179). Individuals listing only intravenous drug abuse as their risk behavior (N = 219) noted membership in a risk group as the reason for seeking testing 63% of the time, with risk-group membership and at least one other reason given by 26% of the respondents. Approximately 10% of both homosexual men and intravenous drug users failed to list risk-group membership as one of their reasons for seeking testing, while more than 24% of the bisexual men omitted risk-group membership as a reason for testing. It appears that intravenous drug users and homosexual men had learned of and recognized the risk behavior in which they participate. Bisexuals had not achieved the same level of awareness. At the mid-point of the study period, just under 60% of the cumulative male incidence was associated with homosexual or bisexual activity, and slightly more than 20% was related to intravenous drug use.[7]

DISCUSSION

For the period January to June 1986, the Alternate Site HIV Testing Program in New York State, exclusive of New York City, identified 306 individuals with antibodies to the human immunodeficiency virus. All persons receiving post-test results were given a similar message about safe sex and how to prevent further spread of the infection. Given the anonymous nature of the testing sites, the impact of the knowledge of HIV antibody status on future behavior cannot be assessed in the population tested.

Based on the substantial proportion of individuals identifying themselves as having engaged in high-risk behavior, it would appear that the alternate site program was attracting a substantial number of individuals at potential risk of HIV infection. This observation is reinforced by the similarity of demographic characteristics between persons attending alternate sites with those of persons with AIDS, and by the identification of a significant proportion of those tested as positive. It is important to note that in addition to identifying those who are HIV positive, the program also provided a large number of HIV-negative individuals with information on how to avoid/minimize the risk of coming in contact with the human immunodeficiency virus. Until effective prophylactic or chemotherapeutic agents are developed, education and encouragement of responsible sexual behavior remain the only tools available to control the spread of the disease.

On a programmatic level, data from the first six months' experience were useful in targeting services. For example, in March 1986 minorities and intravenous drug users made up a disproportionately high percentage of AIDS cases in upstate New York (54.5% and 53.1%, respectively),[7] but were contributing only 14% and 22% of the alternate site visits. This finding helped lead to the establishment of testing sites and increased education efforts in areas where these groups could be better served. In addition, the fact that 18% of persons seeking testing already had clinical symptoms reinforced the need to provide adequate clinical referral.

Blood donor studies have shown that some donors may acknowledge risk behavior, but may not believe they belong to a group at increased risk for infection.[8] Specifically, some seropositive males were found who did not consider themselves homosexual, but who had engaged in homosexual activity. This type of data lead to CDC's recommendation that all males with a single homosexual relationship since 1977 should self-defer donating blood.[9] As noted above, we found a similar phenomenon at the alternate sites, in that 3.3% of males not listing themselves as homosexual or bisexual admitted to having had sex

TABLE II. HIV Seropositivity by Self-Reported Risk Groups (Males Only), Upstate New York, January–June 1986

	Total Number Tested	Number Positive	Percent Positive	Relative Risks*	*p* Value
Not a risk-group member	242	8	3.3		
Homosexual	383	86	22.5	6.8	<.001
Bisexual	179	23	12.8	3.9	<.001
Intravenous drug abuser (IVDA)	219	67	30.6	9.3	<.001
Hemophiliac	2	0	0		
Blood transfusion	38	5	13.3	4.0	.02
Homosexual/bisexual	22	2	9.1	2.8	NS[†]
Homosexual/IVDA	6	2	33.3	10.1	.02
Bisexual/IVDA	6	1	16.7	5.1	NS

* Relative risks uses "not a risk-group member" as the standard.

[†] NS = not significant ($p > 0.05$).

Note: *P* values are for the chi-square test with continuity correction or Fisher's Exact Test. *P* values are not adjusted for multiple testing.

with a male since January of 1978. This reinforces the need for educational efforts to emphasize individual behavior rather than only risk-group membership.

Attention has been given to the role of intravenous drug users in heterosexual transmission. Within New York City, a high proportion of the cases of heterosexual transmission are thought to be associated with intravenous drug users.[10] However, among the upstate New York sample population, the observation that one quarter or more of the self-classified homosexuals had had heterosexual relationships since 1977 indicates that this group may also be responsible for spreading the virus, via sexual contact, into the female population. This reinforces the need to emphasize the safe sex message to the population at large and not just to those in high-risk groups.

As the demand for HIV testing grows, and the need to provide timely counseling continues, the New York State Department of Health is moving toward making counseling services available at many locations throughout the state. Family planning and sexually transmitted disease clinics will be offering the HIV test on a confidential basis, or referring individuals to settings such as physicians' offices and alternate sites where counseling and testing can be obtained. Physicians in private practice have been able to conduct confidential HIV testing since the ELISA test became available, and the number of tests has increased as the spectrum of diseases associated with HIV infection has been recognized. Education, counseling, informed consent, and confidentiality will remain integral parts of this expanded testing program.

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Teaching about AIDS in public schools: Characteristics of early adopter communities in Massachusetts

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ABSTRACT. Many teenagers are at risk for contracting AIDS because of their sexual activity and intravenous (IV) drug use. It is important that they be given information about the disease and its prevention. Some communities have taken early initiatives with respect to teaching about AIDS in their public schools. To determine whether or not there are attributes that would predict the likelihood of a community's integrating AIDS information into the public school curriculum, we explored 25 variables in a sample of 63 Massachusetts communities. None of the variables was found to be significant. We conclude that there are obviously other factors as yet unidentified, that explain why some towns teach about AIDS while others do not. Further research is necessary.

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Since October 1985 the US Department of Defense has routinely screened civilian applicants for serologic evidence of infection with human immunodeficiency virus (HIV), the virus that causes acquired immunodeficiency syndrome (AIDS). Results from the first 15 months of testing indicated an overall seropositive prevalence rate of 1.5/1,000 among a population consisting predominately of young adults in their late teens and early 20s.[1] Given a delay of up to several years between exposure and seropositivity, these data provide evidence of HIV infection among the US teenage population. Because applicants for military service may underrepresent those groups at highest risk for the infection (homosexual men and IV drug users), it is possible that the actual prevalence among teens is higher.[2]

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A 1985 survey of San Francisco students (14–18 years) assessing knowledge and beliefs about AIDS showed that 8% were unaware that sexual intercourse was one mode of contracting AIDS and that 40% were unaware that the use of condoms during intercourse may lower the risk of exposure to the disease.[3] A subsequent 1986 random digit dial survey of 860 Massachusetts adolescents 16 to 19 years of age indicated that 70% were sexually active, but only 15% of these reported changing their behaviors in order to reduce exposure to AIDS. Of the teens reporting behavior change, only 20% used methods currently considered effective for preventing AIDS transmission. Of all the respondents in this survey, 8% did not know that AIDS can be transmitted by heterosexual intercourse.[4] In the absence of a vaccine or medical intervention, the US surgeon general and the National Academy of Sciences have identified public education in schools as the primary means of containing the AIDS epidemic.[5,6] In response, many states are in the process of developing model curricula for AIDS education. In Massachusetts this activity has been undertaken by the state's Department of Public Health in conjunction with the Department of Education. This curriculum was presented to local school districts during the summer of 1987. Implementation is scheduled for the following school year.

Many local school districts have taken initiatives on their own with respect to incorporating AIDS education into their curricula. These programs range from one-time lectures to students by visiting health educators to more extensive course materials developed by local district staff. With the introduction of the state's model AIDS education program it is probable that within the next few years most Massachusetts middle and high schools will to varying degrees routinely teach about AIDS. Since AIDS is a relatively new phenomenon, observation of behaviors associated with the prevention of the disease provides the opportunity to study the diffusion of innovation throughout the population and institutions.

The objective of this study was to locate those school districts which were "early adopters" of AIDS education programs and to attempt to identify characteristics distinguishing them from school districts that had delayed teaching about the disease ("late adopters").

METHODS

Community Sample. We drew a sample of 63 Massachusetts communities weighted by population size and stratified by region of the state. In December 1986 the superintendent of public schools for each of the selected communities was contacted by either one of three of the authors (JH,JJ,JS). Superintendents, or school district personnel identified by superintendents, were queried about their schools' current AIDS education activities.

Variables. The dependent variable (AIDSED) for the analysis was whether or not a community's schools had, as of the date of the survey, incorporated information about AIDS into their curricula. This dichotomous variable (yes/no) was operationalized as follows:

- Formal instruction about AIDS including information on means of transmission was taught at at least one grade level (K–12) in a course that was required for at least 80% of the students.
- AIDS instruction was imbedded in school curricula as opposed to being delivered as a special, one-time presentation.

For the purposes of this inquiry, intermittent lectures, film showings, or the distribution of literature about AIDS were not counted as AIDS education measures unless they were part of a broader program of classroom instruction.

Twenty-five independent variables representing community characteristics categorized into six domains were explored to determine which if any were associated with towns that had opted to teach about AIDS. These domains were established arbitrarily by the authors to reduce the number of redundant variables for multivariate analysis. The reduction was required because of the large number of variables relative to sample size.

Data about the political characteristics of the community were derived from 1986 voter registration data and included the percent of registered Republican voters (REPUB), the percent of registered Democratic voters (DEMO), the percent of registered uncommitted voters (UNCOM), and the ratio of Democratic to Republican registered voters (RATIO). Geographic variables included State Department of Public Works districts (DIST), a measure of geographic location, and 1980 US census population size (POP). Economic variables consisted of median family income, 1980 percent of workforce in manufacturing (MANU), and 1985 per capita school expenditures (EXPEN). The demographic variables were all derived from 1980 US census data and included the percent foreign born, the percent having changed residence in the previous five years, the percent speaking a language other than English at home, the percent black, the percent with Hispanic surnames, the percent Asian, and the percent white. Health variables were intended to measure several dimensions reflecting community health behaviors or status. These included fluoridation of town water supply as of 1986, the percent of voters voting in 1986 to maintain the state's seatbelt law, the percent of voters voting in 1986 not to limit Medicaid funding for abortion, cumulative incidence rates for AIDS as of November 1986 in the county in which the town was located, and the teenage birthrate for 1985. Education variables consisted of 1980 census measures for the percent of 3–5-year-olds enrolled in kindergarten, the percent of 18–24-year-olds enrolled in college, the percent of residents over 25 years of age who were high school graduates, and the percent of residents over age 25 who were college graduates.

Analysis. Analysis of the association between early adoption of AIDS education and community characteristics involved several steps. First, univariate analyses of variables were performed. T-tests were used for continuous variables and chi-square was used for dichotomous variables to determine the significance of differences between towns that did and did not teach about AIDS in public schools. Second, on the basis of the results of the univariate analysis, variables were selected from each of the community characteristic domains for inclusion in a multivariate logistic regression model. A stepwise logistic procedure was used with the p value for variable entrance and exit from the model set at $p = 0.15$.

RESULTS

All of the 63 school districts contacted were willing to provide information on their AIDS education activities. Thirty-five (56%) were teaching about AIDS to an extent sufficient to meet our criteria as early adopters. School districts that had had one or more special presentations on AIDS, distributed literature about AIDS, or were planning AIDS curricula were not included in the early adopter category. It should be noted, however, that among school districts that were categorized as teaching about AIDS, there was a great deal of variation with respect to the amount of material and the grade levels taught.

Results of the univariate analyses are presented in Table I. Of the 16 political, economic, and demographic variables explored, only one (% ASIAN) attained significance at the $p \leq 0.05$ level. Several other demographic variables were suggestive, with p values of 0.10 (% BLACK and % changing residency in previous five years). All three of these variables were positively correlated with AIDSED.

Among the health variables, only the percentage of voters sup-

TABLE I. Association Between Community Characteristics and Early Adoption of AIDS Education Measures: Univariate Analysis (N = 63 communities)

Domain/Variable	t-test (p value)	chi-square (p value)
Political		
% Uncommited	.70	—
% Republican	.66	—
% Democratic	.97	—
Ratio	.93	—
Geographic		
Population	.22	—
District	.32	—
Economic		
% Manufacturing	.54	—
Median family income	.78	—
Expenditures	.16	—
Demographic		
% Foreign born	.30	—
% Moved 5 years	.10	—
% Speak other languages	.72	—
% Black	.10	—
% Hispanic	.97	—
% Asian	.05	—
% White	.43	—
Health		
Fluoride	—	.21
Seatbelt	.46	—
Abortion	.08	—
AIDS cases	.83	—
Teen births	.47	—
Education		
% In kindergarten	.78	—
% In college	.41	—
% High school graduates	.44	—
% College graduates	.32	—

porting Medicaid funding for abortion (ABYES) approached significance ($p = 0.08$). AIDS education was more likely to be done in those communities that had voted not to limit Medicaid abortion funding.

None of the variables measuring community education levels were significantly associated with AIDS education.

Ten variables were selected for stepwise logistic regression on AIDSED. These were selected from each of the variable domains on the basis of univariate analysis. The only variable retained by the stepwise process was percent voting to support Medicaid abortion funding, using an entrance p value of ≤ 0.15. The p value for this variable was 0.08. The overall chi-square for the regression model (3.39, 1 df) was not significant ($p = 0.07$).

DISCUSSION

There are several reasons why it might be useful to be able to predict early adopter communities with respect to policies related to the AIDS epidemic. Foremost among them is the targeting of public health resources to encourage communities to institute AIDS prevention measures. At present state governments are focusing on AIDS education in the public schools. However, as the incidence of AIDS increases, other local institutions (eg, emergency medical squads and police departments) will be called upon to adopt new preventive policies. Knowing in advance which towns will adopt early and which will resist these changes could be valuable in allocating education and training resources.

Although this study was primarily exploratory, we proceeded with several assumptions. We supposed that communities with higher levels of education and/or family income would be more likely to be early adopters of AIDS education. In so doing we applied observations derived from behavior at the individual level (eg, income and education are positively correlated with personal health behaviors) to aggregate level characteristics. We also assumed that a community's fiscal capacity, as measured by per capita school expenditure, would be a factor in early adoption. We supposed that a town's concern with health issues generally, as measured by fluoridation status and voting in support of seatbelt legislation and abortion funding, would be reflected in the propensity to adopt AIDS education measures early. Finally, we supposed that exposure to the need for AIDS education, as measured by the regional AIDS incidence rate, might influence adoption.

None of these hypotheses was confirmed by this investigation. This result has several possible interpretations. As a cross-sectional study, the behavior of school districts was measured at one random moment in time. It is possible that the study was conducted too late in the adoption process; that early adopters could have been differentiated had we identified them at a point when only 10–20% rather than 56% of the communities had initiated AIDS education activities.

More than half of the school districts we surveyed had already adopted AIDS education programs. Others were planning to do so. Thus, it may be that all schools were in fact in the process of adopting AIDS education programs and their particular point of progress was due to a variety of random factors. This interpretation would suggest that within the context of AIDS education in the public schools, the concept of early adoption is irrelevant.

Our sample was small. Accordingly, it is also possible that the failure to detect significant differences between communities was due to lack of statistical power.

It is possible that in this context early adoption is due to the efforts and concerns of a small number of individuals who are themselves early adopters and are in a position to influence policy. In other words, whether or not a community adopts AIDS education early is not the result of consensus in the aggregate community. This would suggest that characteristics of individuals rather than populations would predict the behavior of towns. If such individuals were randomly distributed, ecological community data would not be predictive of local policies. This interpretation would be consistent with the findings of early studies on the dissemination and adoption of agricultural techniques in the American West.[7]

For the foreseeable future, behavior on both the individual and community levels is the single key to the prevention and control of the AIDS epidemic. It is likely that local communities will be encouraged to adopt an increasing number of AIDS-related policies. By understanding the dynamics of the adoption process we can hope to accelerate required change. This study is inconclusive, but our results suggest that ecological data do not predict the behavior of communities with respect to early adoption of AIDS education measures. If this result can be generalized to other AIDS policies, the implication is that efforts to bring about change in communities should focus on identifying and encouraging community leaders. Further research focusing on case studies of AIDS-related policy adoption is required to confirm this interpretation.

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OBSERVATIONS

The highest form of service which the medical profession can perform for mankind is in the prevention of diseases. This is a field which is entirely its own. In the other departments of medicine we come within the realm of the other sciences, such as biology, chemistry and botany, but prophylaxis is distinctly the function of medicine. Medicine can never hope to be of as much value in the treatment of diseases as it can in their prevention. Cholera, small-pox, puerperal septicaemia, yellow fever—it is here that humanity owes its greatest debt to medicine. But curiously, treatment must precede prophylaxis; for it is in the long and futile period of treatment that an intimacy and a familiarity with the disease is bred, out of which grows the knowledge of the disease which makes possible successful prophylactic measures.

It is in the study of the prevention of disease that medicine has no rivals and is the least apt to be misunderstood. This realm is not invaded by the charlatan or the pseudo-scientific sect. The "schools" of medicine have not to do with prophylaxis. They are not interested here. Intelligent and scientific effort in the prevention of disease is tangible and can be measured, and is freer from confusion than is therapeusis. In the treatment of diseases the forces of nature are just as kind to the mercenary quack, with his "incomparable elixir," as they are to the conscientious homeopath, with his harmless waters, who hopes only for the health of his patient. The patients of both will recover; and so, too, will the patient who is given some drug according to the most approved and rational indications. The first two will be done no damage; the last may be either harmed or helped; but misunderstanding and credit misplaced have been always the accompaniments of the art of treatment.

What has been done with cholera can be done with typhoid fever. The fifteen hundred persons who died of this latter disease in this State during the past year are a needless sacrifice, for medicine has developed the knowledge which, if applied, would make typhoid fever an obsolete disease. Politics alone stands in the way. If the medical profession were given *carte blanc* power to eliminate typhoid, it would be done. Medicine has perfected the knowledge of this disease and the means for its prevention, and the people want the disease stopped, but the legislative representatives of the people are busy with mergers and tariffs and appropriations and jobs, while the pale faces of those fifteen hundred, dying of a preventable disease, are seen only by the profession of medicine. When some community is awakened by an awful epidemic, it arouses itself, and some petty local measures are applied, always successfully, to prevent the disease; but what is done for a country village should be done for the State, for the country and ultimately for the world—and the disease would end.

This will be done—it should be done now, but it will not—and then typhoid will pass into history, a conquered disease. In the meantime, *your* son, on the threshold of life's work; and *your* daughter, with the roses of health in her cheeks; and *your* wife, the mother of the laughing babe; and *you*, to whom these observations are addressed, shall return in the autumn, and lie down on your bed and die, for the mergers must be put through, and the tariff must be tinkered, and the appropriations must be passed round, and the jobs are meat and drink.

EDITORIAL
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Barriers to the modification of sexual behavior among heterosexuals at risk for acquired immunodeficiency syndrome

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Acquired immunodeficiency syndrome (AIDS) may no longer be viewed as a disease that only poses a significant threat to homosexual men and intravenous drug users. About 7% of cases diagnosed in 1986 were from among three groups in which the majority of cases are thought to result from heterosexual transmission: heterosexual contacts; persons born outside of the US; and persons with no identified risk factor. The proportion of cases accounted for by these groups is projected to increase to 10% in 1991.[1] If one considers only the cases acquired through heterosexual contact, it is projected that the proportion of all cases in this category will increase from 2% in 1986 to 5% in 1991.[1]

It is difficult to arrive at a reliable estimate of the prevalence of human immunodeficiency virus (HIV) infection within the heterosexual community. One strategy for deriving such an estimate from available data yields a rate of HIV infection of 45 per 100,000 (or 0.045%) among the US adult population with no known risk factor.[2] In a sample of 185 self-described heterosexuals attending a sexually transmitted disease clinic, two, or slightly less than 1%, were positive for HIV.[3] Among 134 members of a Minnesota-based social/sexual club, none of the 75 male members and two of the 59 female members tested positive for HIV antibodies.[4] This constitutes a seroprevalence rate of 3% among the female club members. When 92 prostitutes in Seattle, Washington, were tested, 5% were found to be HIV antibody positive; similarly, when 25 prostitutes in Miami, Florida, were tested, 40% of this group were seropositive.[5] In one study,[6] testing of seven female sex partners of men with AIDS or AIDS-related complex (ARC) yielded HIV antibody in 71%. Another study of 42 women who had had sex with men suffering from AIDS or ARC found a 47% seropositivity rate.[7] Finally, of 21 female sex partners of men with hemophilia who were either HIV positive, had lymphadenopathy, or had AIDS, 10% tested seropositive themselves.[8]

Whatever the true prevalence of infection, it is reasonable to assume that the rate is higher in cities with large, established risk group populations (since it is in these cities that the greatest opportunity for transmission to heterosexuals exists). The rate of infection would also presumably be higher among individuals who have a pattern of sexual behavior that includes unprotected intercourse with multiple partners; and higher among those subgroups that traditionally have been regarded as risk groups for other sexually transmitted diseases (STDs) such as syphilis, gonorrhea, hepatitis, and herpes. A higher infection rate would also be expected among blacks and Hispanics, who are greatly over-represented among AIDS cases in the United States, especially in the categories of heterosexual contacts, women with AIDS, and perinatal transmission.

It is now generally accepted that bidirectional heterosexual transmission can occur, although female-to-male transmission is less well documented and may be less efficient.[5,9-16] It is also recognized that many, if not most, infected heterosexuals are probably unaware of their status and therefore are unlikely to be taking precautions to protect their sexual partners.

The AIDS epidemic has progressed to such a point that all sexually active individuals not in a long-standing monogamous relationship—regardless of sexual orientation—must be considered potentially at risk for the disease. However, there is evidence that many heterosexuals likely to be at increased risk for AIDS are not altering their sexual behavior. A recent study [17] of a random probability sample of high-risk adults in San Francisco (defined as having had two or more partners in the past year or awareness of sexual relations with a homosexual or bisexual male, an intravenous drug user, or a prostitute) found evidence of widespread persistence in recognized risky behaviors. Of those respondents who had ever engaged in vaginal intercourse without a condom, 59% said that they currently did so about as often as in the past, while 22% did so even more often. Of those who had ever had sex with a homosexual or bisexual man, 28% said they currently did so as often as in the past, while 11% said they did so more often. When asked to rate on a scale from 1 to 10 how much impact the AIDS epidemic had had on their sexual behavior or lifestyle (1 = no impact at all, 10 = a great deal of impact), 68% of the respondents chose a

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score of 5 or less; a full 33% selected 1.

These data suggest that many heterosexuals at increased risk for HIV infection do not yet recognize their susceptibility or are not motivated to adopt behavioral modifications, circumstances that create the potential for significant spread of the virus in the general population. In response to this realization, we have begun to see a shift in public health educational efforts toward a greater emphasis on targeting sexually active heterosexuals. As these preventive activities begin to accelerate, it is useful to ask what barriers might impede the adoption of safer-sex recommendations among at-risk heterosexuals. This article focuses on an analysis of those barriers. Recommendations for surmounting or alleviating existing obstacles to change and for more effectively achieving adoption of safer-sex recommendations are also offered.

BARRIERS TO BEHAVIOR MODIFICATION

Perceptions of Low Vulnerability. Most models of preventive health behavior regard a feeling of personal susceptibility to an illness as a necessary condition for an individual to adopt a preventive health action.[18] That is to say, most heterosexuals will not be motivated to modify practices that could place them at risk for infection with the AIDS virus if they do not perceive themselves to be vulnerable to the disease. It has already been documented that a general tendency exists for people to believe that they themselves will not be victims of negative life events—including serious illness.[19-21] People tend to distort reality in a positive direction in order to avoid the anxiety that would result from a more realistic assessment of their vulnerability. Thus, apart from other factors (discussed below) that may contribute to a faulty appraisal on the part of many heterosexuals of their risk for infection, public health educational efforts must also confront a common defensive mechanism that fosters an unfounded optimism regarding personal invulnerability to health (and other) threats.

Unfortunately, several features of the way we have communicated about AIDS both in the media and in public health messages may have inadvertently contributed to a perception on the part of many heterosexuals that they have little cause for personal concern. For example, the widespread emphasis on anal intercourse as a principal mode of sexual transmission may lead many heterosexuals who refrain from this practice to evaluate their risk of infection as being low. Similarly, the emphasis early in the epidemic on a large number of partners as a risk factor may have led many who have had only a small number of partners to feel unthreatened by the disease. In reality, of course, while the probability of infection increases with the number of partners, the possibility of becoming infected exists with even a single partner if unprotected sex occurs.[22] The focus on "risk groups" as opposed to "risk behaviors" is still a feature of communications that contributed to a false sense of security among many who engage in practices that are associated with transmission of the AIDS virus.

Further, people also evaluate their risk of experiencing a negative life event by employing stereotypes, or what Tversky and Kahneman[23] have called the "representativeness" heuristic. That is, people tend to have a preconception in their minds of the typical sort of person who experiences a particular kind of negative event—such as acquiring a venereal infection. They appraise their own vulnerability based on their evaluation of how much or little they resemble their mental representation. Because the victims they imagine tend to be stereotypes, they generally judge themselves as very different and therefore at low risk.

Misperception of the Efficacy of Adaptive Behaviors. While AIDS is a relatively new disease, sexually transmitted diseases have been around for centuries. In 1917, the Venereal Disease Control Division of the Public Health Service was founded to stem the spread of STDs. It has become apparent in the intervening years that while a variety of prophylactic methods (social, mechanical, chemical, and systemic) have been identified, significant barriers to their utilization exist.

In the case of AIDS, the only prophylactic measures believed to be potentially effective at this time, if one continues to engage in risky sexual practices, are reducing one's number of partners, being more selective in the choice of partners, and using condoms.

The reduction of partners, in the absence of condom use, is only effective if the rate of infection in the population from which one selects his or her partners is still quite low. If, on the other hand, the prevalence of infection is high, little protection is conferred by this strategy. For example, in cities with large homosexual male communities, where it is estimated that the rate of infection may be as high as 68%,[24] reducing one's number of partners from, say, 20 to two, still presents a 90% risk of exposure.

Being more selective in the choice of one's partners implies that one has a means of evaluating the probability that a potential partner is infected. However, this necessitates an extensive knowledge of the potential partner's sexual and medical history: Is he or she a member of an established risk group; has he or she been the partner of a member of an established risk group; has he or she had multiple partners during the past several years; has he or she had a blood transfusion during the past several years? Few individuals are likely to possess such knowledge of prospective partners, or to feel comfortable obtaining this information.

Some heterosexuals believe that if they can feel confident that their partner is not a bisexual male or an intravenous drug user, they run little danger of infection. Others believe that if in addition to avoiding these risk group members they also select only partners who are white, middle class, and who hold professional or white collar jobs, they are unlikely to risk exposure to the AIDS virus.

The inadequacies of these strategies for avoiding infection are obvious. First, it is not possible to reliably determine who currently, or within the past ten years, may have engaged in homosexual behavior or intravenous drug use. Many homosexual men have been able to "pass" their entire adult life because they do not fit the stereotypical image of a gay man. Similarly, as our awareness of the pervasiveness of drug use in our society has grown, it has become evident that there are many "respectable" people who have used drugs for years without friends or coworkers learning of their behavior.

Similarly, social class variables are not reliable screening criteria for partner selection. Drug abuse and bisexuality occurs among all social classes, races, and ethnic groups. Recipients of contaminated blood, who are unaware that they may be infecting others, are also represented in every social strata as well as all races.

Barriers to the Adoption of Condoms. Condoms offer a more useful method of controlling the spread of HIV infection. Their efficacy in preventing many of the traditional STDs is already well documented.[25,26] Preliminary data, while inconclusive, also seem to support their efficacy in preventing the transmission of the AIDS virus.[27] It remains to be seen whether heterosexuals will be more motivated to use condoms to prevent AIDS than they have been to use them to prevent other venereal diseases. For despite condoms' efficacy and safety as a technique of STD control, estimates of condom use among STD clinic patients range only from 3% to 20%, and among the general male population, condom use usually does not exceed 25%.[25]

The barriers to the use of condoms have been well documented. These include the following: the belief that condoms compromise the pleasure of intercourse;[28-30] the tendency to view the condom primarily as a contraceptive device rather than as a means of prophylaxis against STD; [31-32] the belief that condom use is unnatural; [28-29] the tendency to underestimate the personal risk of infection present in a situation; [25,33,34] the failure to anticipate and/or prepare in advance for sexual activity;[25,33-35] the belief that one's partner would be offended if a condom were introduced; [32,33,36,37] the belief that safe, effective treatment is available and, therefore, prevention is not important;[31,33] the use of alcohol or drugs before or during sex, which leads to a failure to use condoms or improper usage; [33-35] the stigmatization of condom use through their popular associations with promiscuity, prostitution, and extramarital sex;[37-40] the belief that condoms are ineffective or unreliable;[25,41] the embarrassment or discomfort of purchasing condoms;[42-44] the belief that using condoms makes sex seem premeditated and not spontaneous.[40]

Confusion Regarding the Magnitude of the Threat to Heterosexuals. While there has been intermittent discussion in the mass media about the potential danger of the growing spread of AIDS beyond the established risk groups into the wider heterosexual community, there have also been periodic reassurances that the proportion of AIDS cases accounted for by non-risk-group members has remained relatively stable over time. Such seemingly conflicting messages have resulted in a sense of confusion among many regarding the actual magnitude of the risk of infection to most heterosexuals.

Similarly, while bidirectional transmission of the virus is accepted as possible, the relatively small number of cases of female-to-male transmission that have been documented has contributed to the perception that heterosexual males may incur a low risk of infection from unprotected intercourse. Because it is still often left to males to introduce the use of condoms, a man's low sense of vulnerability may serve as a barrier to his doing so.

The Interpersonal Nature of Sexual Activity. Because of the interpersonal nature of sexual intercourse, decisions to engage in or refrain from risky sex must be "negotiated." To effectively practice safer sex the partners must agree on limits and the use of condoms whenever the opportunity for the exchange of body fluids exists. Whereas with most other health-related behaviors it is only necessary to persuade a single individual to change his behavior to bring about the desired outcome, in the case of "unhealthy" sexual behavior, two individuals must be convinced of the necessity of modifying their actions. When one partner is not motivated to practice safe sex, he or she may not cooperate with the other partner in having a safe encounter. Or the unmotivated partner may undercut the resolve of the other partner to avoid unsafe sex by persuading him or her that no risk exists in unprotected intercourse or by failing to cooperate in the proper use of condoms.

The Stigma of AIDS. Sexually transmitted diseases have always been stigmatized in our society. Venereal infections are widely regarded as the outcome of sexual excess and low moral character. Historically, these diseases have been associated with dirt and uncleanliness.[45] So strongly are sin and moral depravity tied together with STDs in the popular mind that, as has been pointed out, victims of these diseases feel compelled to deny their condition or assert their innocence (eg, "I got it from a toilet seat.")[46]

AIDS, of course, has been no exception. In fact, its association with two of the most stigmatized groups in society—homosexuals and drug abusers—has only served to accentuate the stigma attached to the disease. As a result, the need to dissociate oneself from AIDS or even from the implication that one could possibly be infected may be very great. If introducing a condom might be construed to mean that one acknowledges that one may be at risk, individuals may be unwilling to do so. Further, if doing so is felt to impugn the character of one's partner, one also may resist the use of condoms.

The stigma associated with AIDS may also make individuals unwilling to seek out information—including prevention-related information—about the disease. The wish to deny that the problem has any personal relevance is so strong among many that it may serve as a barrier to their obtaining needed education about the disease.

HEALTH EDUCATION IMPLICATIONS

As can be seen from the foregoing discussion, several barriers exist to heterosexuals' adoption of safer sex practices. Taken singly and in combination, these barriers appear to be formidable, indeed, and overcoming them will not be an easy task. Nevertheless, we present several recommendations for removing or reducing the obstacles to behavior change.

Careful attention must be given to the varying meanings and implications individuals will assign to certain language that may be used in educational materials. For example, communications should focus on risk behaviors (eg, engaging in intercourse without a condom) rather than risk groups (eg, homosexual or bisexual men and intravenous drug users). Talking about risk groups permits most of the public to defend against a sense of personal vulnerability, which may be necessary for the adoption of behavioral modifications; because these individuals do not belong to or identify with the groups, they will not identify with the threat. For the same reason, health educators must work to break down traditional stereotypes about the kinds of people who get sexually transmitted diseases. Such myths may lead many at-risk individuals to underestimate their own susceptibility.

The use of the terms"multiple partners" and "sexually active" are other examples of language that may seem clear and straightforward to health educators but has different meanings to different individuals. It is important to clarify for the public that "multiple partners" refers to two or more partners in a specified time frame—eg, within the last year. It must also be understood that "multiple partners" refers not only to situations in which the individual has two or more partners concurrently, but also to a pattern of serial monogamy, in which the individual has only one partner at a time, but each for a relatively brief period of time (eg, less than a year).

Finally, the term "sexually active" will be interpreted in varying ways. Because early in the epidemic AIDS was identified with homosexual men practicing fast lane" sex, characterized by a large number of partners, AIDS has become associated in the popular mind with sexual promiscuity. As a result, many people will equate the expression "sexually active" with "promiscuous" or having multiple partners. Thus, when they hear that sexually active heterosexuals are at risk and must adopt certain prophylactic actions, they may assume that the messages do not apply to them. If by sexually active we mean, for example, anyone with two or more partners during the past year, we should state that. It is also important to recognize that patterns of sexual behavior and sexual norms may vary somewhat across racial and ethnic groups. Individuals will appraise how risky or active their behavior is by comparing it to the prevailing norms of their social group. If they regard themselves as only typical or even less active than typical, they are likely to judge themselves as sexually conservative and at little risk for AIDS or other STDs regardless of a pattern of multiple partners.

The efficacy of condoms as a prophylactic for STD must be emphasized, and their accessibility, especially to those who are most likely to have unprotected sex, should be increased. Availability of condoms has been shown to influence their use. For example, one study found that distributing condoms to a group presumably at risk for STD (inner-city adolescent males) resulted in an overall increase in condom use from 19% to 68%, with use in the last sexual encounter increasing from 20% to 91%.[47] Health educators also need to promote a change in image for condoms. As was noted above, condoms are often associated in the minds of many with promiscuous or "illicit" sexual activity. Further doubts exist concerning their reliability. Individuals must be shown that proper and consistent use of condoms provides not only contraception, but also an effective and generally reliable prophylaxis for STD. Additionally, through statements by health educators and in the media, condom use must be shown to be acceptable and appropriate among heterosexuals in stable relationships. Finally, since in some cities women now purchase approximately 40% of the condoms sold,[48] this information should be directed at women as well as men.

As was noted above, individuals tend to assume that they can evaluate the riskiness of a potential sexual encounter and will consequently take or not take appropriate preventive action based on these evaluations. The language employed in some health recommendations may promote this tendency. For example, a communication may tell individuals to use condoms if they know or suspect that a potential sexual partner is a risk-group member or has had sexual contact with a risk-group member. This encourages individuals to rely on their judgment or intuition to evaluate the potential risk a prospective partner is likely to represent. Such judgments will usually be based on distorted traditional stereotypes of the "types" of people who are likely to be risk-group members or to contract an STD.

Rather, individuals must be shown that *anyone* can be infected, that they cannot rely on their judgments, and they should thus take appropriate prophylactic steps such as condom use on every sexual encounter. They should also be made aware that they cannot take a sex history and thus rule out risky partners. This is a flawed and unreliable method for assessing risk. Even assuming a potential sexual partner is able (and willing) to recall and relate everything he or she has done sexually in the past seven to ten years, it is extremely unlikely he or she will know about the behaviors and riskiness of his or her past partners.

As has been done in the male homosexual community, heterosexuals should be educated in the process of negotiating limits with potential sexual partners. They must be given the skills to communicate effectively with their partners about their unwillingness to engage in certain risky sexual practices. The greater assertiveness of women in interpersonal matters that has emerged in recent years should be encouraged in sexual negotiations. Both men and women should feel they have both the right to insist on protection as well as the responsibility for providing appropriate prophylactics. Both should be instructed in the proper use of condoms. Above all, this must be done, not in an atmosphere of suspicion, but in one of mutual respect, concern, and caring.

One must, however, be sensitive to the special cultural barriers that may exist. For example, within the Hispanic subculture, sex roles in sexual matters are more rigidly prescribed. For a woman to appear too knowledgeable about sexual matters can call her character into question. Special strategies must be developed for these communities. Grass-roots involvement of community leaders will be essential to the success of educational efforts.

Finally, individuals must be shown and convinced that AIDS and other STDs are no respecters of age, income, race, or social class. Not only poor, non-white, "outgroup" members, but also wealthy, white, and socially well-positioned individuals have contracted and died of these diseases. To this end, AIDS must be destigmatized and its depiction as a disease limited to gay men and drug abusers shown to be false. Perhaps as AIDS continues to move beyond these stigmatized groups, the disease itself may lose some of its stigma. This may largely be a function of time. However, to the extent that health educators are responsible for enlightening the public, they can perhaps focus on showing that AIDS is not just a disease of homosexual men and drug abusers. AIDS sufferers deserve neither moral condemnation nor mere pity, but support and concern. AIDS is not due to a weakness in character, but is a blood-borne disease that happens to be transmitted by sexual contact.

It addition to this health education approach, another source of health communications can be opened. This would involve the use of "opinion leaders," well known and respected individuals whom the public trust and to whom they will listen. Because these individuals are respected, the public is interested in their views on controversial matters and will often seek to emulate their values and attitudes. We should explore the use of opinion leaders to raise public awareness of the problems AIDS poses, to encourage a collective sense of purpose in combating the epidemic and in reshaping sexual norms.

CONCLUSION

Although a reliable estimate of the prevalence of HIV infection among the general heterosexual population (excluding risk group members and their partners) is not available at this time, it is likely that it is still quite low. This circumstance should not promote a sense of complacency, but rather should be regarded as representing an opportunity to significantly limit the spread of the epidemic.

As described in this article, multiple barriers exist to persuading at-risk heterosexuals to modify sexual behaviors implicated in the spread of infection. While knowledge about risky and safe practices is a prerequisite to the adoption of safer sex practices, it is not usually a sufficient condition. As we have shown, many of the barriers to behavior change that exist are perceptual and attitudinal. This means that health education in the case of AIDS must mean more than merely transmitting information, it must also alter perceptions and modify attitudes. We have offered recommendations for accomplishing these objectives.

It appears that the AIDS epidemic will be with us for some years to come. Unfortunately, at this time little empirical data exist for guiding our health education efforts aimed at the general population. Research on the factors that influence heterosexuals' tendency to modify their sexual behavior in response to the threat of AIDS must be assigned a high priority by the government. We need fundamental data on people's high-risk sexual practices, beliefs, and perceptions regarding AIDS to mount an effective national public health campaign.

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The physician's patient-centered ethical imperative: Implications, obligations, and problems*

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The Great Seal of the Medical Society of the State of New York gives 1807 as the society's date of organization, but its roots go back a good bit beyond that. The record shows that it has often been a voice crying in the wilderness, a bellwether society, and not solely a professional group given mainly to ritual and self-interest. The *Oxford English Dictionary* defines *bell-wether* as a chief or leader; but also as "a clamorous person, one ready to give mouth." Both definitions have, from time to time, applied to the society and the great hope is that they still do.[1-3]

The society's concern has all along been a mixture of organizational rules alongside more ephemeral matters that focus on meeting the patient's needs and that are by their nature concerned with ethics. From the latter, many current authors derive what has been defined as the physician's patient-centered ethical imperative, a principle that requires the physician to place the patient's interests, welfare, and legitimate rights above all other considerations within the professional relationship.[5,6]

This fundamental principle, a starting point and deceptively simple, points to further exploration of the physician's obligation to his patient in its many dimensions; and it also represents the physician's end of a special contract with society which, alongside the essential duty to know his professional business, defines the ethical obligation of the physician in more general terms. No doubt other professions, with only small differences in wording, might be included in the sweep of the same definition, but for present purposes my own inherent prejudice and experience lead me to include only two others: the legal profession and the profession of moral philosophy, most of whose members are university-based. It is not too much to say, with some fear and trembling, that the long-term future of the best that is in western society rests critically on the shoulders of physicians, lawyers, and academics.

The burgeoning interest in ethics—especially medical—that appeared in the decades following the end of World War II is a fascinating phenomenon in its own right, the genesis of which no one seems to comprehend fully. Suffice it to say that the prewar writings of Richard Cabot[7] and Francis Weld Peabody[8] and the immediate postwar writings of Dean Willard Sperry[9] and Joseph Fletcher[10] were sources that gave rise to more recent influences and emphases.

All, however, has not been sweetness and light in recent years. The meeting at the bedside of physician and philosopher is often confused and sometimes less than cordial.[11,12] There have also been times when recommendations of the experts concerning ways to improve the physician-patient relationship have seemed to be something like kicking a moribund, if not quite brain-dead, horse.[13,14] All this notwithstanding, the need for meaningful exchange between physician and moral philosopher is no longer seriously debatable, owing in no small measure to the beneficent influence of the Hastings Center, founded in 1969 and continuously influential in moving difficult ethical and legal problems encountered by the medical profession to *modus vivendi*, if not to final solution. Whatever may be the future of bedside consultation between physician and moral philosopher, the center's activities have brought home the need for exposure of the physician to ethical principle and argument beginning early in the convoluted educational process that leads to the MD degree.[15]

Life-and-death ethical problems have been making the headlines for a long time. The Quinlan case, various aspects of euthanasia, the right to die with dignity, and most recently problems relating to the AIDS epidemic, have received almost daily coverage by the media which, quite correctly, treat them as matters of general public interest and concern. This they certainly are. But there are two problems, both of major ethical and legal implications, that seem to me to warrant much greater emphasis than they have received. Both contain very ominous possibilities, and each is far too large for effective analysis by any one profession acting alone. There is the possibility, however, that the three professions I defined earlier—law, medicine, and philosophy—acting in concert and rising above the strictures of narrow self-interest, may be able to initiate the vital process of protecting society from dangers that loom larger and larger on our turbulent horizon.

PROCUREMENT OF ORGANS FOR TRANSPLANTATION

The most spectacular issue, and the most fascinating to the media, is the procurement of human organs, paired

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and unpaired, for transplantation.[16–18] Two initial caveats are in order. First, my argument is not that of the Luddite; there can be no question that the technology of transplantation of organs will approach perfection, probably within the relatively near future. Second, what are the full implications of the fact that, for the first time in the history of our species, it is possible to prolong the life of one human being by terminating that of another—in a sense murdering him—or by irreversibly diminishing him in a physical sense? The problem is compounded, parenthetically, by the strong probability that, as the technique of transplantation approaches perfection, the demand for transplantable organs will continue indefinitely to exceed supply by a wide margin. Therein lie the seeds of social corruption and degradation.

A few weeks after Christian Barnard performed the first cardiac transplant in December 1967, there were disturbing speculations on the supply of living hearts for transplantation. Early in 1968, I myself wrote and delivered a sermon on the topic to which I gave the title "Hypocrisy and Confusion: New Organs for Old." [19] The thrust of this presentation was that politically and economically powerful groups would one day be able, by the adroit use of television and control of the legislative process, to designate certain underprivileged but physically healthy groups as organ donors. The latter would be forced to contribute an organ or organs whenever anyone with the right political connections needed replacement of heart, liver, or kidney. The piece never found a publisher, the standard comment being that such events cannot happen here. But about a year later, an American court, invoking the curious "substituted judgment" doctrine, required a mentally incompetent ward of the state, himself unable to make any sort of choice, to contribute a kidney to a sibling with near-terminal renal failure.[20]

Very recently, with the return of capital punishment in many states, it is maintained by some that condemned murderers should be required to repay society for the damage they have wrought by contributing a vital organ or organs to worthy recipients. From that argument, it is easy to move on to execution by injection,[21] and from there to the transformation of the execution chamber into a special operating room so that organs can be harvested more effectively and conveniently. The final hypothetical step is to persuade judges, as they confer the death sentence, to allow flexible dates of execution in order to coordinate supply with requirement anywhere in the country.

Such things have not happened here. Not yet. But reasoning of this sort is aptly categorized as a "slippery slope" or "entering wedge" argument,[22] and brings to mind the rhetorical question that has been raised a number of times since Barnard's exploit: Had transplantation been feasible in the late 1930s and early 1940s, where would the Third Reich have turned for organ donors for any member of the Master Race that might have needed them?

In our own time, a recent federal statute (PL 98-507)[23] notwithstanding, rich Americans and foreigners are able to purchase organs for transplantation ahead of impecunious patients with more pressing need. Organ merchants, like arms merchants and drug barons, are likely to go right on pursuing their cynical but immensely profitable activities, the law and social damage notwithstanding.[24–29] Obviously, organ transplantation, paired or unpaired, cannot itself be prohibited, nor should research in the field be hamstrung. But we have yet to render the practice ethically positive, or even neutral, in all its aspects. And in these several connections we need to begin to think the unthinkable, something at which moral philosophers are commendably adept.

COMPENSATION OF THE ACCIDENTALLY INJURED

In terms of potential for damage to American society another problem looms as large as or larger than, as urgent as but less dramatic than, procurement of organs for transplantation: that is the matter of litigation for accidental personal injury, to which the generic term tort law, or torts, is customarily applied. The system, which includes professional malpractice law, is failing[30–33] and the damage it is causing to the body social and the body politic is immense. There are many technical reasons for its failure, but probably the most important is the identification of the system with the sweepstakes-megabuck psychopathology.

It is customary for physicians to blame trial lawyers for this grave distortion of a legal institution, the primary purpose of which was once admirable. But the hope of hitting the jackpot at Atlantic City, or by winning a suit for damages, is a characteristic of late 20th century life in these United States. No one is really immune. Physicians are targets for lawsuits to an unreasonable degree, but if they themselves are injured in an accident of any kind they may plunge into the tort lottery as vehemently as any nonphysician, contingency fee contract and all. That so-called average citizen, confronted with what he thinks is the prospect of instant wealth, is not likely to give serious thought to abstract questions concerning right and wrong or damage to social institutions. The ultimate irony is that the most astute diagnosis of the basic disorder comes from a contemporary comic strip: "We have met the enemy, and he is us." [34]

The malpractice problem is bad enough, but it is not, economically speaking, as deleterious as product liability. Even more damaging in a social sense is the inhibiting effect that the threat of personal injury litigation and the heavy burden of rising insurance premiums exert on the programs of some of our most indispensable nonprofit organizations, including schools, colleges, municipalities, and even churches. No less an authority than the chairman of Lloyd's of London, speaking recently in San Francisco, warned that ". . .what is happening is so serious that it is literally dislocating the economy [and other critical functions]." [35]

There have been many efforts to modify major defects in tort law but, to date, they have been remarkably ineffective. The medical profession has thought at times that legislative patchwork would do the trick, but, a recent optimistic appraisal in *The New York Times* notwithstanding,[36] there is not yet even a pinpoint of light at the end of the tunnel's blackness.

The reason for failure to date is, in my own estimate, that the most hopeful approach to tort reform has not yet been mounted. Legislative approaches have failed mainly because of counterpressures from vested interests; and "the institutional limitations of courts" make it unlikely that they can ever initiate comprehensive tort reform. Nor, for good reason, can administrative agencies. What, then, is left?

The answer is in the creation by a sponsor of unimpeachable authority of what I choose to call the *Square One Commission*, to be composed of members who are capable of viewing tort law in its widest dimensions and who can rise above special interests in the public interest. The problem being far grander than any one profession, membership must be drawn from law, medicine, the ranks of the moral philosophers, historians, and any other calling that may show promise of looking at an old problem in new and fresh ways.

The charge to the group must be, first, to design fair, ethical, and effective means to compensate the injured party. Second, the commission must go back to Square One, setting aside every tradition, every legal precedent, every vested interest claim and counterclaim, as it begins to look at tort law as a whole. In this endeavor, the views of moral philosophers are especially significant; for example, Stephen Toulmin's views on the relations between ethical theory and practical ethics, as well as his forceful insistence on the "tyranny of principle;"[37] also Samuel Gorowitz' startling concept of compensable and noncompensable negligence, and his hypothesis that ". . .the absence of actual. . .injury is simply not material to the claim that malpractice has occurred."[38,39] Such views, examined in depth, are matters of considerable substance and are not to be dismissed merely because they are external to our customary patterns of thinking.

The notion of a Square One Commission is, of course, wholly and quite intentionally impractical, for the good reason that it is practical folk who have brought us to the present impasse; people whose feet are so firmly planted on the ground that they are totally incapable of going back to Square One under any circumstances. We ought to know by now that approaches based precisely on the existing system in all its inconsistencies and decay can only give us more of the same.

This new approach, if it ever gets underway, will predictably tax our colleagues in moral philosophy far more than the usual physician-philosopher bedside exchange. As for the medical profession, it has, by sponsoring a series of halfway measures, succeeded in accomplishing little more than to expose its members to accusations that they are concerned only with their own professional wellbeing.

As for the Medical Society of the State of New York, with its splendid tradition of "clamorousness and giving mouth," the hope is that it will join with other professional groups in placing itself on the side of the angels in order to initiate general tort reform—fair and ethical—in the public interest, and in renewed recognition of its very special ethical imperative and contract with society.

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Public Health Service guidelines for counseling and antibody testing to prevent HIV infection and AIDS[*]

These guidelines are the outgrowth of the 1986 recommendations published in the *MMWR*;[1] the report on the February 24–25, 1987, Conference on Counseling and Testing;[2] and a series of meetings with representatives from the Association of State and Territorial Health Officials, the Association of State and Territorial Public Health Laboratory Directors, the Council of State and Territorial Epidemiologists, the National Association of County Health Officials, the United States Conference of Local Health Officers, and the National Association of State Alcohol and Drug Abuse Directors.

Human immunodeficiency virus (HIV), the causative agent of acquired immunodeficiency syndrome (AIDS) and related clinical manifestations, has been shown to be spread by sexual contact; by parenteral exposure to blood (most often through intravenous [IV] drug abuse) and, rarely, by other exposures to blood; and from an infected woman to her fetus or infant.

Persons exposed to HIV usually develop detectable levels of antibody against the virus within 6–12 weeks of infection. The presence of antibody indicates current infection, though many infected persons may have minimal or no clinical evidence of disease for years. Counseling and testing persons who are infected or at risk for acquiring HIV infection is an important component of prevention strategy.[1] Most of the estimated 1.0 to 1.5 million infected persons in the United States are unaware that they are infected with HIV. The primary public health purposes of counseling and testing are to help uninfected individuals initiate and sustain behavioral changes that reduce their risk of becoming infected and to assist infected individuals in avoiding infecting others.

Along with the potential personal, medical, and public health benefits of testing for HIV antibody, public health agencies must be concerned about actions that will discourage the use of counseling and testing facilities, most notably the unauthorized disclosure of personal information and the possibility of inappropriate discrimination.

Priorities for public health counseling and testing should be based upon providing ready access to persons who are most likely to be infected or who practice high-risk behaviors, thereby helping to reduce further spread of infection. There are other considerations for determining testing priorities, including the likely effectiveness of preventing the spread of infection among persons who would not otherwise realize that they are at risk. Knowledge of the prevalence of HIV infection in different populations is useful in determining the most efficient and effective locations providing such services. For example, programs that offer counseling and testing to homosexual men, IV-drug abusers, persons with hemophilia, sexual and/or needle-sharing partners of these persons, and patients of sexually transmitted disease clinics may be most effective since persons in these groups are at high risk for infection. After counseling and testing are effectively implemented in settings of high and moderate prevalence, consideration should be given to establishing programs in settings of lower prevalence.

INTERPRETATION OF HIV-ANTIBODY TEST RESULTS

A test for HIV antibody is considered positive when a sequence of tests, starting with a repeatedly reactive enzyme immunoassay (EIA) and including an additional, more specific assay, such as a Western blot, are consistently reactive.

The *sensitivity* of the currently licensed EIA tests is 99% or greater when performed under optimal laboratory conditions. Given this performance, the probability of a false-negative test result is remote, except during the first weeks after infection, before antibody is detectable.

The *specificity* of the currently licensed EIA tests is approximately 99% when repeatedly reactive tests are considered. Repeat testing of specimens initially reactive by EIA is required to reduce the likelihood of false-positive test results due to laboratory error. To further increase the specificity of the testing process, laboratories must use a supplemental test—most often the Western blot test—to validate repeatedly reactive EIA results. The sensitivity of the licensed Western blot test is comparable to that of the EIA, and it is highly specific when strict criteria are used for interpretation. Under ideal circumstances, the probability that a testing sequence will be falsely positive in a population with a low rate of infection ranges from less than 1 in 100,000 (Minnesota Department of Health, unpublished data) to an estimated 5 in 100,000.[3,4] Laboratories using different Western blot reagents or other tests or using less stringent interpretive criteria may experience higher rates of false-positive results.

Laboratories should carefully guard against human errors, which are likely to be the most common source of false-positive test results. All laboratories should anticipate the need for assuring quality performance of tests for HIV antibody by training personnel, establishing quality controls, and participating in performance evaluation systems. Health department laboratories should facilitate the quality assurance of the performance of laboratories in their jurisdiction.

GUIDELINES FOR COUNSELING AND TESTING FOR HIV ANTIBODY

These guidelines are based on public health considerations for HIV testing, including the principles of counseling before and after testing, confidentiality of personal information, and the understanding that a person may decline to be tested without being denied health care or other services, except where testing is required by law.[5] Counseling before testing may not be practical when screening for HIV antibody is required. This is true for donors of blood, organs, and tissue; prisoners; and immigrants for whom testing is a Federal requirement as well as for persons admitted to state correctional institutions in states that require testing. When there is no counseling before testing, persons should be informed that testing for HIV antibody will be performed, that individual results will be kept confidential to the extent permitted by law, and that appropriate counseling will be offered. Individual counseling of those who are either HIV-antibody positive or at continuing risk for HIV infection is critical for reducing further transmission and for ensuring timely medical care.

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[*] These guidelines were developed by the United States Public Health Service Centers for Disease Control and are reprinted from *Morbidity and Mortality Weekly Report* 1987; 36:509–514. The Council of the Medical Society of the State of New York (MSSNY) has adopted these guidelines as MSSNY policy on the recommendation of the society's Committee for Preventive Medicine.

Specific recommendations follow:

1. *Persons who may have sexually transmitted disease.* All persons seeking treatment for a sexually transmitted disease, in all health-care settings including the offices of private physicians, should be routinely* counseled and tested for HIV antibody.
2. *IV-drug abusers.* All persons seeking treatment for IV-drug abuse or having a history of IV-drug abuse should be routinely counseled and tested for HIV antibody. Medical professionals in all health-care settings, including prison clinics, should seek a history of IV-drug abuse from patients and should be aware of its implications for HIV infection. In addition, state and local health policy makers should address the following issues:
   - Treatment programs for IV-drug abusers should be sufficiently available to allow persons seeking assistance to enter promptly and be encouraged to alter the behavior that places them and others at risk for HIV infection.
   - Outreach programs for IV-drug abusers should be undertaken to increase their knowledge of AIDS and of ways to prevent HIV infection, to encourage them to obtain counseling and testing for HIV antibody, and to persuade them to be treated for substance abuse.
3. *Persons who consider themselves at risk.* All persons who consider themselves at risk for HIV infection should be counseled and offered testing for HIV antibody.
4. *Women of childbearing age.* All women of childbearing age with identifiable risks for HIV infection should be routinely counseled and tested for HIV antibody, regardless of the health-care setting. Each encounter between a health-care provider and a woman at risk and/or her sexual partners is an opportunity to reach them with information and education about AIDS and prevention of HIV infection. Women are at risk for HIV infection if they:
   - Have used IV drugs.
   - Have engaged in prostitution.
   - Have had sexual partners who are infected or are at risk for infection because they are bisexual or are IV-drug abusers or hemophiliacs.
   - Are living in communities or were born in countries where there is a known or suspected high prevalence of infection among women.
   - Received a transfusion before blood was being screened for HIV antibody but after HIV infection occurred in the United States (e.g., between 1978 and 1985).

   Educating and testing these women before they become pregnant allows them to avoid pregnancy and subsequent intrauterine perinatal infection of their infants (30%–50% of the infants born to HIV-infected women will also be infected).

   All pregnant women at risk for HIV infection should be routinely counseled and tested for HIV antibody. Identifying pregnant women with HIV infection as early in pregnancy as possible is important for ensuring appropriate medical care for these women; for planning medical care for their infants; and for providing counseling on family planning, future pregnancies, and the risk of sexual transmission of HIV to others.

   All women who seek family planning services and who are at risk for HIV infection should be routinely counseled about AIDS and HIV infection and tested for HIV antibody. Decisions about the need for counseling and testing programs in a community should be based on the best available estimates of the prevalence of HIV infection and the demographic variables of infection.
5. *Persons planning marriage.* All persons considering marriage should be given information about AIDS, HIV infection, and the availability of counseling and testing for HIV antibody. Decisions about instituting routine or mandatory premarital testing for HIV antibody should take into account the prevalence of HIV infection in the area and/or population group as well as other factors and should be based upon the likely cost-effectiveness of such testing in preventing further spread of infection. Premarital testing in an area with a prevalence of HIV infection as low as 0.1% may be justified if reaching an infected person through testing can prevent subsequent transmission to the spouse or prevent pregnancy in a woman who is infected.
6. *Persons undergoing medical evaluation or treatment.* Testing for HIV antibody is a useful diagnostic tool for evaluating patients with selected clinical signs and symptoms such as generalized lymphadenopathy; unexplained dementia; chronic, unexplained fever or diarrhea; unexplained weight loss; or diseases such as tuberculosis as well as sexually transmitted diseases, generalized herpes, and chronic candidiasis.

   Since persons infected with both HIV and the tubercle bacillus are at high risk for severe clinical tuberculosis, all patients with tuberculosis should be routinely counseled and tested for HIV antibody.[6] Guidelines for managing patients with both HIV and tuberculous infection have been published.[7]

   The risk of HIV infection from transfusions of blood or blood components from 1978–1985 was greatest for persons receiving large numbers of units of blood collected from areas with high incidences of AIDS. Persons who have this increased risk should be counseled about the potential risk of HIV infection and should be offered antibody testing.[8]
7. *Persons admitted to hospitals.* Hospitals, in conjunction with state and local health departments, should periodically determine the prevalence of HIV infections in the age groups at highest risk for infection. Consideration should be given to routine testing in those age groups deemed to have a high prevalence of HIV infection.
8. *Persons in correctional systems.* Correctional systems should study the best means of implementing programs for counseling inmates about HIV infection and for testing them for such infection at admission and discharge from the system. In particular, they should examine the usefulness of these programs in preventing further transmission of HIV infection and the impact of the testing programs on both the inmates and the correctional system.[9] Federal prisons have been instructed to test all prisoners when they enter and leave the prison system.
9. *Prostitutes.* Male and female prostitutes should be counseled and tested and made aware of the risks of HIV infection to themselves and others. Particularly prostitutes who are HIV-antibody positive should be instructed to discontinue the practice of prostitution. Local or state jurisdictions should adopt procedures to assure that these instructions are followed.

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* "Routine counseling and testing" is defined as a policy to provide these services to all clients after informing them that testing will be done. Except where testing is required by law, individuals have the right to decline to be tested without being denied health care or other services.

PARTNER NOTIFICATION/CONTACT TRACING

Sexual partners and those who share needles with HIV-infected persons are at risk for HIV infection and should be routinely counseled and tested for HIV antibody. Persons who are HIV-antibody positive should be instructed in how to notify their partners and to refer them for counseling and testing. If they are unwilling to notify their partners or if it cannot be assured that their partners will seek counseling, physicians or health department personnel should use confidential procedures to assure that the partners are notified.

CONFIDENTIALITY AND ANTIDISCRIMINATION CONSIDERATIONS

The ability of health departments, hospitals, and other health-care providers and institutions to assure confidentiality of patient information and the public's confidence in that ability are crucial to efforts to increase the number of persons being counseled and tested for HIV infection. Moreover, to assure broad participation in the counseling and testing programs, it is of equal or greater importance that the public perceive that persons found to be positive will not be subject to inappropriate discrimination.

Every reasonable effort should be made to improve confidentiality of test results. The confidentiality of related records can be improved by a careful review of actual record-keeping practices and by assessing the degree to which these records can be protected under applicable state laws. State laws should be examined and strengthened when found necessary. Because of the wide scope of "need-to-know" situations, because of the possibility of inappropriate disclosures, and because of established authorization procedures for releasing records, it is recognized that there is no perfect solution to confidentiality problems in all situations. Whether disclosures of HIV-testing information are deliberate, inadvertent, or simply unavoidable, public health policy needs to carefully consider ways to reduce the harmful impact of such disclosures.

Public health prevention policy to reduce the transmission of HIV infection can be furthered by an expanded program of counseling and testing for HIV antibody, but the extent to which these programs are successful depends on the level of participation. Persons are more likely to participate in counseling and testing programs if they believe that they will not experience negative consequences in areas such as employment, school admission, housing, and medical services should they test positive. There is no known medical reason to avoid an infected person in these and ordinary social situations since the cumulative evidence is strong that HIV infection is not spread through casual contact. It is essential to the success of counseling and testing programs that persons who are tested for HIV are not subjected to inappropriate discrimination.

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Massive hemorrhage and gastric rupture from an ulcer eroding the splenic artery

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Upper gastrointestinal bleeding is a common complication of gastric and duodenal ulcers. Standard medical therapy, including the use of saline lavage, H2 blocking agents, and antacids is effective in arresting hemorrhage in three quarters of these cases.[1 (p259)] When these measures fail, most patients can be stabilized while being prepared for surgery, with careful monitoring of and attention to blood and volume requirements. On occasion, a patient is bleeding so rapidly that shock cannot be reversed with volume replacement, and immediate surgery is essential for survival. The gastroduodenal artery or its branches are the most frequent sources of such bleeding, but erosion into the main branches of the celiac axis or even the heart and aorta has been reported.[2-4] We recently treated a patient in whom erosion of an ulcer into the splenic artery resulted in massive hemorrhage and rupture of the stomach.

CASE REPORT

A 53-year-old man presented at the emergency room complaining of sudden, severe abdominal pain, nausea, and vomiting for one hour. His medical and surgical histories were unremarkable. His blood pressure was 120/90 mm Hg; pulse, 120/min; temperature, 37.1°C; and respirations, 22/min. On physical examination, the abdomen was flat, silent, and rigid, with diffuse rebound tenderness and guarding. The stool was guaiac negative. Laboratory results showed a hematocrit of 43%, a white blood cell count of 12,200/mm^3, and normal electrolyte and serum amylase levels. Free subdiaphragmatic air was seen on an upright chest film; the abdominal films were unremarkable.

The patient was treated with intravenous fluids and was taken to the operating room two hours after admission. Surgery revealed a diffuse, moderately severe peritonitis, with a 1-cm perforation on the anterior surface of the mid-body of the stomach. Palpation revealed marked induration of the gastric wall extending over a 5 × 8-cm area around the perforation and reaching to the lesser curvature at its mid-portion (Fig 1). This finding was believed to represent fibrosis and edema from an underlying giant gastric ulcer. A biopsy was taken from the edge of the perforation; the perforation was covered with a tongue of omentum fixed in place with several 2-0 silk sutures placed through the defect.

After surgery, the patient was placed on a regimen of cimetidine and antacids to maintain a gastric pH of at least 5. The nasogastric tube was removed on the fifth day. A soft diet was begun on the seventh day. The biopsy was reported negative for tumor. On the afternoon of the ninth day, the patient suddenly went into shock after passing a large, bloody stool. Blood pressure on palpation was 50–60 mm Hg, with a thready pulse of 130/min. Nasogastric aspiration revealed red blood. An emergency hematocrit was 20%. Treatment with crystalloids and replacement blood produced a rise in systolic blood pressure to 90–100 mm Hg. The gastric aspirate cleared with saline lavage. The patient was transferred to the intensive care unit, where he remained stable for nearly two hours. He then suddenly vomited a large amount of fresh liquid blood and again went into shock. For the first time after surgery, he complained of severe upper abdominal pain, and examination revealed epigastric tenderness with guarding and rebound.

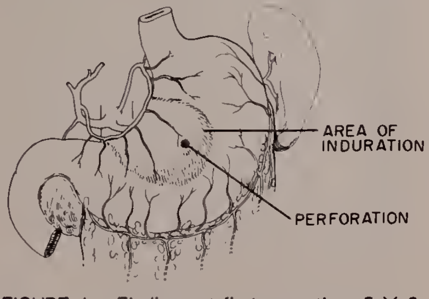

FIGURE 1. Findings at first operation: 5 × 8 cm area of edema surrounding the perforation, reaching to the lesser curvature.

It rapidly became apparent that the patient was experiencing an exsanguinating hemorrhage, which could not be managed conservatively. He was rushed to the operating room; he was conscious but had no detectable blood pressure or pulse. While the anesthesiologists continued resuscitation, the abdomen was opened through the previous incision. The aorta was rapidly isolated and clamped at the hiatus. The systolic blood pressure immediately rose to 60 mm Hg, then slowly increased to 80–90 mm Hg with transfusion. Surgery was then resumed. There was no free blood in the peritoneal cavity, but the omental bursa was distended with blood and clots. The previous plication was intact. The lesser sac was entered. Further exploration revealed that the original ulcer continued around the lesser curve onto the posterior wall for a similar distance and had penetrated deeply into the pancreas. Along the left lateral margin of the ulcer there was a 6-cm opening where the gastric wall had separated from the pancreas, creating a wide communication with the lesser sac (Fig 2). Through this defect, a continuous ooze was seen emanating from a large vessel in the base of the ulcer near its left margin. This was manually controlled while the aorta was momentarily unclamped. A large, tortuous vessel, which immediately distended to the right and left of the bleeding point, was identified as the splenic artery.

The aorta was reclamped until the hole in the splenic artery had been securely oversewn, and the vessel ligated proximally and distally. A subtotal gastrectomy was then performed but was complicated by the development of a coagulopathy with diffuse bleeding. The bleeding was brought under control with the transfusion of fresh-frozen plasma, cryoprecipitate, and platelets.

The patient was transferred to the recovery room in critical condition, with a systolic

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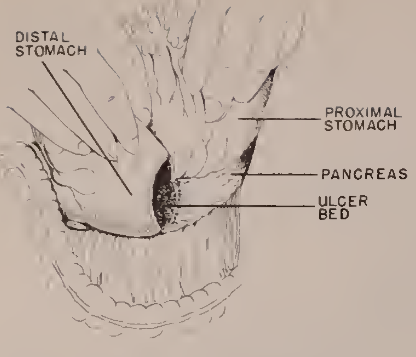

FIGURE 2. Findings on re-exploration: The lesser sac has been entered through the gastrocolic omentum, revealing a large communication with the gastric lumen along the left lateral margin of the ulcer.

TABLE I. Summary of Reported Cases

Year	Age (yr)/ Sex	Duration of Ulcer Symptoms	Ulcer Size	Duration of Bleeding	Surgery	Result
1829[6]	56/M	6 yr	"5–6 rugae"	48 hr		Died
1836[7]	64/F	3 mo	1.5 × 1 inches	5 days		Died
1837[8]	49/F	2–3 mo	2 inch diameter	24 hr		Died
1846[9]*	60/F	—	—	36 hr		Died
1848[10]	?/M	3 yr	1 inch diameter	6 wks		Died
1858[11]	18/M	"few mos"	"silver dollar"	sudden death		Died
1865[12]	76/F	3 wks	2 × 1.5 inches	30 hr		Died
1866[13]	63/M	8 wks	3.5 inch diameter	1 mo		Died
1869[14]	34/F	"many yrs"	2 cm diameter	2 days		Died
1869[15]	20/F	1 yr	1 cm diameter	19 days		Died
1874[16]	34/F	"years"	"shilling"	12 days		Died
1880[17]	32/M	7 wks	1.5 cm diameter	17 days		Died
1896[18]	57/F	2 wks	"large"	6 days		Died
1898[19]	22/F	6 hr	1 inch diameter	6 hr		Died
1915[20]	"60's"/F	"long time"	"5 mark piece"	17 days		Died
1936[21]	43/F	"wks"	—	died day of bleed		Died
1946[22]	56/F	20 yrs	"large"	24 hr	Vessel oversewn	Survived
1955[23]*	56/F	1 yr	1 × 1.5 inches	9 days	Open and close	Died
1966[2]	43/F	4 days	"huge"	4 days	75% gastrectomy, BR-1	Survived
1977[24]	39/M	2 yr	—	19 days	Total gastrectomy	Survived
1978[25]	26/F	1 yr	2 cm diameter	8 days	Total gastrectomy	Survived

* Gastric rupture also occurred.

blood pressure of 80 mm Hg and a hematocrit of 31%. He had received a total of 15 units of packed cells, eight units of fresh-frozen plasma, five units of cryoprecipitate, and five units of platelets from the beginning of treatment on the ward to the time of transfer to the recovery room. Shortly after arrival in the recovery room, he became bradycardic and hypotensive. Corrective measures were ineffectual, and refractory cardiac arrest ensued. Permission for autopsy was denied. The pathologic examination of the distal stomach revealed a large chronic gastric ulcer without malignancy.

DISCUSSION

William Brinton,[5] in an 1864 monograph on diseases of the stomach, wrote the following:

> As regards death by hemorrhage in ulcer of the stomach, its frequency appears to correspond with a proportion of one in 20–30, or 3–5 percent. . .The source of the hemorrhage is almost invariably one of the three large arteries of the stomach, or some primary branch of them. Which of the three, is dictated chiefly by the situation of the ulcer; the posterior surface being the usual seat of ulcer eroding the splenic artery. . .The splenic is the artery which suffers most frequently; its proportions (about 55 percent) being referable, not merely to its larger size, but to its closer fixation, and the greater frequency of the ulcer on the corresponding surface of the stomach. . .

Mr Brinton's statement notwithstanding, it would appear that erosion of a benign ulcer into the splenic artery is a rather uncommon event, as after a careful search of the world literature[2,6-25] and recent texts,[26,27] we could only locate 21 reported cases (Table I).

Including the patient described here, there were seven men and 15 women, ranging in age from 18 to 76 years. The preponderance of women in what is generally a male disease is curious, but of questionable significance in view of the small total number. The mean age was 46 years and did not differ between the sexes. Symptoms of ulcer disease were of varying duration before bleeding started. Once hemorrhage began, it was rapidly fatal in only three instances. In the other cases, the patients lived from one day to six weeks from the onset of bleeding until death or surgical intervention. The ulcers tended to be large, ranging from 1 cm in diameter, to approximately 6 × 15 cm, with an average diameter of 3–4 cm. All were posterior, on a line running from the lower lesser curve to the upper greater curve, overlying the course of the pancreas and splenic artery.

In the first 16 cases described, from 1829 to 1936, the diagnosis was established at autopsy. This is not surprising, since at that time adequate gastrointestinal radiologic techniques and endoscopy were not available, and surgery on patients with such massive hematemesis was not feasible. The first reported case in which a patient underwent definitive surgery was in 1946.[22] Although this 56-year-old woman also arrived in the operating room near death, she survived after gastrotomy with suture of the ulcer and splenic artery. An elective, subtotal gastrectomy was performed uneventfully three months later. Among the five patients reported since 1946 (including the one described here), there have been three survivors. In this patient, at the time of the original procedure, gastric resection was recognized as the procedure of choice,[27] but this was deferred for two reasons. It was clear from examination of the anterior wall of the stomach that a fairly high subtotal gastrectomy would be necessary. We thought that it would be preferable to do this after the peritonitis had resolved. Furthermore, although it appeared that the primary lesion was a giant benign ulcer, the possibility of a neoplastic lesion—which would dictate a total gastrectomy—could not be excluded. The late hour precluded sending a specimen for frozen section.

In 1966, Brewer and Read[2] reported on three cases of upper gastrointestinal bleeding from ulceration of the splenic artery. (Two of their patients had carcinomas that had extended directly into the splenic artery. This may be an even rarer cause of hemorrhage from erosion into the splenic artery, as our review did not pick up any other such cases.) The authors made several points that merit emphasis. They recommended proximal and distal ligation of the artery, rather than simple oversewing at the base. They also reported large-sized ulcers (all measured 8 cm in diameter, as did the one reported here, suggesting that the ulcers were long-standing in nature. They stated that all three patients had mild bleeds that preceded the massive one (the so-called "sentinel" or "herald" bleed); these, they suggested, indicated involvement of a large vessel. In fact, 19 of the 21 patients reported in the literature had "herald bleeds" anywhere from hours to days before the episode of massive bleeding.

Spontaneous rupture of the stomach is also a very uncommon event. Most cases are associated with overindulgence in food and drink, sudden gaseous distention from nasal oxygen administration, or bicarbonate ingestion.[1 (pp281-285)] Gastric rupture caused by intraluminal hemorrhage has been described only three times before this report. The most recent case involved a patient with bleeding varices.[28] He had undergone esophagogastroscopy, placement of a Sengstaken-Blakemore tube, and attempted sclerotherapy only a short time before being taken to the operating room for uncontrolled bleeding. The greater curvature tear that was incidentally discovered at laparotomy might easily have been iatrogenic in origin. In the other two cases, one rupture occurred at the site of a scar on the upper fundus (possibly from an old, healed ulcer) and, in the second, there was a split in the anterior gastric wall.[9,23] In the patient described here, the stomach wall separated from the edge of the ulcer penetrating the pancreas. In these latter three cases, the source of hemorrhage was an ulcer eroding the splenic artery. We hypothesize that massive bleeding from a vessel of this size caused rapid distention of the stomach. This, coupled with sudden, violent muscular contraction such as that caused by vomiting, may produce excessive gastric wall tension leading to rupture. The site of rupture would then most likely be in an area of relative weakness (as in two of the cases), or somewhere near the plane of maximal diameter of the distended organ, where the wall tension would be the greatest.

Supraceliac aortic occlusion was used in this patient to rapidly control the hemorrhage and to allow time for adequate volume replacement and a systematic search for the source of bleeding in an almost dry operative field. In the technique employed,[29] the left index finger is used to bluntly dissect through the upper gastrohepatic omentum and underlying posterior peritoneum and to enter the peri-adventitial plane around the aorta at the level of the angle of His. With a finger on either side of the supraceliac aorta, an aneurysm clamp is applied. Although the same result can be accomplished with control of the thoracic aorta via left thoracotomy, the potential for increased morbidity from the added chest incision is an obvious disadvantage.

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Gastric carcinoid presenting with massive upper gastrointestinal bleeding

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Carcinoid tumors of the stomach represent 0.3% of all gastric tumors and 2–3% of all carcinoids.[1,2] Fewer than 200 cases of gastric carcinoid have been reported in the literature.[2] About 40% present as an incidental finding[3] and, in fact, the first two gastric carcinoids were recognized as incidental autopsy findings by Askanazy in 1923. Symptoms in other patients are nonspecific, the most common being epigastric pain not responding to antacids, followed by nausea, vomiting, and weight loss.[4] Gastric carcinoids ulcerate more frequently than those elsewhere in the gastrointestinal tract and lead to a varying degree of blood loss. Rarely they may present with massive gastrointestinal hemorrhage that requires emergency surgery. The following is a report of such a case.

CASE REPORT

A 43-year-old woman was admitted to the Interfaith Medical Center in May 1985 with hematemesis, melena, and fainting spells. On examination, she was anemic, anicteric, tachycardic, and hypotensive. Laboratory investigations revealed a hemoglobin of 4 g/dL and a hematocrit of 13%. Emergency endoscopy showed a bleeding gastric ulcer in the body of the stomach. Laparotomy, performed for uncontrolled bleeding, showed an indurated area over the posterior stomach wall. No gross evidence of malignancy was found. The regional lymph nodes were not enlarged, and the liver was free of any lesions. A partial gastrectomy with a Billroth II reconstruction was performed. The patient made an uneventful recovery.

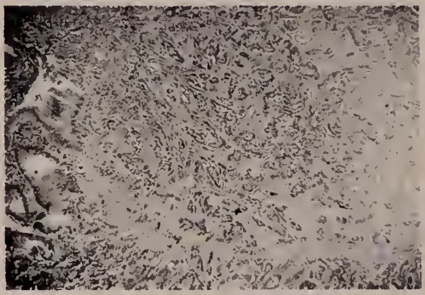

FIGURE 1. Carcinoid tumor within the gastric mucosa with prominent reactive fibrous stroma. Overlying surface mucosa is partly intact (hematoxylin-eosin stain; original magnification × 50).

The histologic examination showed a 1.5-cm ulcer with an indurated base in the posterior wall of the stomach. The tumor cells appeared to arise in the lower mucosal layers, partly ulcerating the surface mucosa and infiltrating the submucosa. The cells were small, uniform, with scanty cytoplasm. They were arranged in well-circumscribed nests with a predominant glandular pattern and associated reactive fibrosis. No mitoses were seen (Figs 1, 2). The overall picture was that of a primary gastric carcinoid.

DISCUSSION

Gastrointestinal carcinoids develop from Kulchitsky cells located in the crypts of Lieberkühn. These cells originate from neural crest. The term "karzinoide" was coined by Oberndorfer in 1907 to denote the tumor's benign nature.[5] However, as many as 25% metastasize, especially if the primary diameter is more than 2 cm.[6] The carcinoids may be singular or multiple. Their association with pernicious anemia and chronic atrophic gastritis is well established.[2] They are usually located in the antrum, but occur in the lesser curvature, body, cardia, and fundus, in that order.[7]

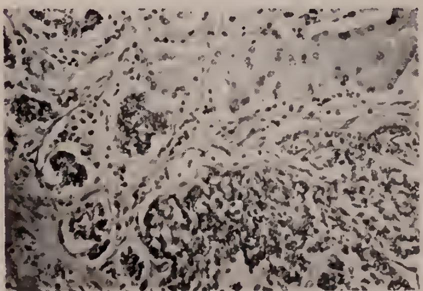

FIGURE 2. Typical uniform small cells exhibiting a mixed glandular and insular pattern (hematoxylin-eosin stain; original magnification × 200).

Gastric carcinoids secrete 5-hydroxytryptophan and not 5-hydroxytryptamine, because they lack the specific L-amino acid decarboxylase. Carcinoid syndrome is seen in only 9% of cases.[8] The clinical presentation and radiographic findings are usually nonspecific, and the endoscopic biopsy is positive in only 40% of cases because of the submucosal location of tumors.[9] A preoperative diagnosis is possible only if patients present with carcinoid syndrome or if the endoscopic biopsy is positive.

Although a minor degree of blood loss is common,[9] presentation with massive hemorrhage is rare.[3,10] Ten percent of patients in the series reported by Martin et al[10] presented with hematemesis, whereas three of nine patients reported by Balthazar et al[9] presented with slight to moderate gastrointestinal bleeding. Schoenfeld et al,[3] in a review of 42 patients from the literature, found significant gastrointestinal bleeding in only five patients, an incidence of 12%. The size of the tumor has no bearing on its tendency to bleed, and Honig and Weingarten[8] reported massive hemorrhage from tumors as small as 1 cm. The tumor in the case presented here was also small.

Tumors less than 1 cm in size rarely metastasize and are adequately treated by local excision. Those larger than 2 cm have a greater tendency to metastasize and require radical subtotal gastrectomy. Total gastrectomy is recommended in those cases with multiple carcinoids.[11] The prognosis is good if the disease is localized, with a 93% five-

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year survival rate. This rate, however, drops to 23% if metastases are present.[1]

Acknowledgment. The authors thank Elenita Estuita, MD, Department of Pathology, for her invaluable assistance.

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Neonatal craniopharyngioma

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Craniopharyngiomas are believed to be benign tumors that originate from embryonic cell rests of an incompletely involuted hypophyseal-pharyngeal duct,[1,2] although this hypothesis remains controversial.[3,4] They are the most common pediatric intracranial tumor of nonglial origin.[5] Although most craniopharyngiomas are diagnosed in children and adolescents,[6,7] they have been diagnosed during the neonatal period in ten reported cases.[8-17]

This report describes the prenatal detection of craniopharyngioma and its subsequent clinical, ultrasonographic, roentgenographic, and neuropathologic confirmation. Previously reported cases are reviewed, and the clinical management of neonatal craniopharyngiomas is discussed.

CASE REPORT

A 2,670-gram girl of 35 weeks' gestation was born to a 21-year-old woman (GIP0). The pregnancy was complicated by the onset of premature labor, for which an obstetric ultrasound study was performed. This showed polyhydramnios and macrocephaly secondary to an intracranial, calcified lobulated mass which replaced all normal anatomical structures. A sonolucent ring of fluid was noted peripheral to the intracranial mass. No hydrocephalus was detected (Fig 1). After initiation of ritodrine hydrochloride, an elective Cesarean section was performed.

The baby's APGAR scores were four at one minute and seven at five minutes. The heart rate was 150 beats/min; respiratory rate, 60/min; blood pressure, 60/30 mm Hg. The head circumference was 43 cm; the anterior fontanelle measured 8.4 cm, and the posterior fontanelle measured 7.0 cm. A firm mass was palpable through the anterior fontanelle, and bitemporal and frontal bossing was noted. The ears appeared low-set. The chest circumference was 25.3 cm; the crown-rump measurement was 32 cm; the crown-heel measurement was 46 cm. The spinal column, back, and extremities were normal. The pupils were equal and reactive to light with spontaneous eye opening. Brainstem reflexes were brisk, and all limbs moved spontaneously. Other findings included icterus, a grade II/VI harsh systolic murmur heard loudest at the upper sternal border radiating to the back and abdomen, and an enlarged liver palpable at 2 cm below the right costal margin. The physical examination was consistent with a gestational age of 33 weeks. The baby demonstrated a poor respiratory effort and needed intubation shortly after birth.

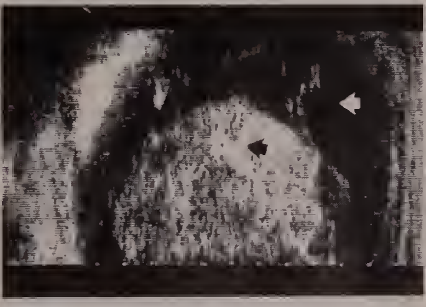

FIGURE 1. The antenatal real-time ultrasound study of the fetal head shows the presence of a lobulated echogenic mass containing calcium fragments (black arrow). There is peripheral fluid accumulation (white arrow); hydrocephalus is absent. There is a loss of normal intracranial anatomy (from Snyder et al, 1985).

Laboratory studies obtained after delivery included an arterial blood gas with pH of 7.32, P_{O_2} of 61 torr and P_{CO_2} of 30 torr. A chest roentgenogram showed changes consistent with cardiomegaly and mild respiratory distress syndrome.

An ultrasound examination of the head performed in the delivery suite confirmed previous findings (Fig 2). A cranial computed tomographic (CT) scan performed at two hours of life demonstrated a calcified intracranial mass extending to the skull base. No hydrocephalus was detected, and only a thin cortical mantle was present (Fig 3).

The baby's hospital course was complicated by severe respiratory distress, bradycar-

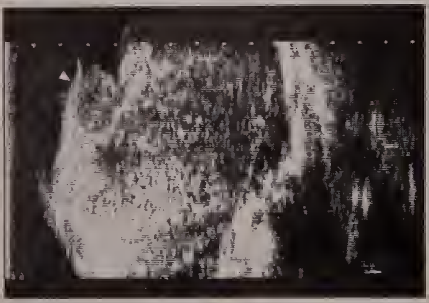

FIGURE 2. An ultrasound study of the neonatal head viewed through the anterior fontanelle was performed shortly after birth, confirming the antenatal ultrasonographic observations.

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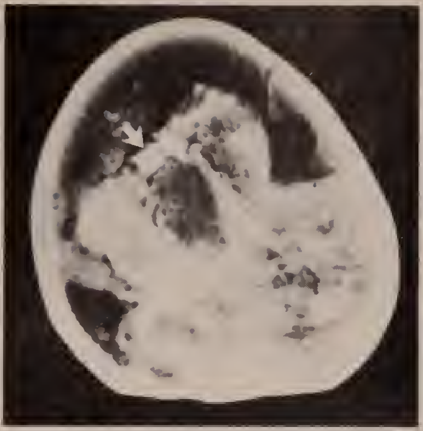

FIGURE 3. A non-contrast CT scan performed at two hours of life shows a large suprasellar mass occupying both hemispheres. Spicules of calcium are seen throughout the tumor (arrow). There is no evidence of hydrocephalus. The normal cortical mantle is markedly aberrant.

dia, and sepsis. With conservative management, she suffered cardiorespiratory arrest and died on the second day of life.

The postmortem examination was performed 18 hours after death. The general examination was remarkable for low-set ears, jaundice, and multiple hemorrhages involving the liver and lungs. The cerebral hemispheres were asymmetric, with the left being much larger than the right. Where evident, the gyral pattern was simple, and the overlying leptomeninges were thickened. In the anterior half of the right frontal lobe, a large, fluctuant area was palpable. Minimal hydrocephalus was noted. The tumor, which in situ measured 12.4 × 7.6 × 10.0 cm, had a wood-like consistency and appeared to occupy the posterior inferior portion of the cranial cavity. The optic nerves, lower brainstem, and cerebellum were grossly intact.

Coronal sections revealed the tumor to be composed of soft, rubbery, pinkish-gray tissue with multiple cysts filled with green mucoid material. In most areas, the tumor was separate from the markedly compressed and distorted brain tissue (Fig 4). The better preserved cerebellum, which consisted of portions of anterior frontal and mid-temporal lobes, showed no gray-white demarcation and a marked underdevelopment of the gyral pattern. No deep structures including basal ganglia or upper brainstem were recognizable. The spinal cord was unremarkable.

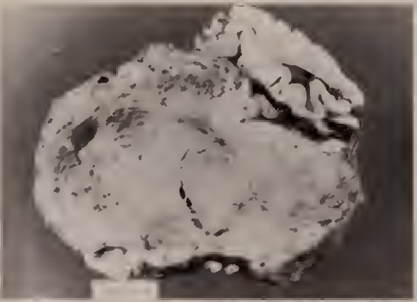

FIGURE 4. Coronal section through the cerebral hemispheres reveals a granular tumor with multiple cysts filled with green mucoid material, which is well demarcated from the markedly compressed distorted brain tissue.

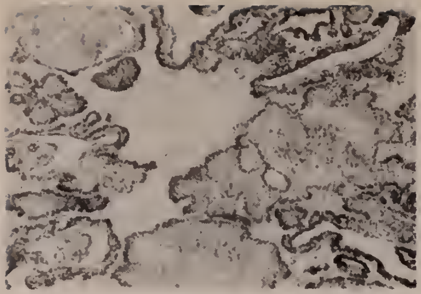

FIGURE 5. Microscopic examination of the tumor shows branching cords and palisading of epithelial cells, focal calcifications, and an inner zone of stellate cells. These findings are diagnostic of a craniopharyngioma.

Microscopic sections (Fig 5) of the tumor revealed nests of squamous cells arranged in an organized fashion within a loose mesenchymal, vascular stroma. The cells on the periphery of the nests were cuboidal and in a palisading pattern. Squamous pearls were formed focally. Cyst-like areas noted on gross examination contained proteinaceous (mucicarmine-negative) material and cellular debris. Broad zones of necrosis were present. Part of the tumor was surrounded by a dense layer of connective tissue, and then more externally by the pia-arachnoid. This appearance was diagnostic of a craniopharyngioma.

DISCUSSION

Craniopharyngiomas comprise 3% of all intracranial tumors[18] and are the most common pediatric intracranial tumor of nonglial origin.[5] In Matson's series,[6] they accounted for 9% of all intracranial tumors in children. Although craniopharyngiomas are thought to be congenital in origin,[1,2] the youngest patient in Matson's series was two years old. The diagnosis of craniopharyngioma is seldom made during the neonatal period,[7] probably due to the lack of associated symptoms.[12] Only ten cases of neonatal craniopharyngioma have been reported previously.[8-17]

This neonate is the first patient to our knowledge with a craniopharyngioma detected in utero using ultrasound. We have uncovered only four other reports of congenital intracranial neoplasms that had been diagnosed prenatally using ultrasound; three were teratomas[19-21] and one was a dysplastic mass.[22] As was noted for the patient under discussion, the large size of these tumors at birth prevented ultrasonographic identification of normal intracranial landmarks, and the histopathologic diagnosis could not be determined from the prenatal scans.

Radiopacity is frequently observed in radiographs of craniopharyngiomas;[11,23-25] in this patient, calcium fragments were noted in the antenatal ultrasound study. In utero roentgenographic calcification has been observed previously with craniopharyngiomas,[10] teratomas,[26] and meningiomas.[7,27] Therefore, demonstration of intracranial radiopacity in the neonatal period is not pathognomonic for craniopharyngioma.

Neonatal craniopharyngioma occurs with almost equal frequency among boys and girls. Of the ten cases in which gender has been described, the male to female ratio is 2:3. Of note is the fact that this is the second case of neonatal craniopharyngioma with the associated anomaly of low-set ears, supporting the hypothesis that this tumor is congenital in origin.[1,2] Other associated anomalies include two cases each of polydactyly and hepatoma with hypoplasia of the lung.[11,15,28]

The prognosis for craniopharyngioma diagnosed in the neonatal period is grave. There are no reported survivors past one year of age. The poor prognosis can be attributed to the large size of tumors and the poor general condition of patients at birth.[28]

Polyhydramnios was observed in all five cases of neonatal brain tumors diagnosed antenatally by ultrasound.[19-22] This may suggest that the finding of idiopathic polyhdramnios warrants a search for brain tumors in otherwise normal appearing and neurologically intact neonates.

Although the number of neonates undergoing any particular form of treatment has been limited, we believe that the radical excision of craniopharyngiomas in the neonatal period is not indicated. We agree with Tabaddor et al[16] and Jooma et al[29] that the surgical goals in the perinatal period should be limited to shunting hydrocephalus, cyst aspiration and biopsy, or subtotal decompression of the tumor to control intracranial pressure and prevent irreversible optic and hypothalmic dysfunction. Earlier radiologic detection of clinically silent congenital brain tumors may facilitate safe radical surgical removal of some tumors during the neonatal period.

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THE SURGICAL TREATMENT OF GASTRIC ULCER

. . . DISCUSSION [Dr H. Grad]. The surgical treatment of gastric ulcer is in the stage of transition. Called upon to treat these cases, one is apt to waver, particularly in the presence of ill-defined symptoms, between the surgical and medical treatment, with the tendency to lean towards those therapeutic measures that preclude surgical intervention. The transition stage, however, will not last long, because of the vast experience that is accumulating in gastric surgery, which will place the operative treatment of these affections on sound surgical principles. Experience has demonstrated over and over that most brilliant results can be expected from these operative measures in gastric ulcers. A vast army of gastric sufferers have been already relieved, comfort and happiness procured for them, by these not too complicated operations of gastro-enterostomy, excision and partial gastrectomy.

A gastric ulcer, even if definitely diagnosed, becomes a surgical disease when medicinal therapeutic measures fail to be curative. If this is admitted for the definitely diagnosed cases of gastric ulcers, then those cases that do not admit a definite diagnosis call most urgently for exploratory incisions. How dreadful are those calamities of gastric perforations, hurling the victim speedily into eternity! How awful to witness a case of death from hemorrhage of the stomach! What thoughts do not crowd into one's mind when called upon to treat these fatal perforations? These calamitous perforations would be largely eliminated if exploratory incisions for all the ill-defined cases were more frequently performed, and were the cases of gastric ulcers more often operated on. . . . It is estimated by competent observers that 3 per cent. of cases suffering with gastric ulcer die of hemorrhage. This is a very low estimate. Fenwick reports that out of a series of 112 fatal cases 16 per cent. died of hemorrhage. In another series of 298 fatal cases of gastric ulcer 9 per cent. of deaths were due to hemorrhage. Welch estimates that 3 to 5 per cent. of deaths in gastric ulcer are the result of hemorrhage. It has also been estimated that 6½ per cent. of gastric ulcers undergo perforation which result in a large percentage of deaths. Mortalities so heavy in any disease deserve consideration. We hardly appreciate the very serious condition of gastric ulcer in the present unstable condition of our knowledge of the affection. The symptoms in the vast number of these cases are so vague that it is refrained from advising operation. It will not be long before such advice will be given more often when symptoms do not disappear under medical treatment, and more frequent operations will be of much benefit to the patients. . . .

(*NY State J Med* 1905; 5:249-251)

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Cutaneous polyarteritis nodosa

TO THE EDITOR: We read the case report[1] and accompanying commentary[2] regarding cutaneous or limited polyarteritis nodosa with interest and would like to make some additional comments. It is often difficult to define specific subsets of diseases of which we know little regarding the etiology, pathogenesis, and optimal forms of therapy.[3] This is particularly applicable to the spectrum of vasculitides, which have been somewhat arbitrarily divided into separate entities based on clinical syndromes, histopathology, and the size of vessel involvement.

We believe caution is necessary before defining and establishing a diagnosis of (isolated) cutaneous polyarteritis nodosa, for the following reasons.

The existence of cutaneous polyarteritis as a distinct entity has not been rigorously proven. In the landmark paper by Diaz-Perez and Winklemann,[4] few efforts were made to exclude a diagnosis of systemic vasculitis by angiography despite the occurrence of myalgias, arthralgias, fevers, and neuropathy in their patients. Similarly, this diagnosis was excluded from the evaluation by Meredith et al.[1]

Aside from the case described by Dr Ball in his commentary, little has been written on the long-term follow-up of patients with cutaneous polyarteritis. It is therefore premature to label this entity a benign disease. In the report by Diaz-Perez and Winkelmann,[4] 12 out of 23 patients were "being treated with drugs and have variable intensity of their symptoms" after an unspecified period of observation.

An additional subset of polyarteritis nodosa with primarily cutaneous and musculoskeletal involvement has been termed "microscopic" polyarteritis. The clinical features of this entity and its prognosis were recently reviewed in a series of 34 patients.[5] Renal, hepatic, and superior mesenteric angiography was performed in 12 of these patients to exclude visceral disease. In spite of negative angiographic studies, renal disease was a common feature of these patients (focal segmental glomerulonephritis). Furthermore, the survival rate at five years was only 65%, and the majority of the long-term survivors had significant renal impairment, many requiring maintenance dialysis. Despite aggressive treatment with corticosteroids and immunosuppressive agents, these patients still had a poor outcome.

In summary, not all patients with what appears to be "limited" polyarteritis nodosa will have a benign course. Caution must be exercised until more data on such entities as cutaneous or microscopic polyarteritis become available.

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In reply. We appreciate the letter from Drs Kaufman and Gruber regarding our case report of a patient with polyarteritis nodosa who presented with an illness that resembled previously described cutaneous or limited disease.[1] We agree that caution is indicated in defining this particular subset, which we noted in our paper and which was noted again by Dr Ball[2] in the accompanying commentary. This debate over a separate entity of limited polyarteritis has existed since Lindberg[3] described the cutaneous condition in 1931. The utility of distinguishing various forms of vasculitis into subsets has become apparent over the years with regard to the course of the illness as well as the long-term prognosis. However, it is clear that not all patients whose disease is apparently limited to the integument will have a benign course.

In our paper we emphasized the fact that polyarteritis nodosa limited to the skin remains a controversial concept. Additionally, we did not rigorously exclude visceral involvement. The thrust of our report was to point out the unusual acute migratory nature of the illness, a presentation that has not previously been reported. We also noted the cutaneous findings, which were clinically suggestive of thrombophlebitis. Based on the comments we received and the commentary by Dr Ball,[2] it certainly appears that the controversy regarding cutaneous or limited polyarteritis nodosa remains unsettled. At present, the evidence appears to support the concept that cutaneous disease has a better overall prognosis and that there may be some utility in attempting to distinguish these entities.

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In reply. Drs Kaufman and Gruber raise caveats pertaining to the distinction between benign and lethal vasculitis which are reasonable and which reinforce those implicit in my commentary.[1] Attempting to distinguish between systemic and cutaneous polyarteritis is more than academic if only to avoid uncritical and unnecessary "aggressive treatment with corticosteroids and immunosuppressive agents" with its attendant hazards. Identifying a problem as cutaneous polyarteritis does not vitiate the need for close surveillance and willingness to alter the course of treatment.

With regard to the long-term evaluation of cutaneous polyarteritis, the condition has been observed to last as long as 25 years.[2] Borrie[3] described 15 patients with cutaneous polyarteritis nodosa in whom the length of follow-up varied from two to 23 years, the mean being 11.5 years. Three of these 15 patients died, one from the consequences of rheumatic heart disease, one from coronary thrombosis, and one by suicide. Six patients were symptom-free for a mean of 5.4 years. Cutaneous nodules persisted in six patients, and one was symptom-free except for peripheral nerve palsy.[3] Thus, prolonged observation of patients in whom no visceral manifestations supervened has been the basis for the identification of cutaneous polyarteritis as a distinct entity.

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Is the human immunodeficiency virus really the initiator of human immunodeficiency?

TO THE EDITOR: I believe that the human immunodeficiency virus (HIV), a lymphotropic virus which selectively infects helper T4 lymphocytes, poses the threat of acquired immunodeficiency syndrome (AIDS) only to those whose immune systems have been suppressed *prior* to infection by the virus, and that among those infected by the virus, only genetically susceptible individuals are likely to progress to the terminal stage of AIDS.

Immunosuppression has been observed in human beings after intense antigenic stimulation by chronic infection.[1] Decreased helper cell as well as increased suppressor cell concentrations have been reported in another disorder of intense immune stimulation, graft versus host disease,[1] which results in lymphadenopathy, opportunistic infections, malignancies, and increased susceptibility to C type retroviral infections.[2] Among diverse segments of the world's population subject to intense and repeated antigenic stimulation by microbial agents are heterosexual inhabitants of places with primitive sanitary facilities, users of illicit intravenous drugs, and male homosexuals, especially those with many sexual partners. The immune systems of these people may be subject to repeated stimulation by a prodigious number of antigens including bacteria, parasites, and viruses. The normal response to such stimulation would include the generation of large numbers of idiotype-specific suppressor cells. Indeed, an absolute increase in suppressor cells has been observed in a group of asymptomatic homosexual men prior to the appearance of anti-HIV antibodies.[3] The production of antigen-induced suppressor cells has been shown to generate nonspecific soluble suppressor factors[4] which should be active against noncommitted helper cells. This would explain the absolute deficiency of helper cells reported in some individuals subject to intense immune system stimulation before evidence of AIDS developed.[5]

The most effective control of viral infections requires the presence of the appropriate genetically determined major histocompatibility complex (MHC) antigens. Zinkernagel and Doherty[6] demonstrated that cytolytic T lymphocytes from animals infected by virus were directed against cell membrane targets which were combinations of specific MHC antigens and the specific viral antigens.[6] They postulated that successful containment of viral infections requires cytolytic T lymphocytes that can destroy infected cells before virus synthesis can be completed. Although antiviral antibodies can usually be evoked by infection in any immunocompetent animal, they are an inefficient means of controlling viral infections because of their limited capacity to destroy the cellular repositories of virus reproduction. Since only certain MHC antigens will link with specific viral antigens to provide the recognition required for cytolytic T lymphocyte activation, evolutionary pressures have favored hosts with the most polymorphic MHC gene system. A similar dual requirement for cytolytic T lymphocyte activation against viruses that infect man has been demonstrated and involves the highly polymorphic (70 allele) HLA-A and HLA-B components of the human MHC system.[7] It is possible that those who progress to the terminal phase of AIDS are genetically deficient in the HLA antigens which provide the recognition required for the HIV virus antigen to induce cytolytic T lymphocyte production. Analysis may reveal that certain HLA-A or HLA-B antigens are present with much greater frequency in the group that has not progressed to the terminal phase of AIDS, thus indicating that these antigens provide a protective effect. Evidence for such protection has been reported in animals and human beings. Resistance to Marek disease, a fatal disease of chickens caused by a T cell lymphotropic virus, correlates with the presence of the B21 antigen of the chicken MHC system.[8] HLA-B 15 has been identified as a protective factor against the development of chronic hepatitis in a group of hepatitis B surface antigen carriers.[9] Even more suggestive, a recent study of the distribution of HLA antigens in 24 patients in the terminal phase of AIDS compared to 34 patients with the lymphadenopathy syndrome alone reported a relative risk ratio indicating a protective effect of HLA-B 44, although the authors' summary did not interpret the study in this way.[10] Only 38 of the 70 HLA-A and HLA-B antigens were analyzed. Usually more than one HLA antigen can provide the appropriate recognition site for any virus antigen; therefore, it is possible that typing for all the HLA class I antigens in a large group of patients could identify several HLA antigens that are protective against HIV.

The T4 antigen has been identified as the cell membrane receptor for HIV, explaining the proclivity of the virus for helper cells. The T4 antigen is intimately associated with the Ia antigen[11] which is expressed on helper cells only after activation by exposure to soluble antigens.[12] It has been postulated that cell surface structures in addition to the T4 antigen alone may be required for cellular access by HIV.[10] The Ia molecule may serve this function. If so, activated helper cells would be more likely to become infected by virus than uncommitted helper cells.

The validity of these propositions can be tested by complete HLA class I analysis of the patients in a study such as that of the Multicenter AIDS Cohort,[13] which includes homosexual men who are HIV antibody negative, those who are antibody positive, and those who have progressed to the terminal phase of AIDS. Testing should also include Ia antigen analysis of helper T4 lymphocytes in such a group and in a normal control group. If the results identify HLA antigens that protect HIV antibody-positive homosexual men against progression to the terminal phase of AIDS, then this should provide reassurance both to some individuals in the high risk groups (ie, those subject to intense immune system stimulation) and to those who are not in the high risk groups. If Ia antigen analysis reveals the correlation of helper T4 lymphocyte activation with the presence of anti-HIV antibodies, this finding could also reassure those who are not members of high risk groups.

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Systemic hypersensitivity reaction following a barium enema examination

TO THE EDITOR: There are few reports in the literature concerning systemic hypersensitivity reactions to the barium sulfate mixture used for barium enema examinations of the colon and rectum.[1-4] We found only one previous example of this type of reaction occurring at Roswell Park Memorial Institute, despite the large number of barium enema examinations performed. (From 1983 to 1985, 1,851 barium enemas were performed.) Due to the rare incidence of this problem, we find interest in the reporting of this case and a review of the literature.

Case Report. A 51-year-old man with occult blood noted on a screening stool guaiac test was referred for barium enema examination. Prior proctosigmoidoscopy and rectal examination were negative. The patient had no history of gastrointestinal symptoms and no history of allergies or allergic reactions. He was taking no medications.

He received the usual saline cathartic preparation the day prior to the examination. A single contrast barium enema examination was started using a standard barium sulfate suspension (E-Z-EM, Inc). No glucagon was utilized. Shortly after the barium was introduced, the patient began to notice increased lacrimation and the sensation of retro-orbital pressure. He developed peri-orbital erythema, edema, and chemosis which was accompanied by generalized pruritus. The procedure was terminated, and 50 mg of intravenous Benadryl was immediately administered. His vital signs remained stable. He was given a tapwater cleansing enema, and his symptoms gradually resolved over the next several hours.

Discussion. USP barium is an inert substance. However, commercially available barium preparations contain a number of additives, including carboxymethylcellulose, simethicone, silica, acacia, tragacanth, polyethylene glycol, bentonite, and methylparaben.[5] These substances are included by the manufacturers in order to enhance such properties as miscibility, preservation, coating, and suspension. Manufacturers do not list the exact make-up of their barium sulfate suspensions on the product information inserts. The commercial formulation of the barium mixtures is guarded as a trade secret.[4,5] Some of these same substances are also found in a number of other pharmaceutical, cosmetic, and food products.

These additives, rather than the barium sulfate, are believed to be the most likely cause of systemic reactions. It has been postulated that access to the systemic circulation may occur during a small percentage of barium enema examinations. This can occur with normal mucosa as well as inflamed or abnormal mucosa such as in diverticulitis, ulcerative colitis, or Crohn disease of the colon.

The exact mechanism underlying the systemic reactions is not known. Schwartz et al[2] demonstrated immunologic hypersensitivity to methylparaben by performing a skin test on an individual who had experienced a systemic reaction during a barium enema examination. This evidence suggests that an IgG-mediated hypersensitivity reaction may occur in susceptible individuals previously exposed to the substance. Reexposure to that substance by systemic absorption during a barium enema examination would, therefore, elicit a hypersensitivity reaction.

The parenteral use of glucagon during barium examinations has become common practice. Glucagon has also been cited as an etiologic agent causing allergic and anaphylactic reactions. The administration of glucagon has been known to cause skin rashes, periorbital edema, erythema multiforme, and anaphylaxis. These reactions have been reported after a single injection.[3]

The incidence of allergic reactions during barium examinations is difficult to determine. Janover[4] conducted a mail survey of radiologists, major commercial suppliers of barium, the Food and Drug Administration, and the Eli Lilly Company (manufacturer of glucagon). He was able to compile 106 reactions to barium enema preparations.[4] The obvious limitations of this type of survey are small sample size, risk of overlapping reports, and voluntary reporting; however, the data gathered are of interest. Of the 106 reactions reported, 61% involved the skin (pruritus, hives, wheals, and rashes) and 8% involved the respiratory tract, 8% of the patients lost consciousness, and 21% of the reactions were not classified. Most of the reactions occurred with double contrast studies rather than single contrast. The reason for this is not clear. It is possible that more double contrast studies were performed during the reporting period, that the barium preparation used for double contrast is different, or that the differences in the procedures make systemic absorption more likely. Eli Lilly estimates the likelihood of an adverse reaction to glucagon to be one per million. Regardless of the exact figures, rare, but occasionally severe, reactions can occur during barium enema examinations.

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Increasing rate of *Salmonella enteritidis* infections associated with consumption of raw and undercooked eggs

Salmonella enteritidis infections have increased nearly fivefold in the northeastern United States since 1975, and in 1985 accounted for 3,176 confirmed infections (over 25% of all reported cases). In 1985, *S enteritidis* replaced *S typhimurium* as the single most commonly reported serotype in New York, New Jersey and New Hampshire. Symptoms of salmonellosis may include mild to severe diarrhea, fever, headache and, occasionally, vomiting. Symptoms generally appear one to three days after exposure.

This increase has been strongly, though not exclusively, associated with consumption of raw and undercooked commercial grade A shell eggs originating from farms in a number of locations. During the last two years, at least 29 outbreaks have been reported in northeastern states in association with consumption of raw or undercooked eggs. How the eggs are becoming contaminated is unknown, but is being actively investigated.

Within New York State there has also been a marked increase in the number of reported cases of salmonellosis (see Figure 1). A review of the 25 *S enteritidis* outbreaks in New York State from 1985 through August 1987 (Table 1) has shown at least 16 (64%) to have been egg associated.

Two recent New York State *S enteritidis* outbreaks occurred in health care facilities in lower New York State. Undercooked or raw egg-containing ingredients were the implicated food items. One outbreak involved 28 cases with no fatalities; the second affected over 500 patients and staff with eight related deaths. These outbreaks demonstrated the extent *S enteritidis* can lead to significant morbidity and mortality and underscored the need for close scrutiny of food service operations.

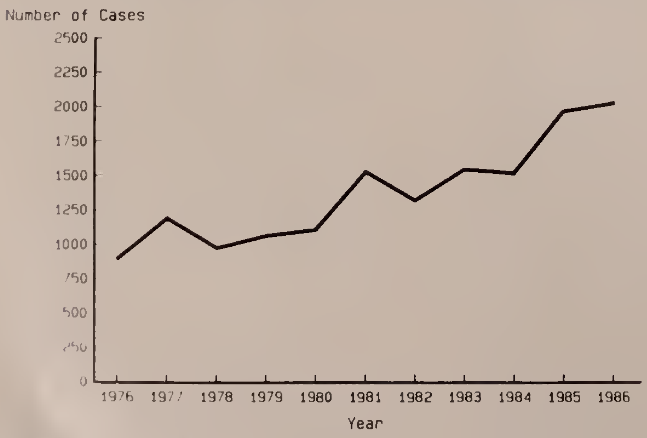

FIGURE 1. Reported cases of salmonella in New York State, 1976–1986 (Source: Bureau of Communicable Disease Control—Sporadic Case Reports).

TABLE I. *S enteritidis* Outbreaks in New York State, 1985–August 1987

Total outbreaks	25
Vehicles unknown	4
Vehicles identified	21
No known egg association	5
With egg association	16
Eggs	4
Eggs Benedict with Hollandaise Sauce	3
Pasta Product	3
Blended Food (egg in salad dressing)	1
Others with egg ingredient	5

In response to the emerging problem of egg-associated *S enteritidis* infections, the New York State Department of Health issued a press release on July 27, 1987, advising New Yorkers to exercise care in their consumption of raw eggs. State Health Commissioner Dr David Axelrod emphasized that there was no reason to stop eating eggs, but that they, like other foods of animal origin (eg, raw milk, raw meat, raw clams), should be cooked thoroughly before consumption.

Due to the increased risk for the institutionalized and elderly, a Health Series Memorandum (87-75) was also distributed to health care facilities and state institutions with the following recommendations:

1. Inform staff and patients that eggs, like other raw food of animal origin, may cause *Salmonella* infections. Raw eggs should not be considered "health food." Raw eggs in the diets of immunocompromised or other debilitated persons is to be discouraged.
2. Review menus, recipes and foodhandling practices in your facility and in food services for the homebound elderly to prevent exposure to undercooked eggs. The nursing home population appears to be at high risk for severe outcomes and deaths from *S enteritidis* infections. Blenders used for both raw eggs and pureed foods warrant special attention to prevent cross-contamination. These machines should be thoroughly washed, rinsed and sanitized after each use with raw eggs. Separate blenders for raw and ready-to-eat foods should be used. Substitution of pasteurized egg products for fresh eggs in nursing homes is recommended. Foods that have eggs as an ingredient should be cooked to an internal temperature of 165° Fahrenheit.
3. Primary egg recipes such as scrambled eggs or omelets may be safer if eggs are cracked individually for each order rather than cracked in quantity and pooled; if convenience dictates use of pooled eggs, pasteurized egg products should be substituted. Recipes containing raw eggs

(eg, Caesar salad, eggnog, hollandaise sauce, homemade ice cream, homemade mayonnaise) are to be considered high risk, and would be safer if a pasteurized egg product is substituted for raw egg in such foods. Recent egg-associated *S enteritidis* outbreaks have occurred in commercially prepared stuffed shells, gefilte fish, rice balls, French toast and Monte Cristo sandwiches. Therefore, all foods containing eggs, including those obtained from outside the facility, should be thoroughly cooked to an internal temperature of 165° Fahrenheit. This should be verified by temperature checks with probe thermometers.

4. Treat raw poultry, beef and pork as if they were always contaminated and handle accordingly.

- Fresh, uncooked meat should be handled and stored in such a way as to prevent blood from dripping upon or contaminating other foods.
- Refrigerate foods promptly upon receipt, usually at 45° Fahrenheit or less.
- Minimize holding at room temperature by preparing foods as close to service time as possible.
- Cutting boards and counters used for preparation should be washed, rinsed and sanitized immediately after use to prevent cross contamination with other foods.
- Avoid eating raw or undercooked meats.
- Ensure that the correct internal cooking temperature (at least 165° Fahrenheit) is reached, particularly when using a microwave.
- Use a probe thermometer to measure internal temperatures and assure thorough cooking.
- Encourage careful handwashing before and after food preparation, especially between handling raw and ready to eat foods.

The New York State Department of Health and Department of Agriculture and Markets are working with other agencies to investigate the problem further. Physicians diagnosing patients with salmonellosis should report those cases to the state through their local health department.

References (sections abridged from)

1. CDC: Increasing rate of *Salmonella enteritidis* infections in the northeastern United States. *MMWR* 1987; 36:10-11.

2. CDC: Update: *Salmonella enteritidis* infections in the northeastern United States. *MMWR* 1987; 36:704-705.

3. *Salmonellosis Prevention in Health Care Facilities*. New York State Department of Health—Health Facilities Series Memorandum—87-75, 9/1/87.

AIDS drug assistance program

The AIDS Drug Assistance Program (ADAP) is a federally funded assistance program administered by the New York State Health Department's AIDS Institute. The program provides azidothymidine (AZT) free of charge to medically and financially eligible individuals who are residents of New York State.

AZT, which is being marketed by Burroughs Wellcome Company under the brand name Retrovir, is an antiviral agent which inhibits the production of reverse transcriptase. Reverse transcriptase allows human immunodeficiency virus (HIV) to copy its RNA into the DNA of the host cell (primarily T4 lymphocytes) and thereby reproduce. When the infected T4 cells die, the individual's immune system becomes compromised and the symptoms associated with HIV infection may appear.

AZT was first developed in 1964 as an anti-cancer drug. The drug was not found to be useful for that purpose, but was "rediscovered" in late 1984 when researchers at the Burroughs Wellcome Company began in vitro testing of AZT's actions against HIV. Early clinical trials were begun in mid-1985 in conjunction with the National Institutes of Health.

Results were encouraging and a double blind placebo controlled study was begun in February 1986. This trial was ended prematurely when a review board determined that preliminary results indicated a significantly lower mortality rate in patients treated with AZT versus the control placebo group.

Of 144 patients treated with AZT, only one died during the course of the trial, while in the placebo group of 137 patients there were 19 deaths. In addition, patients on AZT were less likely to contract opportunistic infections, more likely to gain weight and more likely to have reversal of their skin anergy. The study clearly demonstrated that AZT was efficacious for the group of patients studied. A number of patients in the trial did experience side effects; the most significant was severe bone marrow depression often necessitating blood transfusion. In March 1987, the US Food and Drug Administration approved AZT for the treatment of certain patients with AIDS and ARC.

Individuals who use Retrovir take two capsules every four hours, every day. A one-year supply would require 44 bottles with 100 capsules each. The whosesale price of Retrovir averages $225.36 per bottle.

Participants in the program receive a card that they present to their pharmacist to obtain their prescription. It is estimated that approximately 1,100 New York residents will receive assistance through this program.

A toll free hotline, 1-800-542-2437, has been established for people seeking information about ADAP. Mail inquiries should be sent to: ADAP, PO Box 2052, Empire Station, Albany, NY 12220.

Survey of injury prevention knowledge among low-income families

One of the US surgeon general's 1990 objectives states that the proportion of parents of children under age ten who can identify appropriate measures to address the three major risks for serious injury to their children (motor vehicle injuries, burns, poisonings) should be greater than 80%.

The New York State Department of Health and the Niagara Falls Chapter of the American Red Cross conducted a mail survey of 332 low-income families who borrow child safety seats from local service organizations to assess injury prevention knowledge among low-income families. The survey was designed to establish baseline levels of injury knowledge in the low-income population which, according to recent research, seems to have the highest injury mortality rate.

The survey was administered by project coordinators of 15 child safety seat loan programs across the state. One parent per family was asked to answer ten questions about his/her knowledge of prevention of burn injury, automobile injury and poisoning.

Of 332 families surveyed, 67% did not have a poison control sticker on their phone. However, 57% did know to call, as their first action, a poison control center in a poisoning emergency. Most parents (60%) identified soaps and detergents as poisonous from a list of six toxic substances.

Additional results of the survey showed that only 40% of the low-income respondents recognized that turning down the temperature of hot water heaters can prevent scalds or that installing smoke detectors can prevent fire-related hazards. Fifty-five percent of respondents said they wear seat belts each time they ride in a car.

Surveys of injury knowledge and attitudes cannot replace well-designed observational sampling studies of risks and injuries in the development and evaluation of injury prevention programs. Similarly, any increase in knowledge does not necessarily correlate with favorable reduction in injury risk or morbidity.

Surveys can, however, help to determine baseline knowledge levels upon which to plan further research and develop intervention strategies. Results of this survey are being used to further familiarize state and local officials about the need to develop local injury prevention programs.

A description of the survey appeared in the August 1987 issue of the *American Journal of Public Health.* Reprints of the article and further information about injury prevention can be obtained from: Injury Control Program, New York State Department of Health, Corning Tower, Room 621, Empire State Plaza, Albany, NY 12237.

Epidemiologic study of choriocarcinoma

Choriocarcinoma is an epithelial tumor arising from the trophoblast, a cell layer which contributes to the formation of the placenta during pregnancy. Women ranging from the teens through age 50 are at risk of this rare form of cancer. There is little information on this disease and much of the available information is based on clinical series. Because the study populations are very small, the studies may not be representative of the general population experience. There are, however, striking international differences in the incidence rates of choriocarcinoma. Choriocarcinoma incidence rates in Asian countries are at least ten times higher than those in the US and Europe. Reliable estimates are lacking for African countries, but there is evidence of high rates in some areas of Africa.

Although there has been a poor survival rate from choriocarcinoma in the past, recent advances in chemotherapy treatments have dramatically improved survival. Race, socioeconomic status, infectious agents, blood group, and HLA (histocompatibility antigens) categories have been hypothesized to affect risk of choriocarcinoma. The etiology, however, remains largely unknown.

The Bureau of Cancer Epidemiology has conducted a study of choriocarcinoma in New York State. This project was designed to investigate the relationship between choriocarcinoma and a broad spectrum of environmental exposures. The project has been divided into two segments:

- a descriptive epidemiologic analysis of data from the New York State Cancer Registry
- an exploratory case-control study of choriocarcinoma patients and population controls.

For the descriptive study, there were 177 cases from upstate New York (New York State exclusive of New York City) diagnosed between January 1, 1950, and December 31, 1979, and 29 cases from New York City diagnosed between January 1, 1975, and December 31, 1979. Using denominators composed of all live births and fetal deaths greater than or equal to a gestational age of 20 weeks, the overall incidence rate for upstate New York for the full 30-year period was 2.46 cases per 100,000 pregnancies. The age-adjusted rate for upstate New York between January 1, 1975, and December 31, 1979, was 3.26 per 100,000 pregnancies, and for New York City, it was 2.87 per 100,000.

An examination of the age-specific incidence rates revealed that the rates remained low below the age of 45 and then increased dramatically for women over 45. No dramatic difference over time or between racial groupings were found. As in previous studies, the most common sites for metastases are the lung and the brain. A high percentage of cases are preceded by a pregnancy which resulted in hydatidiform mole, another gestational disease. Hydatidiform mole is an abnormal pregnancy resulting from a pathologic ovum characterized by multiple cystic structures.

For the exploratory case-control study, there were 45 cases of this rare cancer obtained from upstate New York between January 1, 1970, and December 31, 1983, and 101 controls. An increased risk was found to correlate with increased numbers of miscarriages immediately preceding the occurrence of the choriocarcinoma.

Elevated odds ratios (statistically significant at the 5% level) were found for unmarried women, and those employed in jobs categorized as "professional, technical and kindred." These jobs included medical and laboratory workers and teachers. The finding of an increased risk for women with occupations labeled as "medical or laboratory" indicates a need for further studies examining the possible exposures these women encountered on the job. Details on the types, durations, and times of occurrence of the various exposures are needed. Of particular interest is exposure to substances that can reach the trophoblastic epithelium during the course of the index pregnancy.

One set of exposures deserving study involves antineoplastic drugs. Recent studies of nurses and pharmacy personnel exposed to antineoplastic drugs (Selevan et al, 1985; Waksvik et al, 1981; Venitt et al; Nguyen et al, 1982) have demonstrated the presence of mutagens in their urine, increased chromosome abnormalities, and increased fetal losses. Also, these drugs have been shown to be both mutagenic and carcinogenic. More detailed analytic studies are needed to determine their etiologic significance for gestational trophoblastic diseases, particularly choriocarcinoma.

A larger study combining data from several registries and/or nonpopulation based case-control studies from trophoblastic disease centers would be useful. Examination of exposures occurring during the index pregnancy would be an important contribution toward understanding this disease.

This study was conducted in collaboration with Dr John McAna, who is currently with the New Jersey State Department of Health. A report is being prepared to submit for publication within six months. For more information on the study, contact Dr Philip C. Nasca, Director, Bureau of Cancer Epidemiology, (518) 474-7950.

References

Selevan SG, Lindbohm ML, Hornung RW, et al: A study of occupational exposure to antineoplastic drugs and fetal loss in nurses. *N Engl J Med* 1985; 313: 1173-1178.

Waksvik H, et al: Chromosome analyses of choriocarcinoma in Sweden. *Acta Obstet Gynec Scand* 1981; 49:195.

Venitt S, Crofton-Sleigh C, Hunt J, et al: Monitoring exposure of nursing and pharmacy personnel to cytotoxic drugs: Urinary mutation assays and urinary platinum as markers of absorption. *Lancet* 1984; 1:74-77.

Nguyen TV, Theiss JC, Matney TS: Exposure of pharmacy personnel to mutagenic antineoplastic drugs. *Cancer Res* 1982; 42:4792-4796.

Rope on a rocky shore in Greece (Charles R. Perakis, DO).

Rational policies to control the spread of AIDS

The risks of human immunodeficiency virus (HIV) transmission in the health care arena and rational infection control guidelines for HIV infection are the subjects of a recent commentary by J. Louise Gerberding and David K. Henderson, principal investigators of two major studies that addressed these issues (*J Infect Dis* 1987; 156:861-863).

The authors confront what they consider to be the erroneous perception that HIV is a "highly contagious occupational pathogen," fueled in part by sensational reports of occupational transmission. They cite scientific evidence to counter this belief. Drawing on information obtained from their own as well as other studies, they review the major issues that need to be considered in developing institutional infection control policies for HIV.

Prospective studies of medical and dental workers who have intensive occupational exposure to HIV have allowed investigators to evaluate potential means of viral transmission and the degree of risk. Pooled data yielded a rate of HIV transmission of less than 0.1% per year of exposure, even in the absence of standard infection control procedures. "Direct inoculation with infected blood during accidental needlestick injuries produced infection in <0.5% of subjects parenterally exposed and is the only factor associated with infection among study subjects." Therefore, the authors conclude that the risk of HIV transmission to the average health care worker, with a low rate of exposure, is considerably lower than these data suggest. Furthermore, these findings "support contentions that HIV is one of the least-transmissible nosocomial pathogens and that special infection control procedures are unlikely to have a measurable impact on transmission rates."

The authors outline the key characteristics of an infection control policy for HIV: prevents transmission of HIV; is practical to implement and enforce; is economically feasible; and is associated with a minimal risk of adverse medical, legal, or social consequences for all parties involved. When trying to fulfill these criteria and develop institutional policies, policy makers must consider the effects of two recent national trends. First, note the authors, there has been an "increased demand for HIV testing." Second, they cite an "increased commitment to universal precautions for blood and body fluids from all patients regardless of HIV test results."

There are several advantages of increased screening for HIV infection. It should reduce transmission rates by alerting health care workers of an infected patient and revealing asymptomatic and unsuspected infection in patients. Special precautions could then be instituted to reduce the risk of exposure. Prenatal screening would be useful in diagnosing infected newborns. The disadvantages of mass screening programs include the inevitable expense, the potential discrimination against HIV-infected persons, which could cost them their jobs, insurance, housing, and right to quality medical care, and ethical concerns, including confidentiality, the care of patients who refuse testing, and the effects of false-positive test results. "Currently, no evidence supports the contention that knowing a patient is infected with HIV . . . will prevent transmission or infection, even in surgical or other higher risk settings," state the authors.

The use of a single standard of infection control when treating all patients—regardless of HIV status—and when handling blood and other body fluids and laboratory specimens, should help reduce transmission of all blood-borne pathogens and would lessen the risk of discrimination against infected persons. The main problem with a policy of universal precautions is the dramatic changes that would be required in current infection control procedures and in the ways in which health care workers interact with patients. Such a policy would demand increased resources for training, enforcement, and monitoring compliance, as well as increased funds for protective clothing and devices.

The authors recommend that policy makers weigh these potential advantages and disadvantages as they tailor an infection control strategy to their individual institution. "In the final analysis," they conclude, "the greatest benefit of infection control policies for HIV may be the promotion of better infection control for all nosocomial pathogens."

AIDS vaccine approved for testing

The Food and Drug Administration has granted approval to Bristol-Myers Company (New York, NY) to begin human testing of a vaccine against the human immunodeficiency virus (HIV). Lawrence Corey, MD, director of the virology division at the University of Washington School of Medicine, and colleagues will supervise the testing of 30-60 healthy male homosexual volunteers who do not carry HIV. They will receive the experimental virus and a similar control group will receive smallpox vaccine.

Using recombinant DNA technology, researchers at Bristol-Myers introduced the genes for HIV envelope proteins into vaccinia virus—the same virus used to manufacture the smallpox vaccine. These HIV protein components are not infectious, but they should stimulate the body to make antibodies that can protect against subsequent HIV infection.

The FDA-approved study will assess the safety and efficacy of the experimental vaccine. Much research and testing lies ahead. General use of a vaccine against AIDS is not expected until well into the 1990s.

Senate healthcare appropriations total $98 billion

The $98 billion allocated by the Senate to the US Department of Health and Human Services for fiscal year 1988 includes increased funds for Medicaid and for AIDS-related programs (*Modern Healthcare*, November 6, 1987, p 42). State Medicaid grants increased by $4.4 billion, and the Public Health Service acquired $946.4 million in Senate appropriations to fight AIDS.

Additional AIDS-related funding included $24.6 million to the Food and Drug Administration and $468 mil-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

lion for AIDS research conducted by the National Institutes of Health (almost twice the $253 million allocated to NIH in fiscal year 1987).

Members of the Congress must now compromise on a health care bill that merges these Senate appropriations with those already passed by the House of Representatives.

Cost-cutting measures: A national Medicare PPO

The Department of Health and Human Services (HHS) is considering the establishment of Medicare physician franchises, similar to current preferred provider organizations (PPOs), although on a national basis. HHS officials believe that a Medicare PPO would help control overall spending by enabling strict utilization review of participating physicians' services, according to an article in the November 20, 1987, issue of *Hospitals* (pp 28–29).

If such a national physician PPO were established, Medicare carriers would create physician panels in each county. Member physicians would agree to accept predetermined fees as payment in full, excluding deductibles and coinsurance payments. Any physician could join a Medicare PPO during its first year of operation. Participating physicians would receive higher fee updates than nonparticipating physicians. Beneficiaries would have no additional payments if they use a participating physician; otherwise, they would incur coinsurance costs.

A Medicare PPO network would not have a major effect on hospital practices. Hospitals have already become accustomed to the prospective pricing system established for patients on Medicare. Hospitals would encourage staff physicians to become Medicare preferred providers and would tend to offer staff privileges to participating physicians.

The HHS proposal to establish a network of Medicare panel physicians awaits approval by the Office of Management and Budget.

Early detection important in cutaneous melanoma

Cutaneous melanoma is associated with a substantially higher mortality rate than most other forms of skin cancer. This mortality rate can be reduced, however, through minimized sun exposure throughout one's lifetime and the early detection and treatment of cancerous or precancerous lesions. Cutaneous melanoma, which is expected to have developed in almost 26,000 people in 1987 and to have caused about 6,000 deaths, is potentially curable (*JAMA* 1987; 258:3146–3154). Arthur R. Rhodes, MD, MPH, and colleagues write of the importance of screening high-risk individuals to increase the chances of early detection. The authors present the ten main risk factors for cutaneous melanoma: a persistently changing mole; increasing age; irregular, pigmented lesions; the Caucasian race; a previous cutaneous melanoma; a family history of melanoma; immunosuppression; sun sensitivity; excessive sun exposure; and a congenital mole.

Cause of Alzheimer disease still a mystery

Early in 1987, researchers believed they were closing in on the cause of the hereditary form of Alzheimer disease. At that time, evidence pointed to a possible duplication of the gene that encodes the protein amyloid beta. The protein exists in substantial amounts in the plaques that are characteristically found in the brains of patients with Alzheimer disease. However, researchers are now moving away from this theory, although they still postulate a contributing role of the amyloid gene in the development of Alzheimer disease (*Science* 1987; 238:1352–1353). The primary genetic defect responsible for Alzheimer disease remains a mystery.

The gene that causes Alzheimer disease has been localized to chromosome 21—the same chromosome responsible for Down syndrome, or trisomy 21. This finding gained support from the fact that people with Down syndrome who live into their 30s or 40s exhibit changes in their brains similar to those seen in Alzheimer disease. While Down syndrome results from an extra copy of chromosome 21, it was believed that Alzheimer disease is due to a duplication of the amyloid gene. The subsequent finding in three patients with Alzheimer disease of an extra copy of the amyloid gene further supported this theory.

Recently, however, studies of about 100 patients with Alzheimer disease revealed no duplication of the amyloid gene. Furthermore, genetic analyses of families with a history of Alzheimer disease have shown that the amyloid gene and the gene that causes Alzheimer disease are distinct entities.

Researchers have not ruled out a lesser, contributory, role for the amyloid gene in Alzheimer disease. They have discovered at least two forms of amyloid messenger RNA (the initial product of DNA, which is then translated into a protein product)—one longer than the other. The longer message appears to be made in large quantities in the brains of people with Down syndrome and Alzheimer disease. Research must now focus on a possible correlation between the different forms of amyloid mRNA and the neurologic degeneration evident in Alzheimer disease. Based on the current belief that amyloid protein resides in the cell membrane and could function as a receptor involved in cell-to-cell communication, such as occurs in memory storage and recall, the amyloid protein is likely to remain an important variable in future research on Alzheimer disease.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Casco Alston, MD, New York. Died October 22, 1987; age 76. He was a 1942 graduate of Howard University College of Medicine, Washington, DC. Dr Alston was a Diplomate of the American Board of Internal Medicine and a member of the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Fred Jerome Benjamin, MD, Brooklyn. Died September 27, 1987; age 48. He was a 1970 graduate of Howard University College of Medicine, Washington, DC. Dr Benjamin was a Diplomate of the American Board of Radiology and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Michael Warren Bergman, MD, Brewster. Died October 13, 1987; age 48. Dr Bergman was a 1964 graduate of New York Medical College, New York. He was a member of the American Fertility Society, the New York County Medical Society, and the Medical Society of the State of New York.

William John Burke, MD, Croton-on-Hudson. Died September 18, 1987; age 62. Dr Burke was a 1947 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the American Occupational Medical Association, the American Academy of Occupational Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Vincent P. Casey, MD, New Rochelle. Died October 16, 1987; age 88. Dr Casey was a 1925 graduate of Georgetown University School of Medicine, Washington, DC. He was a Fellow of the American Psychiatric Association, a Diplomate of the American Board of Pediatrics, and a member of the Academy of Medicine, the Bronx County Medical Society, and the Medical Society of the State of New York.

Anthony John DiFabio, MD, Yonkers. Died October 17, 1987; age 74. He was a 1938 graduate of Hahnemann Medical College in Philadelphia. Dr DiFabio was a Fellow of the American College of Cardiology and a member of the Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Gerson Elber, MD, Woodside. Died October 15, 1987; age 79. Dr Elber was a 1933 graduate of Faculte de Medecine de l'Universite de Nancy, Nancy, Meurthe et Moselle, France. He was a member of the American Academy of Family Practice, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Anthony John Emmi, MD, Long Island City. Died October 19, 1987; age 77. He was a 1936 graduate of Facolta de Medicina e Chirurgia dell' Universita di Siena, Siena, Italy. Dr Emmi was a Fellow of the American Academy of Family Practice and a member of the American Occupational Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Joseph George Gilbert, MD, Lighthouse Point, Florida. Died May 11, 1987; age 83. Dr Gilbert was a 1927 graduate of State University of New York Health Science Center at Brooklyn. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Braham Hirsch Golden, MD, New York. Died June 27, 1987; age 90. He was a 1921 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Golden was a Fellow of the American College of Surgeons and a member of the Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Hugo Gottlieb, MD, Forest Hills. Died October 2, 1987; age 75. Dr Gottlieb was a 1935 graduate of Deutsche Univerzita Medizinische Fakulta, Praha, Czechoslovakia. He was a member of the American Academy of Family Practice, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Bernard H. Hall, MD, New York. Died October 29, 1987; age 68. He was a 1933 graduate of the University of Kansas School of Medicine, Lawrence-Kansas City. Dr Hall was a member of the American Medical Society for Alcoholism, the New York County Medical Society, and the Medical Society of the State of New York.

Laura Cornelia Harris, MD, Minoa. Died October 16, 1987; age 93. Dr Harris was a 1924 graduate of State University of New York Health Science Center at Syracuse. She was a member of the Academy of Medicine, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Thea M. Herman, MD, New York. Died October 23, 1987; age 84. Dr Herman was a 1926 graduate of Friedrich-Wilhelms-Universitaet Medizinische Fakultaet, Berlin, Germany. Dr Herman was a member of the American Academy of Family Practice, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Constantin A. Jernakoff, MD, St Petersburg, Florida. Died April 29, 1987; age 88. Dr Jernakoff was a 1925 graduate of Medizinische Fakultaet der Friedrich Schiller Universitaet, Jena, Germany. Dr Jernakoff was a member of the New York County Medical Society and the Medical Society of the State of New York.

Irvin Klein, MD, New York. Died February 17, 1987; age 79. He was a 1933 graduate of New York Medical College, New York. Dr Klein was a Fellow of the American College of Cardiology and the American College of Chest Physicians. His memberships included the Academy of Medicine, the New York Cardiological Society, the American Academy of Compensation Medicine, Inc, the American Occupational Medical Association, the American Academy of Occupational Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Victor Kugajevsky, MD, Washington, DC. Died September 4, 1987; age 86. Dr Kugajevsky was a 1931 graduate of Lekarska Fakulta Univerzita Komenskeho, Bratislava, Czechoslovakia. He was a member of the Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Arnold Lam, MD, New York. Died October 14, 1987; age 79. He was a 1937 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. Dr Lam was a member of the American Academy of Family Practice, the New York County Medical Society, and the Medical Society of the State of New York.

Salvatore Luca, MD, Sandusky, Ohio. Died October 18, 1987; age 87. Dr Luca was a 1935 graduate of Facolta di Medicina e Chirurgia dell'Universita di Pisa, Pisa, Italy. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Henry Benedict Makover, MD, Mamaroneck. Died October 22, 1987; age 79. He was a 1933 graduate of Johns Hopkins University School of Medicine, Baltimore. Dr Makover was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Academy of Medicine, the American Public Health Association, the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Peter A. Missier, MD, New York. Died October 31, 1987; age 60. He was a 1954 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. Dr Missier was a Fellow of the American College of Surgeons and the American College of Chest Physicians, and a Diplomate of the American Board of Surgery and the American Board of Thoracic Surgery (affiliated with the American Board of Surgery). His memberships included the New York Society for Thoracic Surgery, the American Thoracic Society, the American Association for Thoracic Surgery, the New York County Medical Society, and the Medical Society of the State of New York.

Abraham Isaac Needles, MD, New York. Died October 19, 1987; age 98. Dr Needles was a 1915 graduate of State University of New York Health Science Center at Brooklyn. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

William Nuland, MD, Bronx. Died September 12, 1987; age 75. He was a 1936 graduate of Faculte de Medecine de l'Universite de Lausanne, Switzerland. Dr Nuland was a member of the Academy of Psychosomatic Medicine, the American Society of Clinical Hypnosis, the Bronx County Medical Society, and the Medical Society of the State of New York.

Burton B. Pfeffer, MD, New York. Died September 25, 1987; age 78. He was a 1935 graduate of Universite de Paris VI, Paris, France. Dr Pfeffer was a Diplomate of the American Board of Psychiatry and Neurology and was a member of the American Academy of Child Psychiatry, the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society, and the Medical Society of the State of New York.

James Anthony Pollack, MD, Johnson City. Died November 15, 1987; age 45. Dr Pollack was a 1969 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics, the Broome County Medical Society, and the Medical Society of the State of New York.

Fiorentino A. Radassao, MD, Pomona. Died September 27, 1987; age 87. Dr Radassao was a 1933 graduate of Facolta di Medicina e Chirurgia dell'Universita di Rome, Roma, Italy. He was a member of the American Psychiatric Association, the Medical Society of the County of Rockland, and the Medical Society of the State of New York.

Louis Judah Rosenfeld, MD, New York. Died October 5, 1987; age 75. He was a 1937 graduate of Universitaet Wien, Medizinische Fakultaet, Wien, Austria. Dr Rosenfeld was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology. His memberships included the Academy of Medicine, the New York State Surgical Society, the New York State Urological Society, the American Urological Association, the American Geriatrics Society, the American Association of Clinical Urology, the New York County Medical Society, and the Medical Society of the State of New York.

Michael J. Tytko, MD, Schenectady. Died October 10, 1987; age 73. Dr Tytko was a 1940 graduate of Albany Medical College of Union University, Albany. He was a Fellow of the American Academy of Family Practice and a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Mark J. Wallfield, MD, Brooklyn. Died October 27, 1987; age 85. Dr Wallfield was a 1925 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Everett H. Wesp, MD, Eggertsville. Died October 19, 1987; age 71. He was a 1939 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. Dr Wesp was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Simon Weyl, MD, New York. Died October 5, 1987; age 93. Dr Weyl was a 1920 graduate of Medische Faculteit Rijksuniversitaet te Leiden, Leiden, Netherlands. He was a member of the American Medical Society for Alcoholism, the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Frank J. Williams, MD, Altamount. Died September 24, 1987; age 97. Dr Williams was a 1913 graduate of the Albany Medical College of Union University, Albany. He was a Diplomate of the American Board of Pediatrics and a member of the Medical Society of the County of Albany and the Medical Society of the State of New York. ☐

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83: 925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

Schedule of Sessions 99
Officers, Councilors, and Trustees 101
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HOUSE OF DELEGATES

April 21 through 24, 1988
New York Hilton

SCHEDULE OF SESSIONS

Thursday, April 21
Opening Session .... 1:00 p.m.

Friday, April 22
Reference Committee Hearings ... 8:30 a.m.–5:00 p.m.

Saturday, April 23
Second Session .... 9:00 a.m.
Third Session .... 1:30 p.m.

Sunday, April 24
Closing Session .... 9:00 a.m.

A Guide to the 1988 Annual Meeting

Policy-making in The House of Delegates

This section of the New York State Journal of Medicine is a call to the officers, delegates, and members of the Medical Society of the State of New York to attend the 182nd Annual Meeting of the Society and the meeting of its House of Delegates; 298 delegates representing 61 county medical societies, 9 district branches, specialty societies, the hospital medical staff section, 12 medical schools, the resident and medical student membership will meet at the Hilton Hotel in New York City on April 21–24, 1988.

The officers, members of the Council and the Board of Trustees, past presidents of the MSSNY, the Commissioner of the New York State Department of Health, the Past President of the American Medical Association from New York, Gerald D. Dorman, MD, and Past Executive Vice-President of the Medical Society of the State of New York, George J. Lawrence, Jr., MD, are also official representatives to the House.

Mandated by the Bylaws of the Society, this section lists the names of elected representatives, delegates who are serving on reference committees, and selections of the Nominating Committee for the election of Officers, Councilors, Trustees and AMA Delegates. Also included are the amendments to the Bylaws which will be considered by the House of Delegates.

Since the House of Delegates is the legislative body of the Society, charged with the general management of the State Society and its affairs, members of the Society are urged to use this information to consult with and advise their representatives. Resolutions introduced by the representatives bring members' concerns to the attention of the entire Society. Representatives not only have the responsibility to present the views of their constituents, but also to inform them of the actions taken by this governing body. The House of Delegates also formulates policy for the Society. No officer, councilor, board, commission, committee, or employee may initiate any policy unless such policy has been expressly approved by the House of Delegates, or by the Council of the Society.

If one were to choose the most important work of the House of Delegates, it would have to be that of the reference committees. Any member of the Medical Society of the State of New York has the right to appear before any reference committee and participate in its deliberations. It is also in the reference committees that people outside of the State Society are asked to give their views. Nonmember physicians, guests or interested observers who wish to express their opinions may, with permission, come before the reference committee and speak to the business of the committee. I strongly urge members to use this forum to express their views.

Charles N. Aswad, MD, Speaker
House of Delegates

Seymour R. Stall, MD, Vice-Speaker
House of Delegates

MEDICAL SOCIETY OF THE STATE OF NEW YORK

ORGANIZED 1807

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Nassau
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Charles D. Sherman, Jr.

Monroe
President-Elect

John A. Finkbeiner

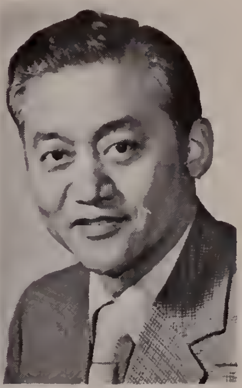
New York
Past-President

David M. Benford

New York
Vice-President

John H. Carter

Albany
Secretary



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Oneida
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Robert A. Mayers

Westchester
Assistant Treasurer

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Broome
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Seymour R. Stall

Dutchess
Vice-Speaker

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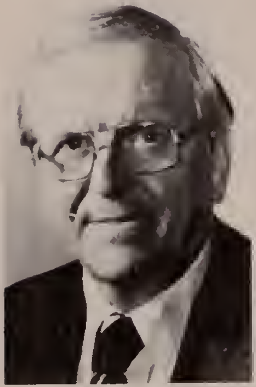
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Warren

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Broome

Sidney Mishkin

Nassau

Robert E. Fear

Suffolk

Stanley L. Grossman

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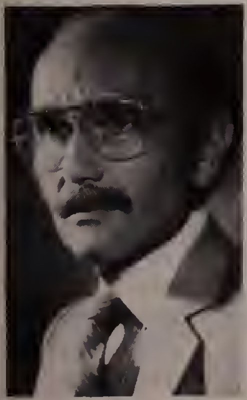
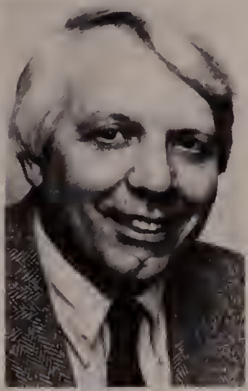
Thomas D. Pemrick

Rensselaer


Ralph E. Schlossman

Queens


Duane M. Cady

Onondaga

William A. Dolan

Monroe

Robert E. Gordon

Kings

Stuart I. Orsher

New York

Kathleen E. Squires

New York
Resident

Michael Paciorek

Onondaga
Medical Student

1988 HOUSE OF DELEGATES
MEMBERS

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Speaker—Charles N. Aswad, Broome
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Richard A. Hughes, Warren
Antonio F. LaSorte, Broome
Sidney Mishkin, Nassau

Term Expires 1989
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Stanley L. Grossman, Orange
Thomas D. Pemrick, Rensselaer
Ralph E. Schlossman, Queens

Term Expires 1990
Duane M. Cady, Onondaga
William A. Dolan, Monroe
Robert E. Gordon, Kings
Stuart I. Orsher, New York (elected to serve until 1988)

Student Councilor
Term Expires 1988
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Term Expires 1988
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Daniel F. O'Keeffe, Warren
Allison B. Landolt, Westchester

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1971–1972—George Himler, New York
1972–1973—Edward Siegel, Clinton
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1975–1976—Ralph S. Emerson, Nassau
1976–1977—George L. Collins, Jr., Erie
1977–1978—Carl Goldmark, Jr., New York
1978–1979—George T. C. Way, Dutchess
1980–1981—Ralph M. Schwartz, Kings
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1983–1984—Bernard J. Pisani, New York
1984–1985—Allison B. Landolt, Westchester
1985–1986—Daniel F. O'Keeffe, Warren
1986–1987—John A. Finkbeiner, New York

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Past President of the American Medical Association
Gerald D. Dorman, New York

Speaker of the American Medical Association
John L. Clowe, Schenectady

Commissioner, New York State Department of Health

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Second—Bruce H. Berlin, Nassau
Third—Thomas W. Greenlees, Schoharie
Fourth—
Fifth—Frederic A. Stone, Jefferson
Sixth—Anton A. Vreede, Broome
Seventh—Norman R. Loomis, Wayne
Eighth—Robert J. Reszel, Niagara
Ninth—John A. Ramsdell, Westchester

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New York State Society of Dermatology—David Sibulkin, New York
New York Chapter American College of Emergency Physicians—Ralph Altman, New York
New York State Academy of Family Physicians—James F. Wright, Warren
New York State Society of Internal Medicine—Robert M. Kohn, Erie
New York State Neurosurgical Society, Inc.—Russel H. Patterson, Jr., New York
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New York State Society of Orthopaedic Surgeons, Inc.—Leo A. Green, Queens
New York State Society of Otolaryngology—Head and Neck Surgery—Fouad B. Michael, Nassau
New York State Society of Pathologists—Edward C. Zaino, Nassau
American Academy of Pediatrics—District 11—
New York Society of Physical Medicine and Rehabilitation, Inc.—
New York Regional Society of Plastic and Reconstructive Surgery—
New York State Psychiatric Association Inc.—George L. Ginsberg, New York
The New York State Radiological Society, Inc. (A Chapter of the American College of Radiology)—Victor A. Panaro, Erie
New York State Society of Surgeons, Inc.—Theodore C. Max, Oneida
New York State Urological Society, Inc.—Marcus L. Shoobe, Albany

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James H. Puleo, Albany
Nathan P. Reed, Albany

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Sana Bloch, Bronx
Marcos A. Charles, Bronx
Dattatraya G. Lanjewar, Pelham Manor
Charles LaVerdi, Bronx
Stanley Luftschein, Bronx
Hugo M. Morales, Bronx
Raul Pohorille, New Rochelle
Howard S. Schwartz, Bronx
Juana Toporovsky, Bronx

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Theodore R. Poritz, Binghamton
Jeffrey A. Ribner, Binghamton
Irwin J. Rosenberg, Endicott

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Adil Al-Humadi, Olean

Cayuga (1)
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Chautauqua (2)
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Dana Wheelock, Dunkirk

Chemung (2)
John S. Forrest, Elmira
Charles R. Friend, Elmira

Chenango (1)
Norman C. Lyster, Jr., Norwich

Clinton (2)
Soham S. Patel
Robert T. Potter

Columbia (1)

Cortland (1)
S. Robert Paik, Cortland

Delaware (1)
Gary Preiser, Walton

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Stephen R. Chernay, Fishkill
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Arthur W. Menken, Poughkeepsie

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Thomas W. Bradley, Kenmore
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Allen L. Lesswing, Snyder
Leo E. Manning, Amherst
M. Luther Musselman, Buffalo
Nancy H. Nielsen, Orchard Park
James F. Phillips, Buffalo
Anthony P. Santomauro, Tonawanda

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Franklin (1)
Alfred A. Hartmann, Sr., Malone

Fulton (1)

Genesee (1)
Bruce E. Baker, Le Roy

Greene (1)
Karuvath Enu, Catskill

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Vashti R. Curlin, Brooklyn
Paul Finkelstein, Brooklyn
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Bentley D. Merrim, Brooklyn
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Abdul Rehman, Staten Island
Veronica Santilli, Brooklyn
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M. Theodore Tanenhaus, Brooklyn

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Livingston (*1*)
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Madison (*1*)
Leonard A. Argentine, Oneida

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Warren Glaser, Rochester
William A. Kern, Jr., Rochester
Mario B. LoMonaco, Rochester
Joyce M. McChesney, Rochester
Carl C. Sansocie, Brockport
Edward C. Tanner, Rochester

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Lee S. Binder, Valley Stream
Jeffrey T. Kessler, Great Neck
Arnold D. Lurie, Baldwin
Alfred W. Marks, Rockville Centre
Felix A. Monaco, Rockville Centre
John A. Ostuni, Freeport
E. John Pesiri, Massapequa
Neal A. Sckolnick, Rockville Centre
Arthur J. Wise Jr., Manhasset

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Melvin H. Becker, Manhasset
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Irwin J. Cohen, New York
Hugh Clark Davidson, New York
Jonas M. Goldstone, New York
Wilbur J. Gould, New York
George Hyams, New York
Virginia Kanick, New York
Vance Lauderdale, Jr., New Jersey
Samuel H. Madell, New York
Aaron G. Meislin, New York
Alfred Moldovan, New York
Richard B. Nolan, New York
Donna B. O'Hare, New York
Richard N. Pierson, Jr., New York
William B. Rawls, Pleasant Valley
William B. Rosenblatt, New York
Albert M. Schwartz, New York
Clifford L. Spingarn, New York
Meyer Texon, New York

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Claus M. Fichte, Niagara Falls
James J. Kropelin, Niagara Falls

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Neville W. Harper, Rome
Robert S. Manogue, Utica

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John A. Hoepner, Syracuse
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Arthur Lehrman, Syracuse
Ronald A. Naumann, Syracuse
George P. Tilley, Syracuse

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Kenneth T. Steadman, Geneva

Orange (*4*)
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Stanley F. Brunn, Port Jervis
Michael A. Parmer, Port Jervis
Seymour J. Wiener, Port Jervis

Orleans (*1*)
David B. L. Meza III, Albion

Oswego (*1*)
Warren Hollis, Lacona

Otsego (*1*)
Rodman D. Carter, Cooperstown

Putnam (*1*)

Queens (*16*)
Marvin L. Blumberg, Jamaica
Daniel Chansky, Forest Hills
John W. V. Cordice, Jr., St. Albans
Daniel G. Deckler, Richmond Hill
Alexander M. De la Garza, Flushing
Robert M. Farrell, Flushing
Fred S. Fensterer, Glendale
Fred N. Flatau, Flushing
Lorraine M. Giordano, Jamaica Estates
Victor Guarneri, Bayside
Nicetas H. Kuo, Great Neck
Maxwell J. Marder, Forest Hills
Frank Ratner, Flushing
Hilda Ratner, Beechhurst
Sheldon D. Sax, Whitestone
Ezra A. Wolff, Manhasset

Rensselaer (*2*)

Richmond (*4*)
Albert B. Accettola, Jr., Staten Island
Robert Bonvino, Staten Island
Tano S. Carbonaro, Staten Island
Gerald J. Lustig, Staten Island

Rockland (*4*)
Herbert Z. Geller, New City
Michael A. Kalvert, New City
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Stanley H. Oransky, Bardonia

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John M. Spring, Schenectady
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Schuyler (*1*)
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Clive D. Caplan, Islip Terrace
Frank R. Collier, Port Jefferson
John G. Egner, West Islip
Robert A. Held, Islip Terrace
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Eliot Rubinstein, Ithaca

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Geraldine T. Keyes, Stone Ridge

Warren (*2*)

Washington (*1*)
Arnold B. Wise, Cambridge

Wayne (*1*)
William J. Braell, Palmyra

Westchester (*9*)
Ann C. Cea, Rye Brook
Richard L. Fenton, Tarrytown
Mark L. Fox, Bronxville
Thomas A. Lansen, Valhalla
Armond V. Mascia, Tarrytown
Stanley M. Mendelowitz, Tarrytown
Richard L. Petrillo, Mount Vernon
Thomas D. Rizzo, Bronxville
William J. Walsh, Jr., White Plains

Wyoming (*1*)

Yates (*1*)

Medical School Delegates (12)
Albany Medical College—Robert L. Friedlander, Albany
Albert Einstein College of Medicine of Yeshiva University of New York—Melvin N. Zelefsky, Bronx
Columbia University College of Physicians and Surgeons—Linda D. Lewis, New York
Cornell University Medical College—Lawrence Scherr, Nassau
Mount Sinai School of Medicine of the City University of New York—David I. Cohen, New York
New York Medical College—Marilyn Pearl, New York
New York University of Medicine—Stanford Wessler, New York
State University of New York Health Science Center at Brooklyn—Schuyler G. Kohl, Kings
State University of New York at Buffalo School of Medicine and Biomedical Sciences
State University of New York at Stony Brook School of Medicine—Dorothy Spiegel Lane, Suffolk
State University of New York Health Science Center at Syracuse—John Bernard Henry, Onondaga
University of Rochester School of Medicine and Dentistry—Thomas E. Cardillo, Monroe

Medical Student Delegates (3)
Ellen Polokoff, New York
Victor Sloan, New York
Martin Trott, Monroe

Resident Membership Delegate (1)
Matthew H. Carabasi, New York

Hospital Medical Staff Delegate (1)

REFERENCE COMMITTEES

Reports of Officers and Administrative Matters

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Rodman D. Carter, Otsego
Stanley Luftschein, The Bronx
Donna B. O'Hare, New York
George L. Ginsberg, New York State Psychiatric Association (Observer)

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President-Elect
Secretary
Treasurer
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Budget and Finance
Executive Vice-President
New York Delegation to the AMA
District Branches
Liaison Committee with the Nursing Profession
Physicians' Home
Administrative Committee for the MSSNY Pension Program
Membership
General Insurance
Membership Programs
Publications, Library, and Archives
  Ad Hoc Advisory Committee on Real Estate

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Mark L. Fox, Westchester
Robert L. Held, Suffolk
Hilda Ratner, Queens

Governmental Affairs and Legal Matters

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Eleanor C. Kane, Dutchess
Norman R. Loomis, Seventh District Branch
Gerald J. Lustig, Richmond
Gary Preiser, Delaware
Nancy H. Nielson, Erie (Observer)

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Judicial Council
State Legislation
Federal Legislation
New York State Association of the Professions
Liaison Committee with HANYS
Professional Medical Liability Insurance and Defense Board
Conduct of OPMC Investigations

Scientific and Educational Activities

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Victor Guarneri, Queens
Mario B. LoMonaco, Monroe
James F. Wright, New York State Academy of Family Physicians
Bruce H. Berlin, Second District Branch (Observer)

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Accident and Injury Prevention
Aging and Nursing Homes
Alcoholism
Cancer
Cardiovascular Disease
Child Abuse
Drug Abuse
Emergency Medical Services
Environmental Quality
Home Health Care and Hospice
Maternal and Child Health
Mental Health
Physical Medicine and Rehabilitation
Preventive Medicine
School Health and Sports Medicine

Bioethical Issues
Continuing Medical Education
Forensic Medicine
Information Technology in Medicine
Medical School Relations
Physicians' Health
Scientific Program
  Ad Hoc Committee on Gellhorn Report

Socio-Medical Economics

Joyce M. McChesney, Monroe, *Chairman*
Vernal G. Cave, Kings
Irwin J. Cohen, New York
Roy H. Lieberman, Rockland
Arthur J. Wise, Nassau
Lawrence Scherr, Cornell University Medical College (Observer)

ANNUAL REPORTS REFERRED TO COMMITTEE:

Health Care Delivery Systems
Hospital and Professional Relations
Interspecialty
Medicaid
Medical Care Insurance
Social Security Disability Benefits Program, Committee to Provide Input Into
Workers' Compensation and Occupational Health

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Elise H. Korman, Suffolk
E. John Pesiri, Nassau (Observer)

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James J. Kropelin, Niagara, *Chairman*
Henry J. Dobies, St. Lawrence
David B. L. Meza, III, Orleans
Veronica Santilli, Kings
Ezra A. Wolff, Queens
Ronald A. Naumann, Onondaga (Observer)

Sergeant at Arms

Robert M. Bonvino, Richmond, *Chairman*
Neville W. Harper, Oneida
M. Luther Musselman, Erie
Eliot Rubinstein, Tompkins
William Braell, Wayne (Observer)

Tellers

Stanley Brunn, Orange, *Chairman*
Victorino Anllo, Erie
Sana Bloch, The Bronx
Matthew W. Carabasi, Resident Membership
Marilyn Pearl, New York Medical College
Jonas M. Goldstein, New York (Observer)

NOMINATING COMMITTEE

To the House of Delegates, Ladies and Gentlemen:

In accordance with Article XI, Section 2 of the Bylaws of the Medical Society of the State of New York, the Nominating Committee met at the Society's headquarters, 420 Lakeville Road, Lake Success, New York, on Thursday, October 22, 1987 at 2:15 p.m.

Members of the committee present were:
Member-at-Large—**John A. Finkbeiner, M.D.,** New York, *Chairman*
First District Branch—**Stuart I. Orsher, M.D.,** New York
Second District Branch—**John A. Ostuni, M.D.,** Nassau
Third District Branch—**Gloria S. Aitken, M.D.,** Sullivan
Fourth District Branch—**John D. Fulco, M.D.,** Schenectady
Fifth District Branch—**Robert M. George, M.D.,** Oneida
Seventh District Branch—**James M. Flanagan, M.D.,** Wayne
Eighth District Branch—**Leo E. Manning, M.D.,** Erie
Ninth District Branch—**Seymour R. Stall, M.D.,** Dutchess
Member-at-Large—**James H. Cosgriff, Jr., M.D.,** Erie

Excused was: Anthony F. LaSorte, M.D. of the Sixth District Branch. The President appointed Samuel M. Gelfand, M.D., to represent the interests of that district on the committee.

After careful consideration of the recommendations submitted by the district branches, the county medical societies, and the resident and student membership in response to a memorandum, dated July 1, 1987, your committee respectfully nominated the following candidates for election on April 24, 1988:

President—**Charles D. Sherman, Jr., M.D.,** Monroe
President-Elect—**David M. Benford, M.D.,** New York
Vice-President—**Robert A. Mayers, M.D.,** Westchester
Secretary—**John H. Carter, M.D.,** Albany
Assistant Secretary—**George Lim, M.D.,** Oneida
Treasurer—**Morton Kurtz, M.D.,** Queens
Assistant Treasurer—**James H. Cosgriff, Jr., M.D.,** Erie
Speaker—**Charles N. Aswad, M.D.,** Broome
Vice-Speaker—**Seymour R. Stall, M.D.,** Dutchess
Councilors: (*four for three years*)—**Franklin A. DePeters, M.D.,** Warren
—**Sidney Mishkin, M.D.,** Nassau
—**Jeffrey A. Ribner, M.D.,** Broome
—**Anthony P. Santomauro, M.D.,** Erie
(*one for two years*)—**Stuart I. Orsher, M.D.,** New York
Resident Councilor: (*one for one year*)—**Robert J. Newborn, M.D.,** New York
Student Councilor: (*one for one year*)—***Penny Stern Schulman,** The Bronx
Trustees: (*two for five years*)—**John A. Finkbeiner, M.D.,** New York
—**James M. Flanagan, M.D.,** Wayne
(*one for one year*)—**M. Theodore Tanenhaus, M.D.,** Kings

Delegates to the American Medical Association for two years commencing January 1, 1989:
Charles N. Aswad, M.D., Broome
Edgar P. Berry, M.D., New York
Randall D. Bloomfield, M.D., Kings
John Lee Clowe, M.D., Schenectady
Hugh Clark Davidson, M.D., New York
William A. Dolan, M.D., Monroe
Richard D. Eberle, M.D., Onondaga
Robert E. Fear, M.D., Suffolk
Louis W. Giordano, M.D., Broome
Robert E. Gordon, M.D., Kings
Stanley L. Grossman, M.D., Orange
James J. Kropelin, M.D., Niagara
Dattatraya G. Lanjewar, M.D., The Bronx
Samuel H. Madell, M.D., New York
Robert A. Mayers, M.D., Westchester
Joyce M. McChesney, M.D., Monroe
Donna B. O'Hare, M.D., New York
Richard N. Pierson, Jr., M.D., New York
John Piper, M.D., Onondaga (Resident)
Irwin J. Rosenberg, M.D., Broome
Anthony P. Santomauro, M.D., Erie
Ralph M. Schwartz, M.D., Kings
Stanley A. Steckler, M.D., Suffolk
George T. C. Way, M.D., Dutchess

The twelve nominees receiving the largest number of votes will be delegates; the second twelve will be alternates.

Respectfully submitted:
John A. Finkbeiner, M.D., *Chairman*

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*Medical Student

HOUSE COMMITTEE ON BYLAWS

To the House of Delegates, Ladies and Gentlemen:

The members of the House Committee on Bylaws are as follows:

George Lim, M.D., Chairman	Oneida
Stanley F. Brunn, M.D.	Orange
Rodman D. Carter, M.D.	Otsego
Robert M. Kohn, M.D.	Erie
Sidney Mishkin, M.D.	Nassau
James R. Nunn, M.D.	Erie
Donna B. O'Hare, M.D.	New York
Hilda Ratner, M.D.	Queens
Stanley A. Steckler, M.D.	Suffolk
Samuel M. Gelfand, M.D., President, ex officio	Nassau
John H. Carter, M.D., Secretary, ex officio	Albany
Charles N. Aswad, M.D., Speaker, ex officio	Broome
Donald F. Foy	Executive Vice President, ex officio
Robert J. O'Connor, M.D.,	Deputy Executive Vice President, ex officio
Donald R. Moy, J.D.	General Counsel, ex officio

A meeting of the Reference Committee on Bylaws was held at the Hilton Hotel in New York City on March 13, 1987. The Reference Committee prepared a transcript of testimony and a summary of its recommendations to the House Committee on Bylaws. The input received from the meeting of the Reference Committee on Bylaws proved to be of great assistance to the House Committee on Bylaws when it met on October 1, 1987.

In all, seven resolutions asking for amendments to the Bylaws were referred to the Committee for its consideration.

Resolution 87-9, Direct Election of Councilors by District Branches

Introduced by New York State Society of Orthopaedic Surgeons and New York State Society of Surgeons

Whereas, An Ad Hoc Committee of the House of Delegates in 1975 studied the matter of direct election of councilors by members of the District Branches and recommended that they be so elected; and

Whereas, The Committee to that end recommended that the Council be increased to 16 members, and the House recommended that the allocation be as follows:

District 1 — New York and Richmond 3, Kings 1, Queens 1, Bronx 1
District 2 — Nassau 1, Suffolk 1
District 3–8 — 1 councilor each
District 9 — 2 councilors;

and

Whereas, The reasoning behind the call for direct election of councilors is as valid, if not more valid, today as it was in 1975; therefore be it

Resolved, That the Medical Society of the State of New York amend Article III, Section 2, paragraph 2 of the Bylaws to read as follows: (deletion in brackets)

"It shall elect the officers, [councilors] and trustees of the State Society and the delegates to the American Medical Association. A student member of the Medical Society of the State of New York, who is in good standing, is eligible for election as delegate to the American Medical Association, provided he is a member of the AMA.";

and be it further

Resolved, That Article III, Section 3, paragraph 15 of the Bylaws be amended to read as follows: (deletions in brackets)

"As the first business of the last scheduled session of the annual meeting of the House of Delegates, the officers, [councilors] and the trustees of the Medical Society of the State of New York, and the delegates to the American Medical Association shall be nominated and elected.";

and be it further

Resolved, That the Medical Society of the State of New York amend Article IV, Section 1, paragraph 1 of the Bylaws to read as follows: (additions italicized, deletions in brackets)

"The Council shall be composed of the president, president-elect, vice president, immediate past-president, secretary, assistant secretary, treasurer, assistant treasurer, speaker, vice-speaker, chairman of the Board of Trustees, and [14] *16 District* councilors, [elected by the House of Delegates"] *nominated and elected by the members of the District Branches which they are to represent. The 16 councilors shall be allocated to the District Branches as follows:*

District 1 — New York and Richmond 3, Kings 1, Queens 1, Bronx 1
District 2 — Nassau 1, Suffolk 1
District 3–8 — 1 councilor each
District 9 — 2 councilors;

and be it further

Resolved, That Article IV, Section 1, paragraph 3 of the Bylaws be amended to read as follows: (deletions in brackets)

"[Four councilors shall be elected annually by the House of Delegates, each for a term of three years.] Two councilors, one councilor representing the student membership and one councilor representing the resident membership to the Medical Society of the State of New York, shall be elected every year by the House of Delegates, each for a term of one year. Article IV, Section 1, paragraph 4 is not applicable to the term of office of a resident or student councilor. [In the event of a vacancy, a councilor shall be elected by the Council to serve until the next meeting of the House of Delegates, at which time the House of Delegates shall elect a councilor to fill the unexpired term.]";

and be it further

Resolved, That Article IV, Section 1, paragraph 6 of the Bylaws be amended to read as follows: (deletions in brackets)

"[Councilors other than the councilor representing the student membership and the councilor representing the resident membership shall be assigned to specific county societies as liaison for the Council in accordance with the provisions of Article V, Section 2.] Councilors shall be required to disseminate information of Council activity as well as returning information to the Council, and shall report regularly to the Council on their activity. The councilor representing the student membership must be a student member of the Medical Society of the State of New York; the councilor representing the resident membership must be a resident member of the Medical Society of the State of New York.";

and be it further

Resolved, That Article V, Section 2, paragraph 8 be deleted. Article V, Section 2, paragraph 8 reads as follows: (deletions in brackets)

"[He shall assign Councilors as liaison between the Council and specific county medical societies for the purpose of the dissemination of information to such societies.]";

and be it further

Resolved, That the changes affected by this resolution shall take effect for the election of councilors to the Medical Society of the State of New York which is held in the year 1988, but that as to incumbents shall take effect as their terms expire until all 16 councilors allocated to the District Branches shall have been elected by the members of the District Branches they are to represent; and be it further

Resolved, That the Council shall establish, after consultation with the president of each District Branch, standing rules governing the method and procedures to be followed by each District Branch in nominating and electing the councilor or councilors to which it is entitled.

The House of Delegates has considered the issue of Direct Elections of Councilors in 1978, through Resolution 77-1. The question regarding Direct Election failed to obtain a majority, let alone the required two-thirds majority. The issue was considered again in 1979 through Resolutions 78-1 and 78-8. Again, less than a simple majority favored direct elections. In 1980, the House of Delegates considered the issue one more time through Resolution 79-3. As in previous years, less than a simple majority supported direct elections.

It is noted that Resolution 87-9 was not introduced by a district branch. Furthermore, the Reference Committee on Bylaws heard no testimony in favor of Resolution 87-9. The House Committee on Bylaws, therefore, sees no reason to support the concept of direct election.

The House of Delegates has also considered many previous proposals to increase the number of councilors (e.g. Resolution 77-1, 77-3, 78-1). These proposals have been rejected by the House of Delegates in previous years and the House Committee on Bylaws has not heard any additional testimony why an increase in the number of councilors is warranted at this time.

The Committee, therefore, unanimously recommends that Resolution 87-9 **NOT BE ADOPTED.**

Resolution 87-23, Life Membership

Introduced by Medical Society of the County of Rensselaer, Inc.

Whereas, Resolution 83-6, which changed the Bylaws requirement for life membership status, is repugnant to the Medical Society of the County of Rensselaer, Inc. and appears to create a financial drain on the physicians with minimal benefits to the retired members of the Medical Society of the State of New York; therefore be it

Resolved, That Article II, Section 1, paragraph 6 of the Bylaws of the Medical Society of the State of New York be amended as follows: (additions italicized, deletions in brackets)

"An active member in good standing who has completely withdrawn from the active practice of medicine and who has been a member in good standing for the ten consecutive years prior to the attainment of the age of [sixty seven] *sixty three* years or an active member in good standing who continues in the active practice of medicine and who has been a member in good standing for the ten consecutive years prior to the attainment of the age of [seventy-two] *seventy* years, or an active member in good standing for ten consecutive years or more who is permanently disabled, may apply for life membership. The House of Delegates or the Council may waive the requirements in a proper case where there appear to be extenuating circumstances. He/*she* shall apply for such life membership to the component county medical society of which he/*she* is a member. His/*her* application shall be governed by the constitution and bylaws of the component medical society relative to active membership."

The House Committee on Bylaws is concerned that the adoption of this resolution would cause a serious loss of income to the Medical Society. According to a Medical Society estimate, the projected loss of income to MSSNY for 1989 alone would be $305,000. In addition, there would be losses to individual county medical societies. The Committee believes that it is unnecessary to lower the age requirement for life membership because the Bylaws of the Medical Society of the State of New York already provide procedures in which a member with financial hardship may apply for a remission of dues or a waiver of life membership requirements.

The Committee, therefore, unanimously recommends that Resolution 87-23 **NOT BE ADOPTED.**

A memorandum prepared by Eunice M. Skelly, Director, Division of Membership Support Services, provides projected losses of income under Resolutions 87-23, 87-73, 87-75 and 87-76. This memorandum is appended to the end of this report.

Resolution 87-73, Special Consideration for Limited Practice or Early Retirement

Introduced by Onondaga County Medical Society, Inc.

Whereas, Physician members who may be sixty years of age and over (but less than life membership age), and have been dues paying members for twenty-five years or more, may discontinue or limit their practice to minimal office work, although they are not sufficiently ill to warrant consideration as an invalid; and

Whereas, Because of their longstanding loyalty, they wish to retain some affiliation with the Medical Society of the State of New York and their county medical society; and

Whereas, Modifying the dues structure for physicians who discontinue or limit their practice prior to attaining the age required for life membership to recognize their diminished income and changed status would be evidence that their support and counsel are appreciated by the Medical Society of the State of New York; therefore be it

Resolved, That the Medical Society of the State of New York amend its Bylaws Article XV by inserting a new paragraph between paragraph 3 and paragraph 4 to read as follows: (additions italicized)

The dues and assessments of those active members who are not yet eligible for life membership and who have been dues paying active members of the Medical Society of the State of New York for twenty-five years or more who limit their practice or discontinue their practice shall be one-third the amount levied on all other members, until such time as they become eligible for life membership.

and

Resolution 87-75, Retired Physicians Membership Category

Introduced by Medical Society of the County of Niagara, Inc.

Whereas, There are many physicians who are retiring from the active practice of medicine at an age which is younger than that which is now cited in the Bylaws of the Medical Society of the State of New York as a requirement for life membership; and

Whereas, It is important to make it possible for all our members to maintain their membership after retirement; and

Whereas, Many of these members feel it is not in their best interest to continue such membership because of the expense related to dues; and

Whereas, This also leads to a loss of members at the county level; and

Whereas, If we are to truly represent all physicians, we require as many members as possible and must retain retired physicians as members; and

Whereas, A decrease in dues will enable the Medical Society of the State of New York, as well as component county medical societies, to retain early retirees as members of their respective societies and enable those members to continue receiving the benefits of such membership; therefore be it

Resolved, That an active member of MSSNY who has completely withdrawn from the active practice of medicine and who is and has been a member in good standing for the ten consecutive years prior to the date of application for such status, may apply for retired status; and be it further

Resolved, That Article II, Section 1, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended to read as follows: (additions italicized, deletions in brackets)

The membership in the Medical Society of the State of New York shall be divided into [five] *six* classes: (a) active, (b) *retired*, (c) life, (d) honorary, (e) resident, and (f) student.

and be it further

Resolved, That Article II, Section 1, paragraph 6 of the Bylaws of the Medical Society of the State of New York be amended as follows: (additions italicized, deletions in brackets)

An active member in good standing who has completely withdrawn from the active practice of medicine and who has been a member in good standing for [the] ten consecutive years [prior to the attainment of the age of sixty-seven years or] *may apply for retired status*. An active member in good standing who continues in the active practice of medicine and who has been a member in good standing for the ten consecutive years prior to the attainment of the age of seventy-two years, or an active member in good standing for ten consecutive years or more who is permanently disabled, may apply for life membership. The House of Delegates or the Council may waive the requirements in a proper case where there appear to be extenuating circumstances. He shall apply for such *retired status or* life membership to the component county medical society of which he is a member. His application shall be governed by the constitution and bylaws of the component county medical society relative to active membership.

and be it further

Resolved, That Article II, Section 2, paragraphs 1 and 3 of the Bylaws be amended to read as follows:

Article II, Section 2, paragraph 1 (additions italicized)

Active members vote and hold office in the Medical Society of the State of New York. *Retired members may not vote and hold office in the Medical Society of the State of New York.*

Article II, Section 2, paragraph 3 (additions italicized, deletions in brackets)

Life members shall not be subject to Medical Society of the State of New York dues or assessments but shall be accorded all the rights and privileges of active membership. [They] *Life members and retired members* shall be entitled to receive the New York State Journal of Medicine and the News of New York, but the Medical Directory of New York State only on request.

and be it further

Resolved, That Article XV, paragraph 3 of the Bylaws be amended to read as follows: (additions italicized)

The dues and assessments of those active members who have been graduated from medical or osteopathic college not more than five calendar years, not counting active service in the armed forces of the United States or in the United States Public Health Service, shall be one third the amount levied on all other active members. The dues and assessments of those active members who have not completed their continuous residency training shall be one tenth the amount levied on all other active members. In no case may an active physician continue to pay such reduced dues more than seven years after the date of graduation from medical or osteopathic school. The dues and assessments of all active members entering practice for the first time, who are not otherwise eligible for reduced dues as set forth in prior provisions in this paragraph, shall be one half the amount levied on all other active members for the first two years of practice. Once an active member pays full dues, he shall no longer qualify for half-dues status. *The dues and assessments of retired members shall be 25% of the amount levied on all other active members.*

and

Resolution 87-76, New Dues for Fully Retired Physicians

Introduced by Medical Societies of the Counties of Genesee and Orleans, Inc.

Whereas, There are an increasing number of physicians who are retiring from medical practice at a much earlier age than in previous years due to the continuing professional medical liability insurance premium crisis and other socioeconomic reasons; and

Whereas, Many of these physicians are members of the Medical Society of the State of New York and its component county medical societies who are not eligible for life membership due solely to their age; and

Whereas, Many such physicians have expressed an interest in continuing their membership in the County, State, and American Medical Associations; and

Whereas, The AMA has recently introduced a new, fully retired physicians dues category; therefore be it

Resolved, That the Medical Society of the State of New York establish a new fully retired dues category similar to that of the American Medical Association; and be it further

Resolved, That physicians who have been members in good standing for ten consecutive years, are fully retired from active practice, but are not eligible for life membership be afforded the opportunity to continue their membership in the County and State Medical Associations at a dues rate of 20% of the regular active dues; and be it further

Resolved, That Article II, Section 1, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended to read as follows: (additions italicized, deletions in brackets)

The membership in the Medical Society of the State of New York shall be divided into [five] *six* classes: (a) active, (b) *retired*, (c) life, (d) honorary, (e) resident, and (f) student.

and be it further

Resolved, That Article II, Section 1, paragraph 6 of the Bylaws of the Medical Society of the State of New York be amended as follows: (additions italicized, deletions in brackets)

An active member in good standing who has completely withdrawn from the active practice of medicine and who has been a member in good standing for [the] ten consecutive years [prior to the attainment of the age of sixty-seven years or] *may apply for retired status.* An active member in good standing who continues in the active practice of medicine and who has been a member in good standing for the ten consecutive years prior to the attainment of the age of seventy-two years, or an active member in good standing for ten consecutive years or more who is permanently disabled, may apply for life membership. The House of Delegates or the Council may waive the requirements in a proper case where there appear to be extenuating circumstances. He shall apply for such *retired status or* life membership to the component county medical society of which he is a member. His application shall be governed by the constitution and bylaws of the component county medical society relative to active membership.

and be it further

Resolved, That Article II, Section 2, paragraphs 1 and 3 of the Bylaws be amended to read as follows:

Article II, Section 2, paragraph 1 (additions italicized)

Active *and retired* members vote and hold office in the Medical Society of the State of New York.

Article II, Section 2, paragraph 3 (additions italicized, deletions in brackets)

Life members shall not be subject to Medical Society of the State of New York dues or assessments but shall be accorded all the rights and privileges of active membership. [They] *Life members and retired members* shall be entitled to receive the New York State Journal of Medicine and the News of New York, but the Medical Directory of New York State only on request.

and be it further

Resolved, That Article XV, paragraph 3 of the Bylaws be amended to read as follows: (additions italicized)

The dues and assessments of those active members who have been graduated from medical or osteopathic college not more than five calendar years, not counting active service in the armed forces of the United States or in the United States Public Health Service, shall be one third the amount levied on all other active members. The dues and assessments of those active members who have not completed their continuous residency training shall be one tenth the amount levied on all other active members. In no case may an active physician continue to pay such reduced dues more than seven years after the date of graduation from medical or osteopathic school. The dues and assessments of all active members entering practice for the first time, who are not otherwise eligible for reduced dues as set forth in prior provisions in this paragraph shall be one half the amount levied on all other active members for the first two years of practice. Once an active member pays full dues, he shall no longer qualify for half-dues status. *The dues and assessments of retired members shall be 20% of the amount levied on all other active members.*

Because of the similarity of content, the Committee considered Resolutions 87-73, and 87-75 and 87-76 at the same time. Resolution 87-73 would provide that members who have been dues paying active members of the Medical Society of the State of New York for 25 years or more and are "semi-retired" would pay one-third the dues paid by active members. Resolution 87-75 would create a "retired" membership category consisting of active members who have been in good standing for ten consecutive years and who have completely withdrawn from the active practice of medicine. The dues for a "retired" member would be 25% of the dues paid by an active member. Resolution 87-76 is similar to Resolution 87-75 except under Resolution 87-76, a retired member would pay 20% of the dues paid by an active member.

The House Committee on Bylaws believes that all three resolutions would cause an unacceptable loss of income to the Medical Society of the State of New York and county medical societies.

The Committee, therefore, unanimously recommends that Resolutions 87-73, 87-75 and 87-76 **NOT BE ADOPTED.**

Resolution 87-72, Deletion of Reinstatement Penalty

Introduced by Frank J. Piper, M.D., as an Individual

Whereas, It is a stated goal of the Medical Society of the State of New York to increase membership by 2,000 in each of the next four years; and

Whereas, Every effort should be made to support the Executive Vice-President, the staff of the Division of Membership Support Services, and the Membership Committee of the Medical Society of the State of New York to meet that challenge; and

Whereas, Amended Resolution 84-11 provided for the temporary deletion of the reinstatement penalty and this provision should be reactivated for another four years; therefore be it

Resolved, That the Medical Society of the State of New York amend Article II, Section 2, paragraph 15, of its Bylaws to read as follows: (deletions in brackets, additions italicized)

The provision in Article II, Section 2, paragraph 14 that no member dropped for nonpayment of dues and assessments shall be reinstated until he has, in addition, paid his dues and assessments for the year in which he was dropped, shall not be in effect for the period beginning June 6, 1985 through April 1, [1986] *1991.*

and be it further

Resolved, That Article XV, paragraph 9 be amended to read as follows: (deletions in brackets, additions italicized)

The provision in Article XV, paragraph 8 that no member dropped for non-payment of dues and assessments shall be reinstated until he has, in addition, paid his dues and assessments for the year in which he was dropped, shall not be in effect for the period beginning June 6, 1985 through April 1, [1986] *1991.*

Resolution 84-11 was approved by the House of Delegates in 1985 to temporarily delete the reinstatement penalty under paragraph 8 of Article XV of the MSSNY Bylaws. By its own terms, the deletion of the reinstatement penalty was to run from June 6, 1985 through April 1, 1986. At the time Resolution 84-11 was adopted, it was intended that the deletion of the reinstatement penalty would be for a short period of time. It was believed that a longer "grace" period might encourage some members to drop their membership. Furthermore, a prolonged extension of the grace period would have the effect of penalizing members who have no lapses in membership.

Accordingly, the House Committee on Bylaws does not believe that the deletion of the reinstatement penalty should be extended.

The Committee, therefore, unanimously recommends that Resolution 87-72 **NOT BE ADOPTED.**

Resolution 87-79, Election Process

Introduced by the Council

Whereas, Article III, Section 3, paragraph 15 of the Bylaws of the Medical Society of the State of New York provides that as the first business of the last scheduled session of the annual meeting of the House of Delegates, the officers, councilors, and the trustees of the Medical Society of the State of New York, and the delegates to the American Medical Association shall be nominated and elected; and

Whereas, Depending upon the circumstances, there are occasions where it is not opportune to hold nominations and elections as the first item of business of the last scheduled session; and

Whereas, Article VIII, Section 1 of the Bylaws of the Medical Society of the State of New York provides election and balloting procedures; and

Whereas, Election and balloting procedures should be defined in procedural rules rather than Bylaws, since this would make it more feasible for the House of Delegates to incorporate state of the art procedures; therefore be it

Resolved, That Article III, Section 3, paragraph 15 of the Bylaws of the Medical Society of the State of New York be amended to allow for a flexible election schedule; and be it further

Resolved, That Article III, Section 3, paragraph 15 of the Bylaws be amended to read as follows: (deletions in brackets, additions italicized).

[As the first business of the last scheduled session of the annual meeting of the House of Delegates,] the officers, councilors, [and the] trustees of the Medical Society of the State of New York, and the delegates to the American Medical Association shall be nominated and elected *at the annual meeting of the House of Delegates.*

and be it further

Resolved, That paragraphs 1 and 2 of Article VIII, Section 1 of the Bylaws be repealed and replaced by the following new paragraphs to read: (deletions in brackets, additions italicized)

All elections for offices of the State Society and delegates to the American Medical Association shall be by ballot. When after the call for nominations from the floor, there is only one candidate for an office, the speaker shall direct the secretary to cast one ballot on behalf of the delegates.

The speaker shall appoint a Committee on Rules and Order of Business which shall recommend election and balloting procedures to be approved by the House of Delegates.

[After the appointment of a sufficient number of tellers by the speaker and after all nominations have been made, the secretary of the Medical Society of the State of New York shall cause to be displayed in full sight of the House of Delegates a list of nominees for each office of the Medical Society of the State of New York and for delegates to the American Medical Association, arranged in alphabetical order, and shall also cause to be distributed a sufficient number of blank ballots for the use of the House of Delegates. These ballots shall have printed or stamped thereon the appropriate headings for each office with spaces thereunder in which may be written the name of the candidate or candidates to be voted for.]

[All elections for offices of the State Society and delegates to the American Medical Association shall be by ballot, each member of the House of Delegates depositing his ballot on roll call individually. When after the call for nominations from the floor, there is only one candidate for an office, the speaker shall direct the secretary to cast one ballot on behalf of the delegates. If there are more than two nominees for an office, with the exception of that of delegate to the American Medical Association, and none receives a majority of votes on the first ballot, the nominee receiving the lowest number of votes shall be dropped and a new ballot taken for that office. This procedure shall be continued until one of the nominees receives a majority of the votes cast when he shall be declared elected.]

and be it further

Resolved, That this resolution shall take effect one year after it is adopted by the House of Delegates.

The House Committee on Bylaws strongly believes that Article III, Section 3, paragraph 15 and paragraphs 1 and 2 of Section 1 of Article VIII are unduly rigid and do not provide the House of Delegates with the flexibility that may be necessary to accommodate unexpected contingencies.

The Committee believes that the Resolution should be amended by deleting the last "Resolved", and by inserting in its place a 'Resolved" to read, "That this resolution shall take effect immediately after it is adopted by the House of Delegates."

The last "Resolved" would then read (additions italicized, deletions in brackets.)

Resolved, [That this resolution shall take effect one year after it is adopted by the House of Delegates.] *That this resolution shall take effect immediately after it is adopted by the House of Delegates.*

As an editorial matter, paragraph 1 of Article VIII should be amended to be consistent with Article III, Section 3, paragraph 15. Paragraph 1 of Article VIII should be amended to read (additions italicized, deletions in brackets)

"[As the first business of the last scheduled session of the annual meeting of the House of Delegates,] the officers, councilors, and the trustees of the Medical Society of the State of New York, and the delegates of the American Medical Association shall be nominated and elected *at the annual meeting of the House of Delegates.*"

The Committee, therefore unanimously recommends that Resolution 87-79 be **ADOPTED AS AMENDED.**

Your Chairman would like to thank, in addition to the members of the Committee and the physicians who testified before it, those members of staff who helped this Committee; Eunice M. Skelly, Director, Divison of Membership Support Services, Donald R. Moy, J.D., General Counsel, Ellen Panzeca, J.D., Staff Attorney, and Kathleen Wellborn for her secretarial assistance.

Respectfully submitted,

George Lim, M.D. *Chairman*

APPENDIX

September 29, 1987

TO: Donald R. Moy, J.D.
General Counsel

FROM: Eunice M. Skelly, Director
Division of Membership Support Services

RE: Projected Effects on Dues Income of Creating a Membership Category for Retired or Semi-Retired Physicians (Resolutions 87-75; 87-76 and 87-73); and of Reducing the Age Requirements for Life Membership (Resolution 87-23)

RETIRED/SEMI-RETIRED CATEGORY

The following projections are based on data from the National Center for Health Statistics concerning retirement patterns in the general work force. These data show that in 1986, 34% of men aged 55–64 were no longer in the work force, and that for the age group 65 and older, 84% of men were not in the work force.

MSSNY currently has 5,164 members who will fall into the first age group in 1989, and an additional 2,475 who will be in the second age group, who already meet the requirement for 10 consecutive years' prior membership.

PROJECTED LOSSES OF INCOME UNDER THE THREE RESOLUTIONS APPEAR BELOW:

	Resolution 87-76 (20% dues rate)	Resolution 87-75 (25% dues rate)	Resolution 87-73* (33% dues rate)
Ages 55–64	$386,100	$361,968	$323,358
Ages 65–71	457,380	428,793	383,055
	$843,480	$790,761	$706,413*
Offset by retention	−14,190	−17,737	−23,413
Net loss	$829,290	$773,024	$683,000

* Inclusion of semi-retired physicians, as proposed in Resolution 87-73, would undoubtedly exacerbate the effects beyond what is projected here. I have assumed the same number of physicians would qualify for the category, because of the 25 year membership requirement, but in fact, of all Active Members who meet the current 10 year membership requirement, the average length of membership is almost 24 years.

REDUCING AGE LIMITS FOR LIFE MEMBERSHIP

Resolution 87-23 proposes to reduce the age requirements for Life Membership to age 63 for retired physicians, and to age 70 for all others. (The current age requirements are 67 and 72.)

Again, based on the data from the National Center for Health Statistics, I have assumed that 34% of physicians between the ages of 63 and 66 in 1989 who meet the requirement for 10 years' prior consecutive membership would qualify for Life Membership on the basis of retirement. Projected loss of income for this group in 1989 alone is as follows:

611 members @ $275 = $168,025

Additionally, all of our present dues-paying members with 10 consecutive years of membership who are currently age 68 or 69 would be eligible in 1989 for Life Membership on the basis of age. The resulting loss of income for the first year only would be:

501 members @ $275 = $137,775

Totaling the two figures, the projected loss of income *for 1989 only* would be *$305,800.*

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

COURTESY DAVID S. ARSENAULT

Medical Society of the State of New York

ANNUAL CME ASSEMBLY

Newer Approaches to Habit Abuse & Dependency

April 22–24, 1988

and the

Annual Meeting of the House of Delegates

April 21–24, 1988

The New York Hilton
Avenue of the Americas at 53rd/54th Streets
New York City

HIGHLIGHTS . . .

- 25 Sessions
- Panel Discussions
- Technical Exhibits
- Two Receptions
- President's Reception & Dinner Dance

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REMARKS ______________________________________

(*please print*)

NOTE: Reservations must be received no later than March 30, 1988 and will be held only until 6 p.m. on day of arrival unless guaranteed.

ARRIVAL DATE ______________________

(*check-in time is 3 p.m.*)

☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.

DEPARTURE DATE ______________________

(*checkout time is 1 p.m.*)

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MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
April 21–24, 1988
Annual CME Assembly
April 22–24, 1988

Circle Preferred rate:
Singles: $135 $150 $160
Doubles/Twins: $160 $175 $185
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Suites: (Parlor & 1) $365 $400
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Rates subject to 8¼% N.Y. State sales tax; $2.00 per room per night, and 5% N.Y.C. occupancy taxes. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

GENERAL INFORMATION

Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. Bring your family to New York City to enjoy the many sights.

All physicians, members of the allied professions, and their guests are invited to attend the Medical Society of the State of New York's Annual CME Assembly to be held April 22–24, 1988, at the New York Hilton. The two and one-half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. Over 125 outstanding speakers, including a number from out-of-state, will present papers at 25 sessions.

The theme of the CME Sessions will be "Newer Approaches to Habit Abuse and Dependency". A few of the subjects to be presented will be, Human Abuse Through the Ages: "From the Womb to the Tomb"; Managing Life — Style Abuses of the G.I. Tract; Wake Up! Sleep Apnea Can Kill You; AIDS and Self-Induced Skin Diseases. For details, see the Preliminary Program which appears in the January 1988 issue of the *New York State Journal of Medicine*. Our program will offer the clinician timely approaches in diagnosis and treatment of diseases in a wide range of medical specialties.

Here are comments from some of the attendees at the 1987 CME Assembly: "The best program I have attended in 25 years; "The speakers were stimulating and gave scholarly presentations", "The subjects were timely and practical". "The sessions were well organized and informative".

Professional representatives from more than 75 companies will be in the Rhinelander Gallery Exhibit Hall on Friday and Saturday to discuss their products and services. The Technical exhibits will include drug products, office management systems, medical and surgical suppliers, health insurance plans, pension plans and more.

SPECIAL EVENTS

Thursday, April 21

The Annual Dinner Dance, in honor of our President, Samuel M. Gelfand, M.D., will be held in the Trianon Ballroom of the New York Hilton. The reception will be held in the Mercury Ballroom starting at 7:00 p.m. All attendees are welcome. Tickets are $75.00 per person and should be ordered in advance from the Medical Society.

THE WOOLWORTH BUILDING
— *Courtesy David S. Arsenault*

Friday, April 22 and Saturday, April 23

A reception for physicians, their guests, the allied professions and exhibitors will be held both days in the Exhibit Hall between 5:00 p.m. and 6:00 p.m. A free drawing for a color TV set will take place at both receptions.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than March 30, 1988. After that date, rooms are on an availability basis only. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Exhibit Hall on Friday, April 22 and Saturday, April 23 from 8:00 a.m.–5:00 p.m., and on Sunday, April 24, registration will take place on the 2nd floor Promenade from 8:00 a.m.–12:30 p.m. There is a $25.00 registration fee for MSSNY members or members of any state medical society.

House of Delegates registration will take place on the 2nd and 3rd floor Promenades at the following hours: Wednesday, April 20 from 12 noon–6:00 p.m.; Thursday, April 21, Friday, April 22 from 8:30 a.m.–5:30 p.m., Saturday, April 23 from 8:30 a.m.–5:00 p.m., and Sunday, April 24 from 8:30 a.m.–12 noon. All MSSNY members are invited to attend the meetings.

CME MEETING HOURS

Friday, April 22 —	8:30am – 1pm
	2pm – 5pm
Saturday, April 23	8:30am – 1pm
	2pm – 5pm
Sunday, April 24	8:30am – 1pm

EXHIBIT HOURS

Friday, April 22 —	9am – 6pm
Saturday, April 23	9am – 6pm

1988 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City

Newer Approaches to Habit Abuse and Dependency

FRIDAY, APRIL 22

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Plenary Session: Human Abuses Through the Ages: "From the Womb to the Tomb," *Sutton South/Regent Parlor*
2:00 p.m. - 5:00 p.m.	Women's Medical Society/Occupational Health, *Gramercy B*
	Cardiovascular Diseases, *Sutton South*
	Physical Medicine & Rehabilitation/Family Practice/Orthepedics, *Gramercy A*
	Emergency Medicine/Critical Care, *Murray Hill B*
	Pathology/Pediatrics, *Bryant Suite*
	General Surgery/Radiology, *Regent Parlor*
	Neurosurgery, *Murray Hill A*
	Medical Liability Mutual Insurance Co., *Rendezvous Trianon*

SATURDAY, APRIL 23

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Internal Medicine (All Day), *Regent Parlor*
	Ophthalmology (All Day), *Sutton South*
	Hand Surgery/Neurology (All Day), *Sutton Parlor North*
	Psychiatry (All Day), *Gramercy B*
	Urology/Radiology, *Nassau A*
	Dermatology, *Murray Hill A*
2:00 p.m. - 3:00 p.m.	Medical Technology
2:00 p.m. - 5:00 p.m.	Internal Medicine, *Regent Parlor*
	Ophthalmology, *Sutton South*
	Hand Surgery/Neurology, *Sutton Parlor North*
	Psychiatry, *Gramercy B*
	Chest Diseases/Otolaryngology, *Murray Hill B*
3:00 p.m. - 5:00 p.m.	Computers in Medicine, *Nassau A*

SUNDAY, APRIL 24

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Allergy & Immunology, *Gramercy B*
	Obstetrics and Gynecology, *Murray Hill A*
	Gastroenterology/Colon and Rectal Surgery, *Nassau A*
	Orthopedics/Physical Medicine & Rehabilitation/Anesthesiology, *Regent Parlor*
	Sleep Disorders—Apnoea, *Nassau B*

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians.

□

Technical Exhibits

United Nations Headquarters Overlooking the East River
Photo courtesy of David S. Arsenault

Advance Registration Form

1988 Annual CME Assembly, April 22-24
The New York Hilton, New York City

Complete and return this form to the Medical Society address as indicated below.

Name____
PLEASE PRINT LAST FIRST

Address____

City/State/Zip____________________________________

Medical Specialty__________________________________

REGISTRATION FEE *Please check categories*

☐ Active Member*	$25.00
☐ Non-Member Physician*	$50.00
☐ Allied Profession	$10.00

- ☐ Nurse
- ☐ Physician Assist.
- ☐ Dentist
- ☐ Lab Technician
- ☐ PT/OT
- ☐ Other____________ *Specify*

NO FEE *Please check category*

- ☐ MSSNY Life Member
- ☐ Resident/Intern
- ☐ Medical Students
- ☐ Medical Assist.
- ☐ Speaker
- ☐ Spouse/Family
- ☐ News Media/Medical Writer

REMITTANCE ENCLOSED $____________

* of any state medical society

Please make check payable to:
Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services/MSSNY, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

(*continued from p 10A*)

Conference. 20 Cat 1 Credits. Mammoth Lakes. Contact: Medical Conferences, Inc, c/o CME Travel Service, 1615 S. Mission Rd, Suite #3, Fallbrook, CA 92028. Tel: 1-800-457-2777.

Mar 7–10. **Neuroradiology Update.** *Mar 7–11.* **Magnetic Resonance Imaging.** *Mar 8–11.* **MRI for Technologists.** 28 Cat 1 Credits. Hotel del Coronado, Coronado (San Diego). Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

Mar 12. **Behavioral/Developmental Pediatrics Symposium: Stress, Vulnerability and Child Health.** 6½ Cat 1 Credits. San Francisco. Contact: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Mar 17–20. **The International Symposium on Hair Replacement Surgery.** 30 Cat 1 Credits. Four Seasons Hotel, Los Angeles. Contact: American Academy of Facial Plastic and Reconstructive Surgery, 1101 Vermont Ave, NW, Suite 404, Washington, DC 20005.

Mar 17–20. **The Third ICN-UCI Symposium—The Molecular Basis of Genetic Disease.** Dana Point Hotel, Dana Point. Contact: Nita Driscoll, Symposium Office, Cancer Research Institute, Department of Molecular Biology and Biochemistry, University of California, Irvine, CA 92717. Tel: (714) 856-5886.

Mar 17–20. **Ninth Annual Meeting—The American Society of Outpatient Surgeons.** Century Plaza Hotel. Contact: American Society of Outpatient Surgeons, PO Box 33185, San Diego, CA 92103. Tel: (619) 692-9918.

Mar 19–21. **Transfusion-Associated Infections and Immune Response.** 24 Cat 1 Credits. The Fairmont Hotel, San Francisco. Contact: Sara Burke, Extended Programs in Medical Education, Box 0742, University of California, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Mar 25. **Infectious Diseases for Pulmonary and Critical Care Specialists.** 7 Cat 1 Credits. San Francisco. Contact: Continuing Education S/P, University

(*continued on p 16A*)

Medical Society of the
State of New York

Annual Dinner Dance

in honor of
Samuel M. Gelfand, M.D.
President

Thursday
April 21, 1988
Trianon Ballroom
The New York Hilton
53rd St. & Avenue of the Americas

Cocktail Hour
7 P.M.

Superlative
Dinner

Subscription $75.00 per person

Reservation Form

Make check payable to:
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Attn: Meeting Services
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Lake Success, New York 11042

Please make reservation(s)
for ______ at the Annual Dinner
Dance on April 21, 1988
Check enclosed for $________

PLEASE NOTE: Reservations and ticket sales will close at 12 Noon on Thursday April 21, one hour before the House Convenes.

Name____

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Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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(*continued from p 15A*)

of California, Box 0446, San Francisco, CA 94143. Tel: (415) 476-4194.

COLORADO

Mar 2–5. **The International Conference on Pulmonary Rehabilitation and Home Mechanical Ventilation.** Marriott Hotel, City Center, Denver. Contact: Webb-Waring Lung Institute, 4200 E 9th Ave, Box C-321, Denver, CO 80262. Tel: (303) 394-8231.

Mar 5–11. **Eighth Annual ENT Ski Conference.** Keystone Lodge. Contact: Marge Adey or Brenda Ram, Center for Continuing Education, University of Nebraska Medical Center, 42nd and Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

Mar 14–18. **Second Annual Update on Primary Care.** The Ranch, Steamboat Springs. Contact: Larry G. McLain, MD, Department of Pediatrics, Loyola University Medical Center, 2160 South First Ave, Maywood, IL 60153. Tel: (312) 531-3195.

DISTRICT OF COLUMBIA

Mar 27–31. **Thoracic Imaging '88.** 32 Cat 1 Credits. Willard Inter-Continental. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

FLORIDA

Mar 16–19. **Highlights in Women's Health Care—Cardiovascular Fitness, Breast Disease, Menopause.** 18 Cat 1 Credits. Orlando. Contact: Susan Larson, Director, Scott and White Office of Continuing Medical Education, Temple, TX 76508. Tel: (817) 774-4083.

ILLINOIS

Mar 5–12. **31st Tutorial on Clinical Cytology.** 80 Cat 1 Credits. Knickerbocker Chicago Hotel, Chicago. Contact: 31st Tutorial on Clinical Cytology, University of Chicago, 5841 South Maryland Ave, HM #449, Chicago, IL 60637. Tel: (312) 702-6569.

Mar 13–15. **The Second International Conference on Artificial Intelligence**

(*continued on p 21A*)

The World's Most Popular K*

Slow-K®
potassium chloride
slow-release tablets
8 mEq (600 mg)

It means "dependability" in almost any language

*Based on worldwide sales data on file, CIBA Pharmaceutical Company.
Capsule or tablet slow-release potassium chloride preparations should be reserved for patients who cannot tolerate, refuse to take, or have compliance problems with liquid or effervescent potassium preparations because of reports of intestinal and gastric ulceration and bleeding with slow-release KCl preparations.

Before prescribing, please consult Brief Prescribing Information on next page.

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The World's Most Popular K

For good reasons

- ☐ **It works**—a 12-year record of efficacy[1]
- ☐ **It's safe**—unsurpassed by any other KCl tablet or capsule[2]*
- ☐ **It's acceptable vs liquids**—greater palatability, fewer GI complaints, lower incidence of nausea[2]
- ☐ **It's comparable to 10 mEq**—in low-dosage supplementation[3]†
- ☐ **It's economical**—less expensive than all other leading KCl slow-release supplements on a per tablet cost to the patient[1]

Slow-K®
potassium chloride
slow-release tablets 8 mEq (600 mg)

For patients who can't or won't tolerate liquid KCl.

*The most common adverse reactions to potassium salts are gastrointestinal side effects.

†Pooled mean serum potassium following oral administration of 30 mEq K-Tab compared to 24 mEq Slow-K in diuretic-treated hypertensives (n = 20) over 8 weeks.

C I B A

References: 1. Data on file, CIBA Pharmaceutical Company. **2.** Skoutakis VA, Acchiardo SR, Wojciechowski NJ, et al: Liquid and solid potassium chloride: Bioavailability and safety *Pharmacotherapy* 1980;4(6):392-397. **3.** Skoutakis VA, Carter CA, Acchiardo SR. Therapeutic assessment of Slow-K and K-Tab potassium chloride formulations in hypertensive patients treated with thiazide diuretics *Drug Intell Clin Pharm* 1987;21:436-440.

Slow-K®
potassium chloride USP
Slow-Release Tablets
8 mEq (600 mg)

BRIEF SUMMARY (FOR FULL PRESCRIBING INFORMATION SEE PACKAGE INSERT)

INDICATIONS AND USAGE

BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS.

1. For therapeutic use in patients with hypokalemia with or without metabolic alkalosis; in digitalis intoxication and in patients with hypokalemic familial periodic paralysis.
2. For prevention of potassium depletion when the dietary intake of potassium is inadequate in the following conditions: patients receiving digitalis and diuretics for congestive heart failure; hepatic cirrhosis with ascites; states of aldosterone excess with normal renal function; potassium-losing nephropathy; and certain diarrheal states.
3. The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases supplementation with potassium salts may be indicated.

CONTRAINDICATIONS

Potassium supplements are contraindicated in patients with hyperkalemia, since a further increase in serum potassium concentration in such patients can produce cardiac arrest. Hyperkalemia may complicate any of the following conditions: chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e.g., spironolactone, triamterene) (see OVERDOSAGE).

All solid dosage forms of potassium supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract. In these instances, potassium supplementation should be with a liquid preparation. Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to an enlarged left atrium.

WARNINGS

Hyperkalemia (See OVERDOSAGE).
In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally. Potentially fatal hyperkalemia can develop rapidly and be asymptomatic.

The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment.

Interaction With Potassium-Sparing Diuretics
Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e.g., spironolactone or triamterene), since the simultaneous administration of these agents can produce severe hyperkalemia

Gastrointestinal Lesions
Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths. These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage, or perforation. Slow-K is a wax-matrix tablet formulated to provide a controlled rate of release of potassium chloride and thus to minimize the possibility of a high local concentration of potassium ion near the bowel wall. While the reported frequency of small-bowel lesions is much less with wax-matrix tablets (less than one per 100,000 patient-years) than with enteric-coated potassium chloride tablets (40-50 per 100,000 patient-years) cases associated with wax-matrix tablets have been reported both in foreign countries and in the United States. In addition, perhaps because the wax-matrix preparations are not enteric-coated and release potassium in the stomach, there have been reports of upper gastrointestinal bleeding associated with these products. The total number of gastrointestinal lesions remains approximately one per 100,000 patient-years. Slow-K should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs.

Metabolic Acidosis
Hypokalemia in patients with metabolic *acidosis* should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, or potassium acetate.

PRECAUTIONS

General:
The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis *per* se can produce hypokalemia in the absence of a deficit in total body potassium, while acute acidosis *per* se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium

Information for Patients
Physicians should consider reminding the patient of the following:

To take each dose without crushing, chewing, or sucking the tablets.

To take this medicine only as directed. This is especially important if the patient is also taking both diuretics and digitalis preparations

To check with the physician if there is trouble swallowing tablets or if the tablets seem to stick in the throat.

To check with the doctor at once if tarry stools or other evidence of gastrointestinal bleeding is noticed.

Laboratory Tests
Regular serum potassium determinations are recommended. In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis

Drug Interactions
Potassium-sparing diuretics: see WARNINGS

Carcinogenesis, Mutagenesis, Impairment of Fertility
Long-term carcinogenicity studies in animals have not been performed

Pregnancy Category C
Animal reproduction studies have not been conducted with Slow-K. It is also not known whether Slow-K can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Slow-K should be given to a pregnant woman only if clearly needed

Nursing Mothers
The normal potassium ion content of human milk is about 13 mEq/L. It is not known if Slow-K has an effect on this content. Caution should be exercised when Slow-K is administered to a nursing woman.

Pediatric Use
Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

One of the most severe adverse effects is hyperkalemia (see CONTRAINDICATIONS, WARNINGS, and OVERDOSAGE). There also have been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see CONTRAINDICATIONS and WARNINGS); other factors known to be associated with such conditions were present in many of these patients.

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea. These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose.

Skin rash has been reported rarely.

OVERDOSAGE

The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia. However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see CONTRAINDICATIONS and WARNINGS). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration (6.5-8.0 mEq/L) and characteristic electrocardiographic changes (peaking of T waves, loss of P wave, depression of S-T segment, and prolongation of the Q-T interval). Late manifestations include muscle paralysis and cardiovascular collapse from cardiac arrest (9-12 mEq/L).

Treatment measures for hyperkalemia include the following: (1) elimination of foods and medications containing potassium and of potassium-sparing diuretics; (2) intravenous administration of 300-500 ml/hr of 10% dextrose solution containing 10-20 units of insulin per 1,000 ml; (3) correction of acidosis, if present, with intravenous sodium bicarbonate; (4) use of exchange resins, hemodialysis, or peritoneal dialysis

In treating hyperkalemia in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity

DOSAGE AND ADMINISTRATION

The usual dietary intake of potassium by the average adult is 40-80 mEq per day. Potassium depletion sufficient to cause hypokalemia usually requires the loss of 200 or more mEq of potassium from the total body store. Dosage must be adjusted to the individual needs of each patient but is typically in the range of 20 mEq per day for the prevention of hypokalemia to 40-100 mEq or more per day for the treatment of potassium depletion. Large numbers of tablets should be given in divided doses.

Note: Slow-K slow-release tablets must be swallowed whole and never crushed, chewed, or sucked.

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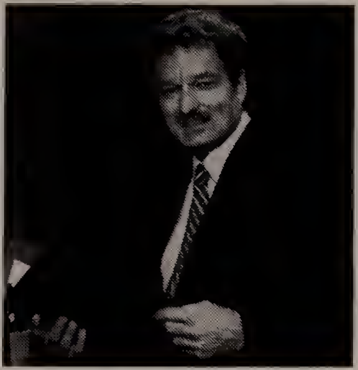

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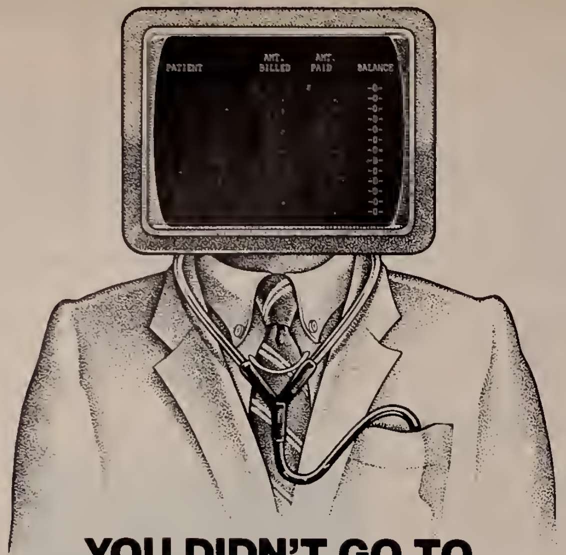

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Mar 2–6. **1988 Pan American Allergy Society Training Course and Seminar.** 35 Cat 1 Credits. Four Seasons Hotel, San Antonio. Contact: Betty Kahler, Executive Secretary, Pan American Allergy Society, 229 Parking Way, Lake Jackson, TX 77566. Tel: (409) 297-5636 or 297-4069.

Mar 7–9. **World Congress III on Cancers of the Skin.** 19 Cat 1 Credits. The Lincoln Hotel Post Oak, Houston. Contact: Conference Services—HMB Box 131, M. D. Anderson Hospital and Tumor Institute, 1515 Holcombe Blvd, Houston, TX 77030.

UTAH

Mar 30–Apr 1. **Tenth Annual Common Problems in Pediatrics.** Salt Lake City. Contact: Katharine C. Blosch, Preferred Meeting Management, Inc, 640 E Wilmington Ave, Salt Lake City, UT 84106. Tel: (801) 466-3500.

VERMONT

Mar 7–10. **New Developments in Clinical Neurology.** Trapp Family Lodge, Stowe. Contact: Office of CME, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

AROUND THE WORLD

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Mar 12–27. **Diagnostic Imaging Down Under.** Contact: Medical Seminars International, 21915 Roscoe Blvd, Suite 222, Canoga Park, CA 91304. Tel: (818) 719-7380.

CANADA

Mar 17–18. **Assessment of Clinical Competence in Specialty Medicine.** Toronto Hilton Harbour Castle Hotel, Toronto. Contact: American Board Medical Specialties, One Rotary Center, Suite 805, Evanston, IL 60201.

Mar 24–26. **A New Look at Psychosomatic Medicine.** Four Seasons Hotel, Toronto. Contact: American Psychosomatic Society, 6728 Old McLean Village Drive, McLean, VA 22101. Tel: (703) 556-9222.

DOMINICAN REPUBLIC

Mar 5–12. **Functional Sterotactic Neurosurgery.** 21 Cat 1 Credits. Casa del Campo. Contact: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251. □

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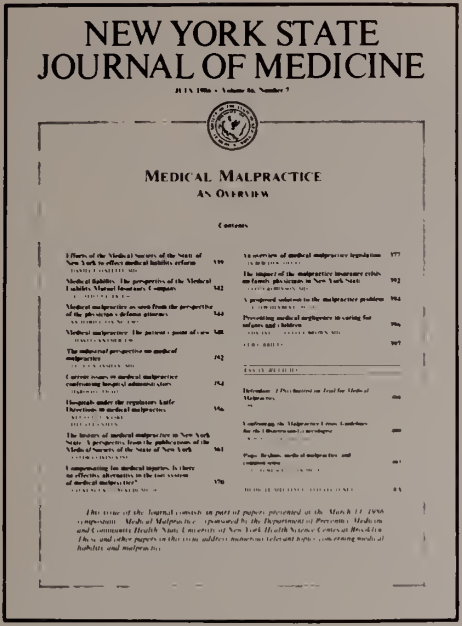
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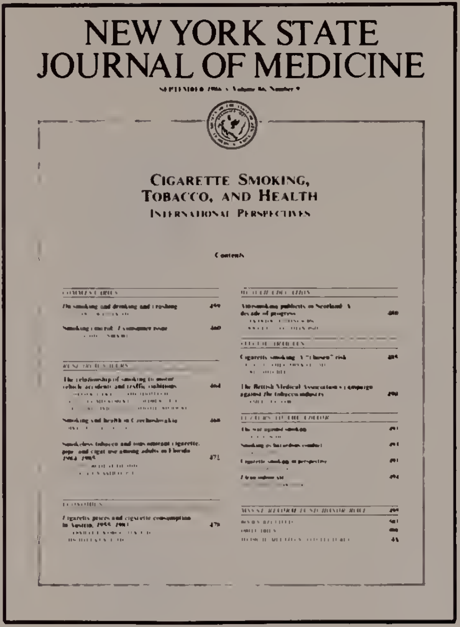
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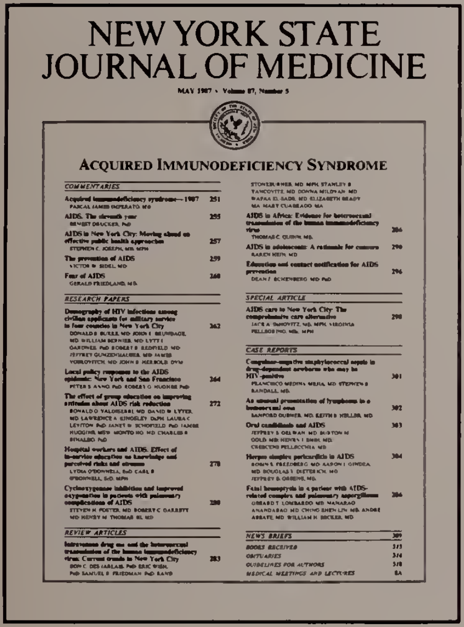
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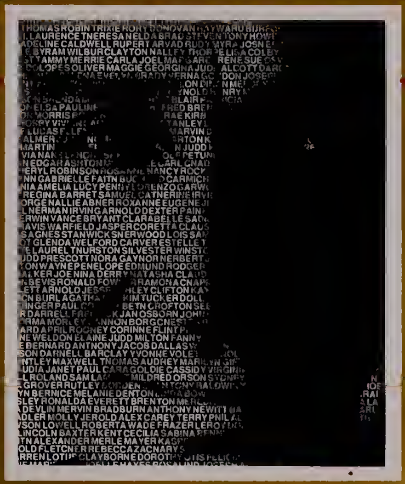

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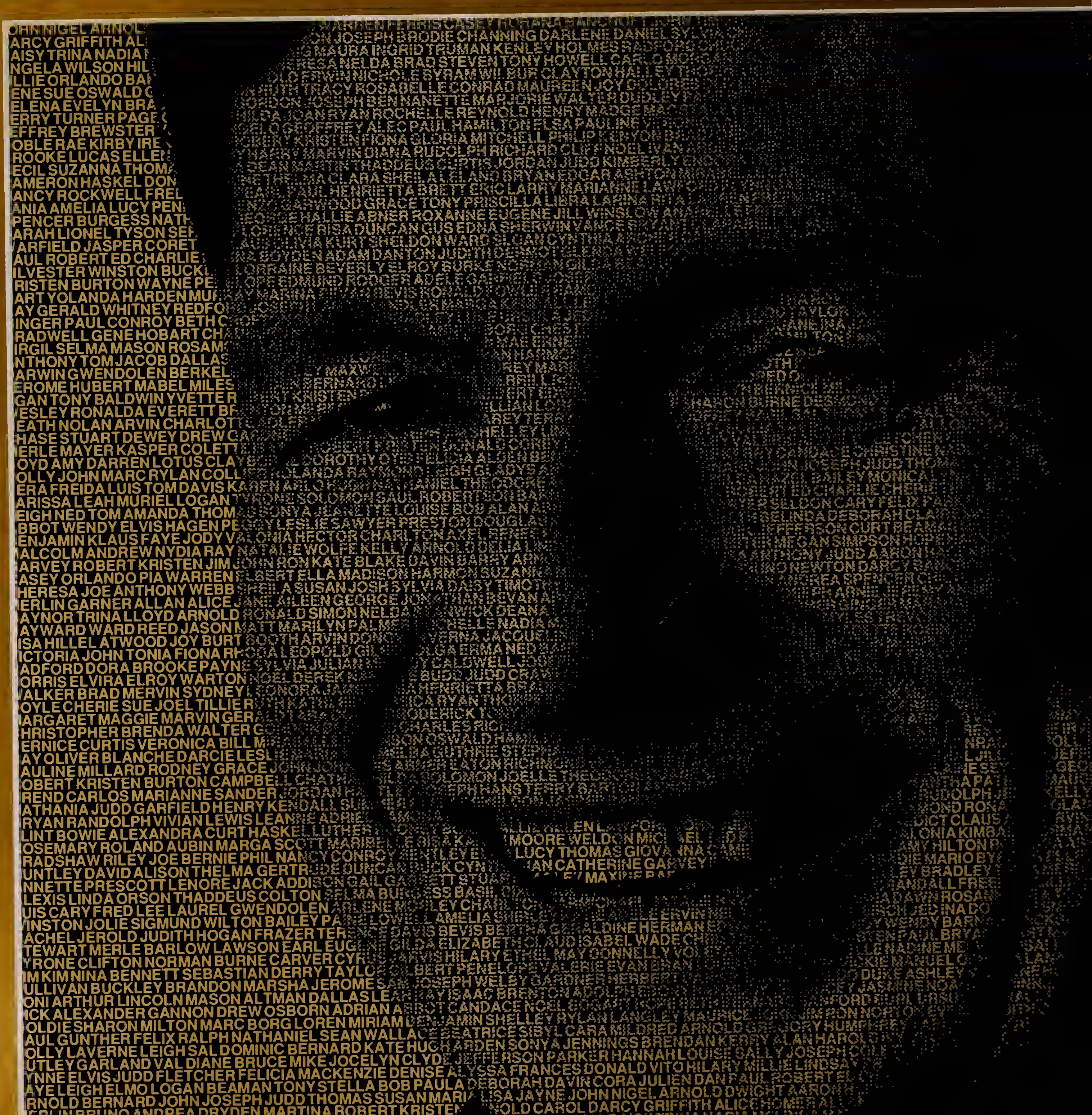
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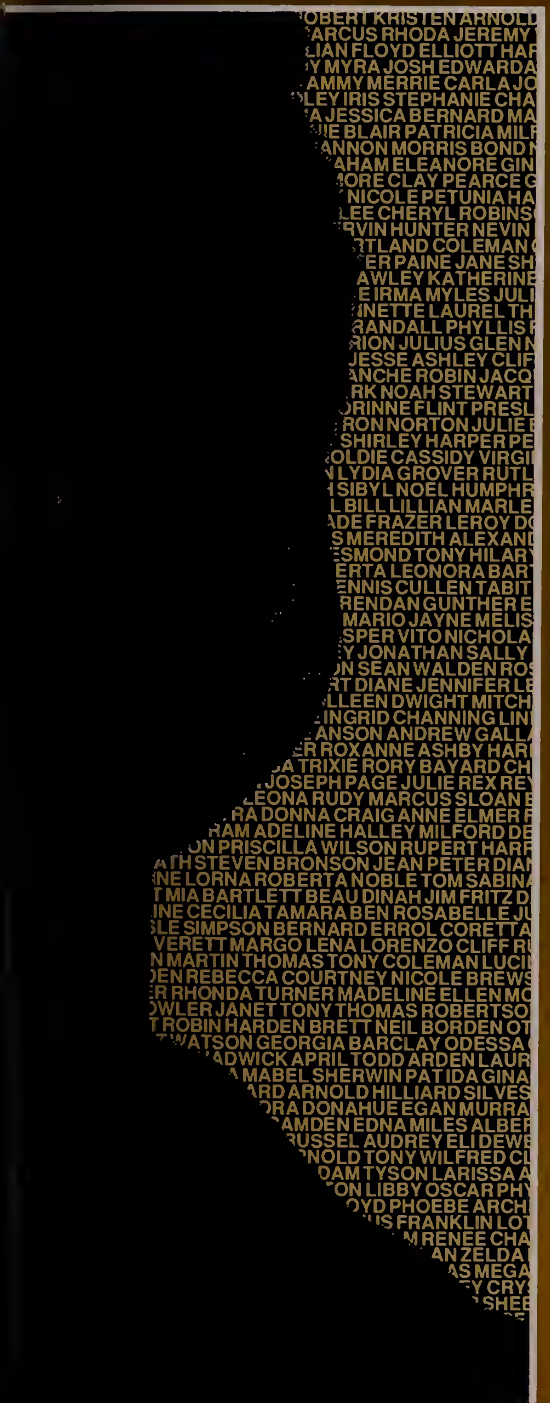

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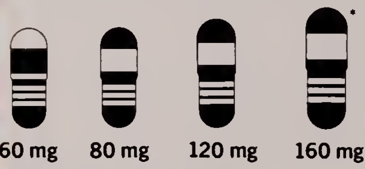

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DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride. INDERAL LA is available as 60 mg, 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure, and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first-degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected. Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL (propranolol HCl) is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Caution should be exercised when patients receiving a beta blocker are administered a calcium-channel-blocking drug, especially intravenous verapamil, for both agents may depress myocardial contractility or atrioventricular conduction. On rare occasions, the concomitant intravenous use of a beta blocker and verapamil has resulted in serious adverse reactions, especially in patients with severe cardiomyopathy, congestive heart failure, or recent myocardial infarction.

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol.

Ethanol slows the rate of absorption of propranolol.

Phenytoin, phenobarbitone, and *rifampin* accelerate propranolol clearance.

Chlorpromazine, when used concomitantly with propranolol, results in increased plasma levels of both drugs.

Antipyrine and *lidocaine* have reduced clearance when used concomitantly with propranolol.

Thyroxine may result in a lower than expected T_3 concentration when used concomitantly with propranolol.

Cimetidine decreases the hepatic metabolism of propranolol, delaying elimination and increasing blood levels.

Theophylline clearance is reduced when used concomitantly with propranolol.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS: INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type.

Central Nervous System: Light-headedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; vivid dreams; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. For immediate formulations, fatigue, lethargy, and vivid dreams appear dose related.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL Tablets to INDERAL LA Capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg-for-mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary, especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—*Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three- to seven-day intervals until optimal response is obtained. Although individual patients may respond at any dosage level, the average optimal dosage appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE—*Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimal migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximal dose, INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of these capsules is a registered trademark of Ayerst Laboratories.

Reference:

1. Data on file, Ayerst Laboratories.

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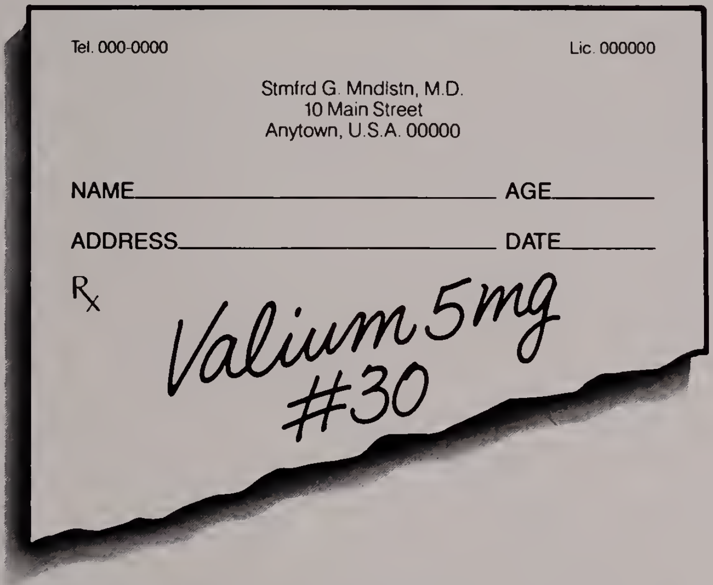

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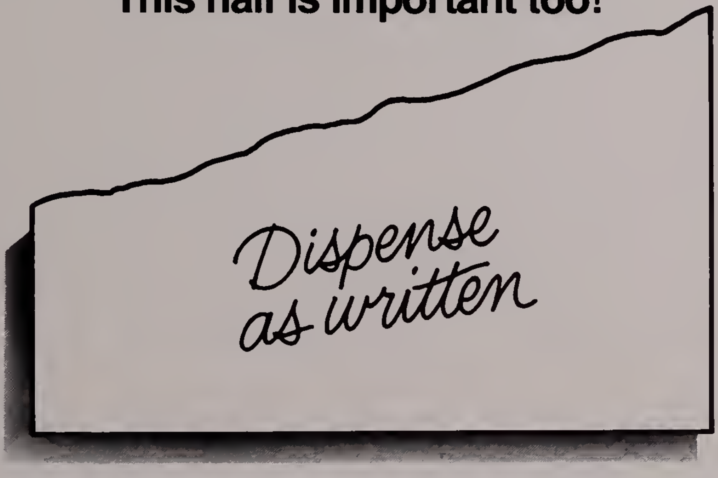

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	CONSTIPATION	RESPIRATORY DEPRESSION	SEDATION	EMESIS	PHYSICAL DEPENDENCE
HYDROCODONE		X			X
CODEINE	X	X	X	X	X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975; 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

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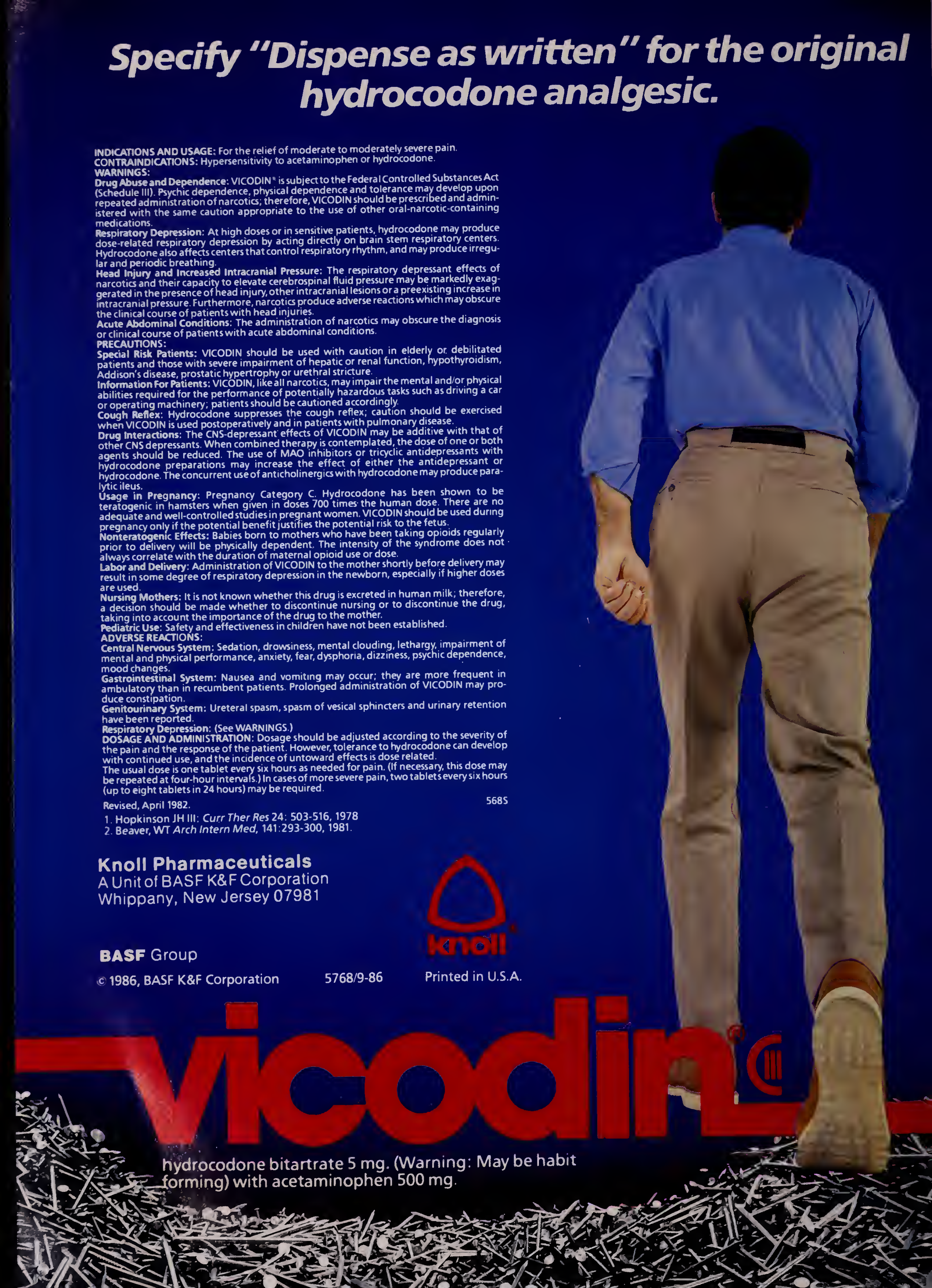

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INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.
WARNINGS:
Drug Abuse and Dependence: VICODIN® is subject to the Federal Controlled Substances Act (Schedule III). Psychic dependence, physical dependence and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN should be prescribed and administered with the same caution appropriate to the use of other oral-narcotic-containing medications.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers. Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing.
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS:
Special Risk Patients: VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Information For Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
Cough Reflex: Hydrocodone suppresses the cough reflex; caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants. When combined therapy is contemplated, the dose of one or both agents should be reduced. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic Effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
Labor and Delivery: Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
Nursing Mothers: It is not known whether this drug is excreted in human milk; therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS:
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients. Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.
Respiratory Depression: (See WARNINGS.)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. However, tolerance to hydrocodone can develop with continued use, and the incidence of untoward effects is dose related.
The usual dose is one tablet every six hours as needed for pain. (If necessary, this dose may be repeated at four-hour intervals.) In cases of more severe pain, two tablets every six hours (up to eight tablets in 24 hours) may be required.

Revised, April 1982. 568S

1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med*, 141:293-300, 1981.

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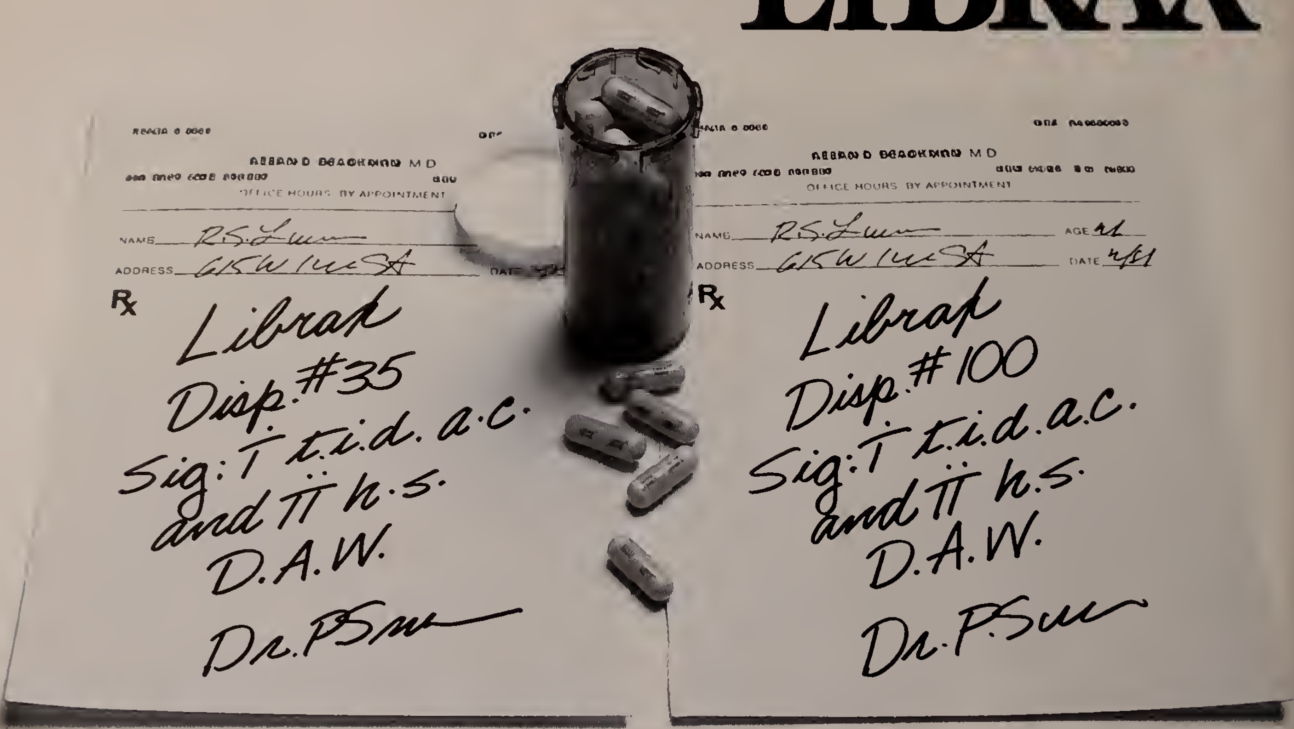

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Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.
Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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In irritable bowel syndrome* anxiety can aggravate intestinal symptoms, which may further intensify anxiety — a distressing cycle of brain/bowel conflict. Librax intervenes with two well-known compounds. The Librium® (chlordiazepoxide HCl/Roche) component safely relieves anxiety. And Quarzan® (clidinium bromide/Roche) provides antisecretory and antispasmodic action to relieve discomfort associated with intestinal hypermotility.

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*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of peptic ulcer and the irritable bowel syndrome.

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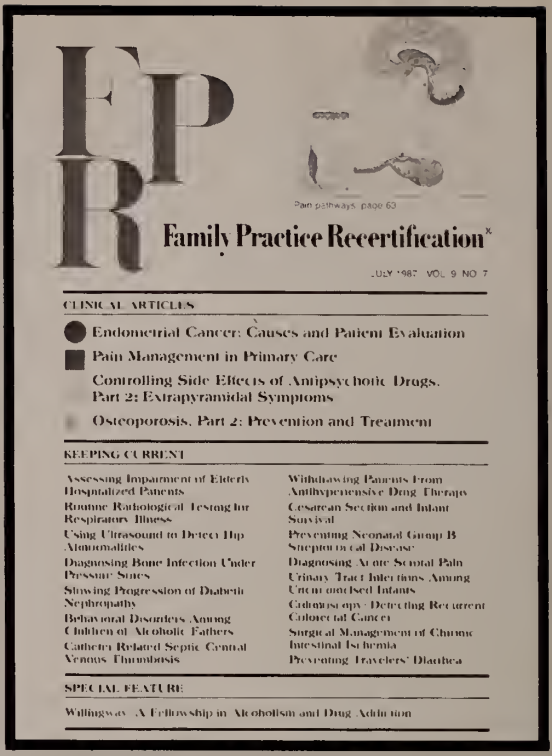

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Pain pathways, page 63

Family Practice Recertification

JULY 1987 VOL 9 NO 7

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● Endometrial Cancer: Causes and Patient Evaluation

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The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

APRIL 1988

AROUND THE STATE

MANHATTAN

Apr 5–9. **#610 Seminar in Neuroradiology.** 27 Cat 1 Credits. The Grand Hyatt New York. *Apr 18–23.* **Comprehensive Review of Physical Medicine and Rehabilitation.** 55 Cat 1 Credits. NYU Medical Center. *Apr 21–23.* **Menopausal Estrogens.** 12 Cat 1 Credits. NYU Medical Center. *Apr 21–23.* **Advances in Dermatology.** 17 Cat 1 Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Apr 8. **Consensus Conference on the Management of Multiple Sclerosis.** Albert Einstein College of Medicine of Yeshiva University. Contact: Labe Scheinberg, MD, Albert Einstein College of Medicine, Multiple Sclerosis Research and Rehabilitation Training Center, 1300 Morris Park Ave, Bronx, NY 10461.

Apr 9. **Clinical Strategies for Remodeling the Atherosclerotic Plaque and Retarding Atherogenesis.** Hotel Pierre. Contact: New York Cardiological Society, 84 Grove St, Suite 2, New York, NY 10014.

Apr 23. **Corneal and External Disease Highlights 1988.** *Apr 28–30.* **1988 Sigmund Schutz Professorship.** Manhattan Eye, Ear and Throat Hospital. Contact: Martha Klapp, Manhattan, Eye, Ear and Throat Hospital, 210 East 64th St, New York, NY 10021. Tel: (212) 605-3762.

Apr 22–24. **Third International Symposium on Eating Disorders (1988).** 15 Cat 1 Credits. Grand Hyatt Hotel. Contact: Albert Einstein College of Medicine/Montefiore Medical Center, Office of Continuing Medical Education, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674.

Apr 27. **Forensic Sculpture: A Technique for Identifying the Missing or Murdered.** Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 130 Gold Pl, Malverne, NY 11565. Tel: (516) 887-4691.

Apr 27–30. **The Fourth Annual International Conference on Computerization of Medical Records—Creating Patient Information Systems.** New York Penta Hotel. Contact: Registrar, Institute for Medical Records, 121 Mt Vernon St, Boston, MA 02108.

ALBANY

Apr 9. **Orthopedic Surgery Teaching Day.** Albany Medical College. *Apr 14.* **Endocrinology Teaching Day.** Albany Medical College. Contact: Albany Medical College, Office of Continuing Medical Education, 47 New Scotland

(*continued on p 10A*)

Specialized ulcer therapy

When advancing age signals reduced acid secretion

If your duodenal ulcer patient is over 55, decreased mucosal resistance is more likely to cause an ulcer than hypersecretion of acid-pepsin.[1] A tendency toward lower acid secretion with advancing age has been shown.[2,3]

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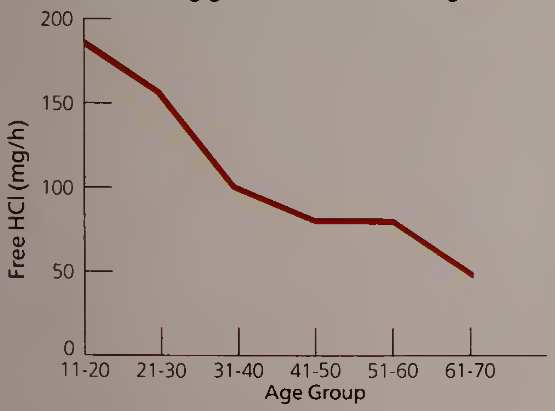

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BRIEF SUMMARY

CONTRAINDICATIONS

There are no known contraindications to the use of sucralfate

PRECAUTIONS

Duodenal ulcer is a chronic, recurrent disease While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that the simultaneous administration of CARAFATE with tetracycline, phenytoin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. The clinical significance of these animal studies is yet to be defined

Carcinogenesis, Mutagenesis, Impairment of Fertility: No evidence of drug-related tumorigenicity was found in chronic oral toxicity studies of 24 months' duration conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose) A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment Mutagenicity studies have not been conducted

Pregnancy: Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS

Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug In studies involving over 2,500 patients, adverse effects were reported in 121 (4.7%). Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

DOSAGE AND ADMINISTRATION

The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED

CARAFATE (sucralfate) 1-gm pink tablets are supplied in bottles of 100 and in Unit Dose Identification Paks of 100. The tablets are embossed with MARION/1712 Issued 3/84

References:

1. Grossman MI: *Scand J Gastroenterol* 58 (suppl 15):7-16, 1980
2. Marks IN, in Hellemans J, Vantrappen G (eds): *Gastrointestinal Tract Disorders in the Elderly.* Edinburgh, Churchill Livingstone, 70-81, 1984
3. Krentz K, Jablonowski H, in Hellemans J, Vantrappen G (eds): *Gastrointestinal Tract Disorders in the Elderly.* Edinburgh, Churchill Livingstone, 62-69, 1984

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Before prescribing, see complete prescribing information in SK&F LAB CO. literature or PDR. The following is a brief summary.

Contraindications: There are no known contraindications to the use of 'Tagamet'.

Precautions: While a weak antiandrogenic effect has been demonstrated in animals, 'Tagamet' has been shown to have no effect on spermatogenesis, sperm count, motility, morphology or in vitro fertilizing capacity in humans.

In a 24-month toxicity study in rats at dose levels approximately 9 to 56 times the recommended human dose, benign Leydig cell tumors were seen. These were common in both the treated and control groups, and the incidence became significantly higher only in the aged rats receiving 'Tagamet'.

Rare instances of cardiac arrhythmias and hypotension have been reported following the rapid administration of 'Tagamet' HCl (brand of cimetidine hydrochloride) injection by intravenous bolus.

Symptomatic response to 'Tagamet' therapy does not preclude the presence of a gastric malignancy. There have been rare reports of transient healing of gastric ulcers despite subsequently documented malignancy.

Reversible confusional states have been reported on occasion, predominantly in severely ill patients.

'Tagamet' has been reported to reduce the hepatic metabolism of warfarin-type anticoagulants, phenytoin, propranolol, chlordiazepoxide, diazepam, lidocaine, theophylline and metronidazole. Clinically significant effects have been reported with the warfarin anticoagulants; therefore, close monitoring of prothrombin time is recommended, and adjustment of the anticoagulant dose may be necessary when 'Tagamet' is administered concomitantly. Interaction with phenytoin, lidocaine and theophylline has also been reported to produce adverse clinical effects.

However, a crossover study in healthy subjects receiving either 'Tagamet' 300 mg. q.i.d. or 800 mg. h.s. concomitantly with a 300 mg. b.i.d. dosage of theophylline (Theo-Dur®, Key Pharmaceuticals, Inc.), demonstrated less alteration in steady-state theophylline peak serum levels with the 800 mg. h.s. regimen, particularly in subjects aged 54 years and older. Data beyond ten days are not available. (Note: All patients receiving theophylline should be monitored appropriately, regardless of concomitant drug therapy.)

Lack of experience to date precludes recommending 'Tagamet' for use in pregnant patients, women of childbearing potential, nursing mothers or children under 16 unless anticipated benefits outweigh potential risks; generally, nursing should not be undertaken in patients taking the drug since cimetidine is secreted in human milk.

Adverse Reactions: Diarrhea, dizziness, somnolence, headache, rash. Reversible arthralgia, myalgia and exacerbation of joint symptoms in patients with preexisting arthritis have been reported. Reversible confusional states (e.g., mental confusion, agitation, psychosis, depression, anxiety, hallucinations, disorientation), predominantly in severely ill patients, have been reported. Gynecomastia and reversible impotence in patients with pathological hypersecretory disorders receiving 'Tagamet', particularly in high doses, for at least 12 months, have been reported. Reversible alopecia has been reported very rarely. Decreased white blood cell counts in 'Tagamet'-treated patients (approximately 1 per 100,000 patients), including agranulocytosis (approximately 3 per million patients), have been reported, including a few reports of recurrence on rechallenge. Most of these reports were in patients who had serious concomitant illnesses and received drugs and/or treatment known to produce neutropenia. Thrombocytopenia (approximately 3 per million patients) and a few cases of aplastic anemia have also been reported. Increased serum transaminase and creatinine, as well as rare cases of fever, interstitial nephritis, urinary retention, pancreatitis and allergic reactions, including hypersensitivity vasculitis, have been reported. Reversible adverse hepatic effects, cholestatic or mixed cholestatic-hepatocellular in nature, have been reported rarely. Because of the predominance of cholestatic features, severe parenchymal injury is considered highly unlikely. A single case of biopsy-proven periportal hepatic fibrosis in a patient receiving 'Tagamet' has been reported.

How Supplied: Tablets: 200 mg. tablets in bottles of 100; 300 mg. tablets in bottles of 100 and Single Unit Packages of 100 (intended for institutional use only); 400 mg. tablets in bottles of 60 and Single Unit Packages of 100 (intended for institutional use only), and 800 mg. Tiltab® tablets in bottles of 30 and Single Unit Packages of 100 (intended for institutional use only).

Liquid: 300 mg./5 ml., in 8 fl. oz. (237 ml.) amber glass bottles and in single-dose units (300 mg./5 ml.), in packages of 10 (intended for institutional use only).

Injection:

Vials: 300 mg./2 ml. in single-dose vials, in packages of 10 and 30, and in 8 ml. multiple-dose vials, in packages of 10 and 25.

Prefilled Syringes: 300 mg./2 ml. in single-dose prefilled disposable syringes.

Plastic Containers: 300 mg. in 50 ml. of 0.9% Sodium Chloride in single-dose plastic containers, in packages of 4 units. No preservative has been added.

ADD-Vantage®* Vials: 300 mg./2 ml. in single-dose ADD-Vantage® Vials, in packages of 25.

Exposure of the premixed product to excessive heat should be avoided. It is recommended the product be stored at controlled room temperature. Brief exposure up to 40°C does not adversely affect the premixed product.

'Tagamet' HCl (brand of cimetidine hydrochloride) Injection premixed in single-dose plastic containers is manufactured for SK&F Lab Co. by Travenol Laboratories, Inc., Deerfield, IL 60015.

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(*continued from p 6A*)

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BUFFALO

Apr 28–29. **International Symposium: The Expanding Role of Folates and Fluoropyrimidines in Cancer Chemotherapy.** Roswell Park Memorial Institute. Contact: Gayle Bersani, RN, Coordinator of Continuing Education Programs, Education Department, Roswell Park Memorial Institute, 666 Elm St, Buffalo, NY 14263. Tel: (716) 845-2339.

QUEENS

Apr 20. **Legal and Medical Issues of Informed Consent.** 2 Cat 1 Credits. Academy of Medicine of Queens County. Contact: Mary Nee, Secretary to the Continuing Medical Education Committee, Academy of Medicine of Queens County, 112-25 Queens Blvd, Forest Hills, NY 11375. Tel: (718) 268-7300.

AROUND THE NATION

ARIZONA

Apr 24–26. **Infectious Diseases for Pulmonary and Critical Care Specialists.** 12 Cat 1 Credits. Scottsdale. Contact: Continuing S/P, University of California, Box 0446, San Francisco, CA 94143. Tel: (415) 476-4194.

CALIFORNIA

Apr 10–15. **Advanced Methods in Pharmacokinetics and Pharmacodynamics.** San Francisco. *Apr 14–16.* **Postgraduate Course in General Surgery.** San Francisco. *Apr 15–17.* **Coronary Artery Disease: A Commemorative Symposium—75 Years Since Anitschkow and Herrick.** San Francisco. *Apr 28–30.* **21st Annual Advances and Controversies in Clinical Pediatrics.** 16½ Cat 1 Credits. Mark Hopkins Hotel. Contact: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Apr 10–17. **Eighth Annual San Diego Residents' Radiology Review Course.** Town and Country Hotel, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0133. Tel: (404) 641-9773.

Apr 23–24. **The Cutting Edge 1988, Innovations in Psychotherapy: Helping Individuals and Couples to Change.** 13 Cat 1 Credits. Hotel Del Coronado, San Diego. Contact: Office of Continuing Medical Education, UC San Diego School of Medicine, La Jolla, CA 92093. Tel: (619) 534-3940.

DISTRICT OF COLUMBIA

Apr 22–24. **National Internal Medicine Leadership Conference.** Washington. Contact: ASIM, 1101 Vermont Ave, NW, Suite 500, Washington, DC 20005.

FLORIDA

Apr 7–11. **Geriatrics Board Review.** Lake Buena Vista. Contact: American

(*continued on p 19A*)

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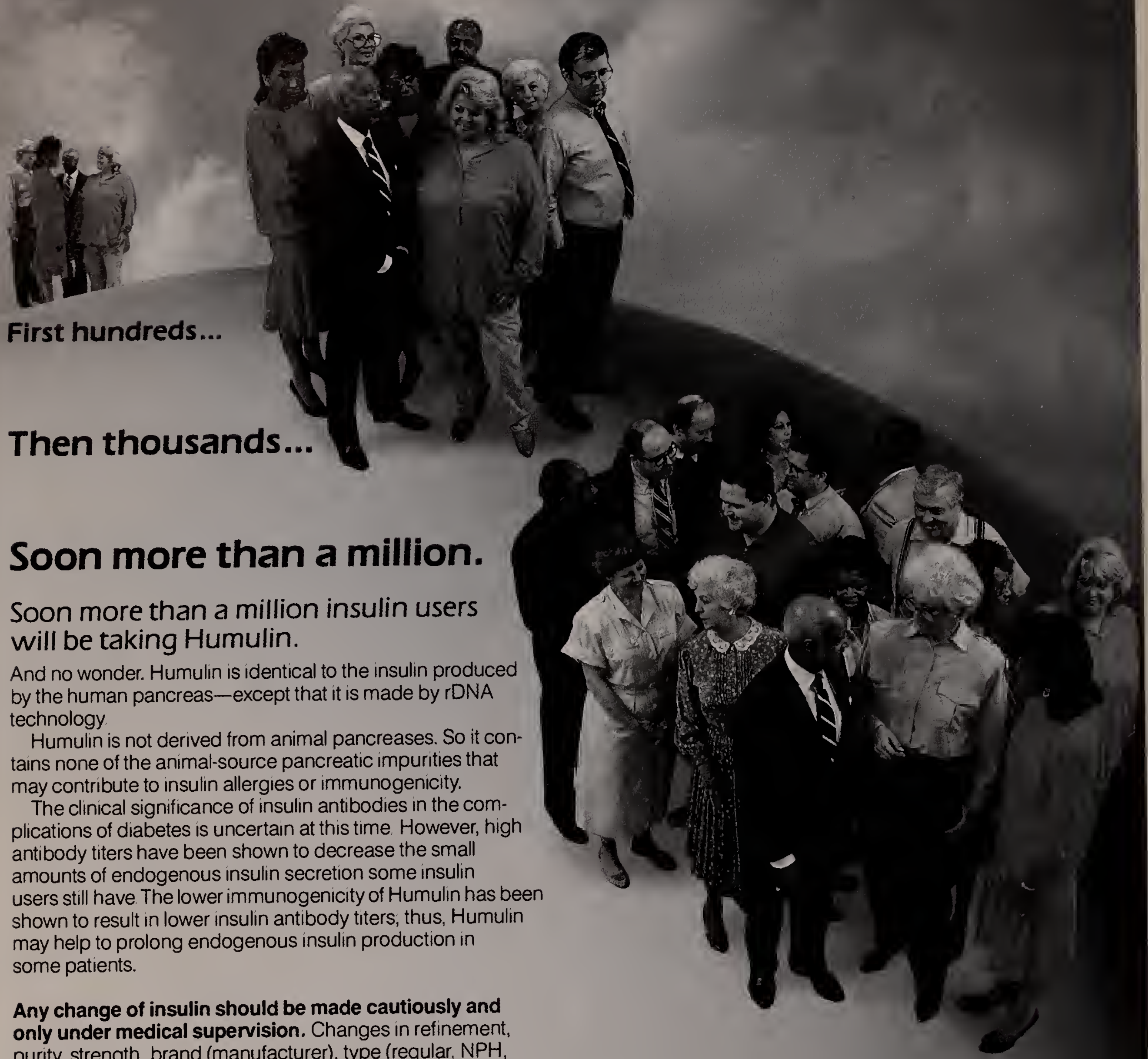

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COMMENTARIES

Physicians and the dispensing of drugs for profit: A need for responsible legislation

Physicians are increasingly becoming commercial purveyors of drugs for profit. Exact figures on the number of physicians who purchase prescription doses from drug wholesalers and then sell them to patients at a marked-up price are not available. However, an estimated 5% of practitioners have already commenced dispensing drugs in this manner.[1]

Should doctors be selling drugs to their patients at a marked-up price, as well as prescribing them? What are the attendant questions of medical ethics? Is there a need for appropriate legislation at the state as well as federal level?

Legislation pertaining to doctors and the dispensing of drugs for profit has been proposed at the federal level. A bill proposed by Representative Wyden (H.R. 2168) would limit the dispensing of certain drugs by medical practitioners.[2] It bars practitioners from dispensing for profit drugs that are to be orally administered and which are not vaccines.

H.R. 2168 spells out certain exceptions to the general rule barring physicians from dispensing drugs for profit.[2] The general prohibition does not apply to the dispensing of drugs for emergency medical reasons, to patients who may have "substantial difficulty" in obtaining drugs from a pharmacy, or to practitioners with offices located in a "rural" area.

It is important to note that the proposed law does not ban physicians from dispensing drugs to patients; it simply states that practitioners cannot dispense drugs *for profit*. Thus, the differential issue of giving penicillin by injection for an appropriate fee as opposed to dispensing it as a drug in tablet form does not arise. H.R. 2168 allows physicians to do both. However, it does not permit physicians to make a profit on medications dispensed. So the ethical question presented is whether physicians may make a profit on drugs that they prescribe or inject, not whether they may give an injection or write a prescription and charge for these services.[3]

Whenever federal legislation seeks to thread itself into the fabric of medical practice, there are inevitably critics who complain that the federal government should "get off the backs" of doctors. The matter of regulating the sale of drugs by physicians for profit may more appropriately be deemed to be within the area of state legislation. The proposed federal legislation, as a matter of fact, does not necessarily interfere with state legislative jurisdiction. The language of H.R. 2168 specifically states that it does not preempt or supersede any state law or regulation that regulates the terms and conditions of, and the charges which may be made for, the dispensing of drugs by licensed practitioners.[2] This statutory proviso may be quite propitious. H.R. 2168 basically focuses on the economic aspect of drug dispensing. This leaves a vacuum with respect to drug distribution in particular states regulated by proper safeguards and audits. Well-crafted legislation at the state level may potentially fill such a vacuum.

New York State has recently considered legislation concerning drug dispensing by prescribers. Assembly Bill 7973-B, for instance, would generally bar prescribers from dispensing more than a 72-hour supply of drugs.[4] During the 1987 regular session, this legislation was approved by the New York State Assembly. However, it failed to gain support in the state senate prior to the legislative recess.

The medical community in New York State would be wise to carefully debate the medical ethics issues raised by the question of physician drug dispensing for profit. There is similarly a need to discuss possible regulatory options affecting drug dispensing by medical practitioners. The information gleaned from such a debate may assist state legislators in carving out responsible legislation that fairly represents the interests and concerns of patients as well as health care providers. Careful discussion may further assist the medical community in New York in deciding what support should be given to federal legislation affecting the dispensing of drugs for profit by practitioners.

A major problem arising from physician dispensing of drugs for profit involves a potential conflict of interest.[1] Pharmacies generally maintain thousands of drug products, dosage strengths, and dosage forms in their inventories. In contrast, the physician selling drugs for profit will usually have only a very limited supply of medications. There is a risk that physicians dispensing drugs for profit may, consciously or unconsciously, overprescribe certain drugs maintained in their limited inventories and thus narrow the drug options of the patient. This of course creates an unethical conflict of interest between the financial interests of the physician and the best medical interests of the patient, which require that physicians should only prescribe a drug based on a reasonable expectation of its effectiveness for a particular patient.

Physicians must not, in effect, function as businesspersons with an inventory of drugs in stock that they earnestly want to sell to patients at a profit, regardless of whether the drugs may be best for the individual patient. For physicians vending drugs to make medical decisions based solely on their own financial benefit is improper. The overriding principle is that potential conflicts between the financial interests of the physician and the medical interests of the patient must always be resolved in favor of the patient.[5]

Proponents of physician drug dispensing for profit may claim that buying drugs from physicians is more convenient and cheaper for the patient.[6] In truth, it is probably neither. Obtaining drugs from a physician may save a trip to the pharmacist in some instances. However, physicians, particularly in comparison with community pharmacies, generally have more limited office hours. Whereas many community pharmacies are open for business seven days a week, most physicians have hours limited to daytime, and their offices may be closed at night, on the weekend, and during holidays. This may be a problem for patients in need of emergency services or suffering from illnesses requiring frequent follow-up prescription services.

Even if the dispensing of drugs by doctors in a particular situation is convenient for the patient, there is no reason why the drug cannot be dispensed at cost.[3] Furthermore, the issue of high prices for prescription drugs will not necessarily be ameliorated by physician dispensing inasmuch as the bulk of the price paid by the patient for the drug represents a cost charged by the manufacturer and probably is outside the control of either the pharmacist or the physician.

Another issue that must be resolved is whether or not physician drug dispensing for profit and the resulting competition with the pharmaceutical profession will result in improved quality of service. It is far from clear whether it actually would. Is it competitive for a patient to be advised by a physician to "stop at the front desk for your medicine"? It should be borne in mind, too, that patients may then be reluctant to obtain their medications elsewhere because of a fear that this action will disturb their relationship with the physician.

Another important consideration is the checks and balances system that currently exists between the medical and pharmacy professions, and which may help safeguard patients from prescribing errors committed by physicians. Patients receiving drugs are entitled to comprehensive pharmaceutical services, and pharmacists generally provide them. The pharmacist may supply drugs; review prescriptions for the suitability of drugs, dosages prescribed, duration of therapy; provide patient counseling; look for potentially adverse allergic reactions and drug interactions; and look for possible duplication of therapy.[7] The pharmacist not only provides valuable services for the patient, but may additionally be an ally of the physician by protecting physicians from potential medical malpractice claims arising from drug-related errors.[7]

Physicians are specifically trained as diagnosticians. (In fact, in some instances, physician dispensing may in truth be done by unsupervised nonphysicians employed at the physician's office.) Pharmacists, however, are highly and specifically trained in appropriate drug use. Physician dispensing of drugs for profit may imperil a long-standing, valuable relationship between the medical and pharmacy professions to the detriment of patients. In sum, it is this author's view that physicians may be well advised not to get into the business of dispensing drugs for profit.

Physicians in New York State should also carefully ponder possible regulatory options concerning drug dispensing by practitioners. A plethora of options and attendant questions arises. Conditions under which physicians may dispense drugs for profit should be spelled out specifically, with limitations clearly identified. Continuing education requirements must be set to keep physicians who dispense drugs abreast of pharmacy-related developments. Close consideration should be given to required personal physician supervision of office personnel when drugs are dispensed. Listings of prescription drug charges separate from other medical charges on the patient's bill should also be mandated. These are merely selected areas of regulatory concern. There are obviously many others.

The issue of drug dispensing for profit by physicians is rapidly developing in the legislative arena. Because of the seriousness of the matters involved, it is decidedly not a proper time for physicians to imitate the proverbial ostrich with its head in the sand. Instead, professional medical groups in New York State, and physicians in general, must debate the issues involved with care and attention, and then make appropriate recommendations to state and federal legislators.

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The real costs of generic substitution

In 1984, Congress enacted the Drug Price Competition and Patent Term Restoration Act, allowing for an expedited Food and Drug Administration (FDA) approval process for generic drugs.[1] Since the law was passed, about 1,000 generic drugs have received such approval.

This burgeoning of generic drugs, coupled with the rapid growth of managed health care systems, has resulted in a profound shift in prescribing and dispensing practices. According to a recent survey, generic drug sales will boom in the next decade because "everything that is going on in the distribution channel basically is leading to generic substitution and to systems that enforce generic substitution."[2] Economic pressures are the sole impetus behind generic substitution. Thus, the generally lower cost of bulk quantities of generic drugs has favored their inclusion in the formularies of hospitals, government programs, health care systems, mail order pharmacy services, and physician in-office dispensing. At the community pharmacy level, cost cutting (with resulting profit enhancement) is a powerful incentive to substitute generics. Finally, patients themselves frequently pressure both physicians and pharmacists to prescribe and dispense generically. This pressure stems from a widespread perception that generics cost less but are "just as good" as brand name products.

Reinforcing the economic pressures toward generic substitution are the statutory requirements in each of the states. All 50 states now permit some form of substitution of drug products, either on a mandatory or a discretionary basis. In New York State, a pharmacist must, with a few exceptions, substitute a lower cost generic drug for the prescribed brand name product unless the prescriber writes "DAW" (dispense as written) in the box at the bottom of the prescription blank. Notwithstanding these statutory requirements, however, there is evidence of widespread disregard for and noncompliance with drug substitution laws. In addition, patients have been unable to realize consistently the full cost savings anticipated and promised by proponents of these generic substitution laws.[3,4]

One of the fundamental problems experienced in New York State by both professionals and nonprofessionals (ie, nonpharmacist procurement officials) has been their inability to comply with the mandate to substitute less expensive generic drugs for brand name drugs. No guidance is provided for determining whether the multitude of multisource drugs available meet the bioequivalency criteria set out in the state law as a prerequisite for substitution. Accordingly, New York has utilized as the standard for its state drug formulary the FDA publication, *Approved Prescription Drug Products with Therapeutic Equivalence Evaluations*.[5] This directory, commonly called the "Orange Book," contains a list of brand name drugs and their generic equivalents along with other pertinent drug data, and rates the generics on their bioequivalency. For most orally administered drugs, the FDA requires testing to show with 90% certainty that the true mean bioavailabilities (blood levels) of an innovator drug and a generic version of that drug do not differ by more than ±20%. Alternatively, the FDA may invoke the "75-75 rule," which requires the bioavailability of the generic drug product to be between 75% and 125% of that determined for the innovator drug product in no less than 75% of the test subjects.[6]

Many physicians, pharmacists, and manufacturers of pioneer drugs have expressed concern about the foregoing methodology and process for FDA approval of "bioequivalent" drug products and their listing in the Orange Book. Confusion, controversy, and differences of opinion still exist as to the interchangeability of various versions of the same drug product. This is demonstrated by the fact that the generic substitution committees in the states of Florida, Illinois, New Jersey, and Rhode Island do not accept the findings of the Orange Book as evidence of bioequivalence.

It can be logically argued that, if generic drugs are less costly than the innovator brand name product (and they usually are), then the only deterrent to generic substitution should relate to differences in quality or therapeutic interchangeability of the two products. For many classes of drugs, especially those with a wide therapeutic margin, few if any such differences have been documented. Generic versions of such drugs can, in all likelihood, be substituted without adverse effects for the patient and with cost savings for all concerned. However, such assurance cannot be given for other classes of drugs where evidence of interchangeability may be lacking or where there may be documented evidence of bioinequivalence. For these agents, product substitution can have serious adverse consequences for patients. Among the drugs in this category are amitriptyline, chlorpromazine, digoxin, furosemide, phenytoin, theophylline, thioridazine, and warfarin.[7] Theophylline will be used here to illustrate the hazards of indiscriminate switching among available products.

Theophylline has, for many years, been a cornerstone in the treatment regimen for chronic asthma. Because of its narrow therapeutic margin, and the enhanced likelihood of compliance errors when four or more doses per day are required, theophylline is utilized today most often as a slow release (SR) formulation. Approximately 30 such formulations are available in the United States, both by brand and generic names. Careful titration of dosage with periodic determinations of serum theophylline levels is considered essential in order to ameliorate asthmatic symptoms and avoid drug toxicity. For most patients, the therapeutic range for serum levels is 10–20 μg/mL. However, the dose needed to reach the therapeutic range can vary considerably from patient to patient depending upon such factors as age, smoking history, other concurrent drugs, and coexisting disease conditions, as well as intrapatient variability in theophylline kinetics. These biological variables, coupled with many physical variables inherent in the manufacturing process, should make it obvious that one SR theophylline product cannot be assumed to be therapeutically equivalent and interchangeable with another. Indeed, wide biological responses with either therapeutic failure or excessive toxicity have been documented when one formulation of SR theophylline has been substituted for another.[8,9] In addition to these adverse biological effects, substitution of SR theophylline products can have adverse economic effects by necessitating additional laboratory tests, office visits, or hospitalization. Hence, it is axiomatic that once an asthmatic patient is stabilized on a particular slow release formulation of theophylline, no other formulation should be substituted for continued maintenance without redetermination of blood levels and retitration of dosage. This caveat applies whether the formulation utilized is a brand name or generic product.

How does the physician assure that the patient will receive the identical theophylline product whenever maintenance refills are dispensed? If the prescription is for a brand name preparation, the physician should write "DAW." If the prescription is for a generic preparation, the physician should specify the name of the manufacturer and write "no substitution." Finally, if a physician is engaged in office dispensing, it is important to demand that the drug repackager always provide an identical formulation from the same manufacturer.

In view of the documented instances of nonequivalence, it will come as no surprise to most physicians that theophylline represents a type of drug for which brand switching should not be permitted without the full knowledge and agreement of the physician. What most physicians do not know, however, is the extent to which state substitution laws are not followed or enforced. If, for example, the physician is not aware that substitution has taken place, a sudden change in the clinical course of the patient may be ascribed to a change in the severity of the disease rather than a change in the medication. Physicians should consider a substitution problem in the differential diagnosis whenever a patient's condition changes for no apparent reason.[10] To protect patients against the possibility of adverse consequences from such unauthorized substitution, the physician should bear in mind that the writing of a prescription is an extension of the cognitive skills employed in physical examination and diagnosis. In recognizing that all patients are not alike, a clinical or cognitive judgment is involved in deciding whether drug substitution should be permitted. This is a judgment or responsibility that should not be delegated to others. By writing "DAW" or "no substitution" for products such as theophylline, the physician is simply asserting his or her own clinical judgment on a matter that is an integral part of the practice of medicine.

The foregoing assertions should not be interpreted as advocating exclusive brand name prescribing or across-the-board prohibition of substitution. Indeed, generic substitution can frequently result in substantial cost savings. However, since the first obligation of physicians and pharmacists is to their patients, the therapeutic value of any drug selected and dispensed for an individual patient must be established before the cost of the drug is considered. Physicians and pharmacists must control the process of drug prescribing and dispensing, and not abrogate that responsibility to business managers, third party payers, procurement officials, or other nonprofessionals. Unfortunately, many physicians and pharmacists employed in managed health care systems may be denied, or inappropriately surrender, their professional rights to select drugs based upon therapeutic value rather than on cost alone.

Are generic versions of brand name drugs really bioequivalent and therapeutically interchangeable? For drugs such as SR theophylline, the answer is clearly "no." For many other drugs, the answer is undoubtedly "yes." But the dilemma for the physician is to determine which of the multisource drug products are truly interchangeable and, therefore, substitutable. Guidance on this complex question may be found in a recently adopted policy statement of the American Medical Association. This states, in part, that "the physician should avoid substitution unless the products have been proven to be bioequivalent, and once a medication has been prescribed and begun (generic or brand name), that no further substitution be made without the attending physician's permission."[11]

Until questions about the validity of the current methodology for approval of bioequivalence for all drug products can be resolved, this is sound advice.

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Reforming New York City's Office of the Chief Medical Examiner

The Office of the Chief Medical Examiner of New York City has an enviable 70-year track record of scientific sleuthing and public service. To read graphic press accounts of recent desolation and demise, however, local citizens might think that it had never been so. The retirement of the revered Milton Helpern in 1974, distastefully pushed by the press and media, resulted in a dramatic change in attitude and thus perception of the office.[1] Shortly before and soon thereafter, charges and countercharges were mounted and acrimony prevailed. Now, for the second time in eight years, amidst further allegations regarding certain improprieties and "leadership" deficiencies, a superbly trained and experienced forensic pathologist has been dismissed.[2,3] Quincy lasted longer on television.

In the early days of the office, the chief medical examiner (there has not been a "coroner" since 1918)[4] was a career pathologist who worked his way up the ladder, conducting scene investigations, performing autopsies, and testifying in court, honing his skills over time. In those days, the protection of civil service was sufficient for these dedicated and respected physicians (would Mayors LaGuardia or Wagner have dared fire the medical examiner? and for what reason?), and the next in line would eventually assume the leadership spot following the retirement of the incumbent. Milton Helpern was appointed chief at the same age that his protégé was recently removed—in his mid-50s.[5] In other words, one proved one's mettle in the trenches, learned how the chief ran the office, and then, after 25 years, was probably ready to take over. But during the last 15 years, all this has changed. Stripping the office of independence and putting the medical examiner under the control of city hall, the financial crunch continuing from the mid-1970s, a markedly increased caseload, and—most important—the lack of qualified, experienced people in their early 50s ready to assume the mantle of leadership, have produced the debacle being played out over these many months. The accompanying tragedy is that public trust has declined, aided and abetted by more media revelations; new, young pathologists come but soon go, research comes to a halt, other physician colleagues look on with disdain, and outside medical examiners along with their national association (who really do care about what is happening) are saddened and distressed.

Is there a prescription, or at least some remedy, after this period of decline and fall? Several therapeutic regimens seem obvious. Many cities and states have enacted model legislation whereby a commission of peers representing law, medicine, police, pathologists, funeral directors, etc, jointly appoint and monitor, via regular meetings, the chief medical examiner and the conduct (if not the fiscal affairs) of the office. If adopted, this measure would remove sole power from the mayor along with the attendant politics. Thus, the commission together with the mayor become partners responsible for the medical examiner and his stewardship. The mayor could neither hire nor fire a chief medical examiner without the concurrence of this watchdog group. The responsibility is spread, yet control is tightened, while independence in forensic matters is assured.

Jurisdictions determined to hire the best person are paying salaries to their chief medical examiners comparable to what the directors of hospital pathology laboratories are earning, well over $100,000 and approaching $150,000 per year. The present $95,000-per-year salary of New York City's chief medical examiner should be the starting salary of each deputy, who similarly must be carefully chosen and constantly groomed. Support personnel, eg, toxicologists, serologists, etc, must also be adequately and appropriately compensated.

How can anyone account for 15,000 autopsies (not to mention some 25,000 other cases certified but not autopsied) with their accompanying trail of paperwork; honor family, insurance company, and attorney requests in a timely fashion; assist police and prosecutor in criminal investigations; and then supervise those employees who handle this load in a city of seven million people? This is hardly what young pathologists learn in hospitals, or what forensic scientists absorb at murder scenes. Medical administrators, on the other hand, are trained to interface with the scientist and professional, and in turn with their fiscal and personnel counterparts in government. Whatever physician and management effort is presently in place could doubtless be strengthened and streamlined.

Salaried and tenured faculty position(s) for the chief medical examiner (and some deputies) should be the rule, not the exception. What they have to offer medical and law students, interns and residents, and the health care, public safety, and environmental communities, at the office or in the classroom, more than justifies this complementary status. But such an arrangement has never been easily accomplished. Helpern was heard to say, "Before you teach the students, you must teach the professors!" (personal communication, 1965). The truth of the matter is that both need each other, now as never before.

Thousands of autopsies cannot be performed by one chief, no matter how talented, nor by five deputies, who must supervise their boroughs. They must be performed by young physicians who accept the challenge of a forensic career and who elect to train at the best medical examiner offices in the United States. In these settings, they can develop camaraderie with a talented, eager, and experienced staff, who in turn encourage them along a career path. Unfortunately, at the national level we are barely replacing retiring forensic experts with new ones, and this bodes poorly for the future. Larger offices (and New York must return to its past prominence here) need to train many more medicolegal pathologists than are presently entering the field, whether full- or part-time.

Restoring an image, albeit tarnished, takes time, measured not by clock but by calendar. A gradual improvement in each area will not come all at once, nor will it be applauded by the many critics when it does arrive. A long-range plan with defined goals, agreed to and continually worked on by every concerned party, is essential. Rather than hindering by exploitation, the press and media need to enlist the support of local and state institutions and societies, medical and otherwise, and engage in constructive prodding while speaking in positive tones.

Whither New York City? Is it ready to listen, to spend, to change, in short, to lead as it did during the first 50 years? The late Richard Childs, longtime president of the National Municipal League, spent a lifetime (well into his 90s) advocating independent, professional, competent, and objective medicolegal death investigation, and helped New York City establish its current legal framework those many years ago.[7] Some of the early guardians as well as stewards of the current system, along with the rest of us, are waiting for some equally long-lasting and requisite strong medicine.

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Trends in medical student views of the community served by an inner-city medical center

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ABSTRACT. A questionnaire survey was conducted in 1986 of second-year medical students at State University of New York Health Science Center at Brooklyn in order to obtain their views of the community served by the center. The results of this survey were compared to those of one conducted at the same institution in 1978, using an identical instrument.

One hundred and fifty-two students responded in 1986 and 181 responded in 1978. Significantly higher proportions of 1986 students (14.5%) expressed intentions of doing their postgraduate training in Brooklyn compared to 2.2% in 1978. A total of 28.9% of the 1986 group compared to 16.6% of the 1978 group planned to train in other areas of New York City. In 1978, 49.7% of respondents gave a high rating to the quality of postgraduate training programs in Brooklyn. This proportion was significantly higher (74.3%) among 1986 respondents.

Significantly more 1986 students (14.5%) expressed an interest in practicing in Brooklyn, compared to 3.3% in 1978. The students surveyed in 1986 expressed less concern about crime and danger and more concern about bad environment, inadequate public services, high tax rates, and housing costs as deterrents to living in Brooklyn and other areas of New York City. Some possible reasons for the significant differences observed in responses between students surveyed in 1978 and 1986 are discussed.

(NY State J Med 1988; 88:125–132)

The State University of New York Health Science Center at Brooklyn (SUNY HSCB) is a large, tertiary care complex located in the geographic center of Brooklyn, which is one of New York City's five boroughs. There are several degree-granting colleges in the center—medicine, health-related professions, nursing, and the School of Graduate Studies. The State University Hospital has 350 beds and is a modern tertiary care facility. The municipal Kings County Hospital Center, which is the third largest general care hospital in the county, with 1,284 beds, is situated across the street from SUNY HSCB. Both hospitals, along with a number of affiliated voluntary hospitals, are used as sites for clinical teaching.

Over the past several decades, major demographic changes have taken place in Brooklyn. At present, most hospitals serve a patient population that is predominantly black or Hispanic. The immediate catchment area of the medical center is populated by a large number of recent immigrants from Haiti and the English-speaking Caribbean.

Like most tertiary care centers, SUNY HSCB maintains expensive and complex tertiary care capabilities, supports biomedical research, and trains large numbers of future physicians, nurses, health-related professionals, and researchers. The center is viewed by the communities that it serves not only as a source of medical care, but also as holding potential solutions to a number of social and economic concerns. Fox[1] has explained that most communities have often tended to "medicalize" these concerns and present them to large, inner-city medical centers for solution. High unemployment levels, poverty, marital problems, crime, and social alienation are not amenable to solutions through the biomedical resources of large, inner-city medical centers. During the 1960s and 1970s, communities often expected and demanded that such centers participate in the solution of these problems. In part, this was due to the fact that major medical centers were the only large institutions left in inner-city areas. During the 1980s, these community expectations and demands have been tempered by an experience that has demonstrated that medical centers cannot affect many of these problems, even when pressed to do so.

These centers must not only provide tertiary care and train large numbers of professionals, but also cope with complex fiscal issues and provide extensive primary care services to a population not served by large numbers of private physicians.[2]

The number of practicing physicians in Brooklyn has steadily declined during the past several decades. One study documented that in 1960 there were 6,000 physicians in practice, whereas in 1975 the number had fallen to 3,000.[3] In 1987, there were a total of 7,294 physicians in Brooklyn, of whom some 3,041 were involved in direct patient care.[4] The remainder, according to the American Medical Association's Physician Master File, were either

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unclassified as to activities (1,456), in medical teaching (101) and research (178), retired (159), semiretired (120), or else listed in a variety of other categories including internship (140), residency training (1,589), fellowship (203), research fellowship (13), administration (170), inactive (159), disabled (8), and other types of activities.

The Medical Society of the County of Kings has a membership of 2,318, representing 31.7% of the 7,294 physicians in the borough. Of the 2,318 members, 916 (39.5%) are life members.[4,5]

The total population of the borough has significantly declined over the past few decades from about 2.8 million in 1950 to 2.2 million in 1980, representing a 21% decline. Minority groups now represent the majority of the population. The decline in physicians, which seems to have stabilized in recent years, was due to retirement and death, relocation, and nonreplacement. As a borough that progressively became an inner-city poverty area, except for a few areas, Brooklyn did not attract many young physicians to practice within the context of the models that functioned for many decades. Local social and economic changes and the ease of even cross-continental relocation for physicians resulted in an exodus from Brooklyn and New York City of most of the center's graduates for many years. These trends, which persisted for the decades of the 1950s through the 1970s, have now undergone change. Important areas of Brooklyn are being redeveloped, neighborhoods are stabilizing, and new industries are being established. Linked to these changes is the surplus of medical school graduates, reducing the success rate of many when applying for internships and residencies out of state, particularly on the West Coast.

In 1978, a survey was conducted of the second-year medical school class at SUNY HSCB in order to obtain their views of the community served by the medical center.[6] This survey was undertaken at a time when New York City had not yet recovered from its fiscal crisis and when opportunities in medicine outside of the local area still seemed limitless to recent medical school graduates.

There have been a number of economic and social changes in New York City and Brooklyn during the 1980s. In addition, postgraduate training opportunities for the center's medical graduates during this decade are not as broad as they were in the previous two decades, largely due to the competition engendered by increased numbers of medical school graduates nationwide. In addition, the urban problems and the deterrents to living in New York City are now widely perceived as not being unique to the city, but as being present in most large cities in the country. Given these changes, it was thought useful to conduct a repeat survey of second-year students in order to both ask the same questions posed in 1978 and compare the responses to those obtained eight years earlier.

TABLE I. Demographic Characteristics of Second-Year Medical Students, SUNY HSCB, 1978 and 1986*

	1978		1986	
Age in Years	No.	%	No.	%
20-22	50	27.6	30	19.7
23-25	106	58.6	87	57.2
26-28	17	9.4	18	11.8
29-31	4	2.2	8	5.3
31	4	2.2	9	5.9
Total	181	100.0	152	99.9†

* Chi-square, 4df = 7.69; $p = 0.10$.

† Column totals in tables may not equal 100% due to rounding.

TABLE II. Second-Year Medical Students by Sex, SUNY HSCB, 1978 and 1986*

	1978		1986	
	No.	%	No.	%
Male	131	72.4	95	62.5
Female	49	27.1	56	36.8
No answer†	1	0.6	1	0.7
Total	181	100.1	152	100.0

* Chi-square, 1df = 3.28; $p = 0.07$.

† Excluded from chi-square analysis.

METHODS

The survey was conducted among second-year medical students in December 1986. The survey instrument used was identical to the one used in 1978 except for some minor modifications. The questionnaire contained 27 questions consisting of both fixed-alternate and open-response types and required an average of 20 minutes for completion. The several open-response questions were included to allow students to give frank expression to their opinions about specific aspects of life in Brooklyn. Student participation was completely voluntary, and students were told not to identify themselves on the survey in any manner. To insure anonymity, students collected the questionnaires and brought them to the departmental offices.

The level of participation was high, with 152 of 214 (71.0%) students answering the survey. Nonparticipation (29.0%) resulted from student absenteeism from the final seminar session of the course during which the questionnaire was administered, and from the failure of a couple of seminar leaders to distribute the questionnaire. The results of the 1978 and the 1986 survey were compared for similarities and differences.

RESULTS

The results obtained in the survey demonstrated a variety of student characteristics and opinions.

Demographic Characteristics of Students. Of the 152 students who responded to the questionnaire, 87 (57.2%) were between 23 and 25 years of age; another 30 (19.7%) were between 20 and 22 years, and 18 (11.8%) were between 26 and 28 years of age. A comparison of the demographic characteristics of the students surveyed in both 1978 and 1986 using chi-square analysis revealed no significant differences between the two groups, although there was a somewhat higher proportion of female respondents in 1986 (Tables I, II, III).

Residence. While enrolled in college, 65 (42.7%) students had lived in one of the five boroughs of New York City. Of these, 24 (15.8%) lived in Brooklyn. Some 40 (26.3%) lived outside New York

TABLE III. Marital Status of Second-Year Medical Students, SUNY HSCB, 1978 and 1986*

	1978		1986	
	No.	%	No.	%
Single†	147	81.2	129	84.9
Engaged	8	4.4	8	5.3
Married	26	14.4	15	9.9
Total	181	100.0	152	100.1

* Chi-square, 2df = 1.6; $p = 0.45$.

† Includes separated, divorced, widowed.

State (Table IV). Chi-square analysis of the data from 1978 and 1986 showed no significant differences between the two years.

While enrolled at SUNY HSCB, 131 (86.2%) said they lived in Brooklyn; another 13 (8.6%) listed other areas of the city as their place of residence (Table V). Chi-square analysis of residence data from 1978 and 1986 showed no significant differences.

College Education. Some 67 (44.1%) students had attended a college within New York City. Forty-six (30.3%) attended college outside of the state (Table VI). Chi-square analysis revealed no statistically significant differences between the groups surveyed in 1978 and 1986.

Familiarity with Brooklyn. Students were asked about their familiarity with Brooklyn outside of the immediate medical school area. Forty-three (28.3%) said they knew it "extremely well"; 51 (33.6%) knew it "fairly well"; and 58 (38.2%) responded "do not know well" (Table VII). Chi-square analysis revealed no significant differences between the 1978 and 1986 responses (Table VII).

Postgraduate Plans. Among the 152 respondents, 22 (14.5%) stated that they planned to do their postgraduate training in Brooklyn; 44 (28.9%) said New York City other than Brooklyn (Table VIII). Chi-square analysis revealed a significant difference between the responses given in 1978 and 1986. Some five-fold more students in 1986 stated that they planned to do their training in Brooklyn compared to 1978.

Students were also asked their subjective impressions of the quality of postgraduate medical training programs in Brooklyn compared to elsewhere. In 1978, 67 of 181 (37.0%) respondents had said that programs in Brooklyn were "as good as" those elsewhere; 16 (8.8%) said "better than"; 7 (3.9%) said "much better than" (Table IX). Forty (22.1%) gave no answer; 9 (5.0%) said the programs "were much worse than elsewhere"; and 42 (23.2%) said the programs were "worse than" elsewhere.

The 1986 data (Table IX) revealed marked differences in the responses to this question, which are statistically significant on chi-square analysis. In 1986, only 13.8% replied that Brooklyn-based programs were "worse than" others elsewhere compared to 23.2% in 1978. Forty-eight percent said they were "as good as" compared to 37% in 1978; 15.1% said they were "better than" compared to 8.8% in 1978; 11.2% said "much better than" compared to 3.9% in 1978. In 1978, 22.1% did not answer this question, whereas in 1986 only 7.9% did not.

Practice Locations. Students were asked about their projected geographic area of medical practice. As shown in Table X, the 1986 results were significantly different on chi-square analysis from those for 1978. In 1986, 14.5% replied that they planned to practice in Brooklyn, a three-fold increase over those who so replied in 1978 (Table X). Thirty (19.7%) students said they planned to practice in other areas of the city compared to 20 (11.1%) who gave this reply in 1978. In 1978, 53.1% of the students did not know their projected practice location, whereas in 1986 only 31.6% gave this reply (Table X).

Table XI presents the responses to the question about population size of the practice location. Chi-square analysis revealed no significant difference between the 1978 and 1986 responses. The majority of students preferred to practice in large cities. It is noteworthy that in 1986 only 21.1% did not state a preference, whereas in 1978, 31.0% had no preference.

In 1986, students were asked about regional preferences for practice, a question that was not asked in 1978 (Table XII). The vast majority of respondents, 120 (79.0%), stated a preference for the Northeast.

Specialty Choice. There was a significant difference on chi-square analysis between the responses given in 1978 and 1986 with regard to specialty choice when "do not know" responses were excluded (Table XIII). More students in 1986, 29 (19.1%), stated a preference for surgery than had in 1978, 18 (9.9%).

Living in Large Cities. Students were asked general questions aimed at eliciting their opinions about living in large cities. Table XIV lists the deterrents to living in Brooklyn from which students were asked to choose. There were significant differences between the data obtained in 1978 and 1986 on chi-square analysis. A bad environment, expense, inadequate public services, housing/rental costs,

TABLE IV. Place of Residence While in College, Second-Year Medical Students, SUNY HSCB, 1978 and 1986*

	1978			1986		
	No.	%		No.	%	
Brooklyn	40	22.1		24	15.8	
Manhattan	8	4.4	23.7	18	11.8	26.9
Queens	22	12.1		14	9.2	
Bronx	11	6.1		4	2.6	
Richmond	2	1.1		5	3.3	
Long Island	26	14.4		22	14.5	
Elsewhere in New York State	34	18.8		25	16.4	
Other than New York State	38	21.0		40	26.3	
Total	181	100.0		152	99.9	

* Chi-square, 4df = 3.30; p = 0.51.

TABLE V. Place of Residence While Attending Medical School, Second-Year Medical Students, SUNY HSCB, 1978 and 1986*

	1978			1986		
	No.	%		No.	%	
Brooklyn	160	88.3		131	86.2	
Manhattan	5	2.8	11.7	4	2.6	12.6
Queens	7	3.9		7	4.6	
Bronx	1	0.6		1	0.7	
Richmond	—	—		1	0.7	
Long Island	6	3.3		6	4.0	
Elsewhere in New York State	—	—		0	0	
Other than New York State	2	1.1		0	0	
No answer†	0	0		2	1.3	
Total	181	100.0		152	100.1	

* Chi-square, 1df = 0.02; p = 0.90.

† Excluded from chi-square analysis.

TABLE VI. Site of College Education of Second-Year Medical Students, SUNY HSCB, 1978 and 1986*

	1978		1986	
	No.	%	No.	%
New York City	86	47.5	67	44.1
New York State	57	31.5	38	25.0
Outside New York State	38	21.0	46	30.3
No answer†	0	0	1	0.7
Total	181	100.0	152	100.1

* Chi-square, 2df = 4.24; p = 0.12.

† Excluded from chi-square analysis.

TABLE VII. Second-Year Medical Students' Familiarity with Brooklyn, SUNY HSCB, 1978 and 1986*

	1978		1986	
	No.	%	No.	%
Know extremely well	55	30.4	43	28.3
Know fairly well	63	34.8	51	33.6
Do not know well	63	34.8	58	38.2
Total	181	100.0	152	100.1

* Chi-square, 2df = 0.42; p = 0.81.

TABLE VIII. Second-Year Medical Students' Choice of Location for Postgraduate Training, SUNY HSCB, 1978 and 1986*

	1978		1986	
	No.	%	No.	%
Brooklyn	4	2.2	22	14.5
New York City other than Brooklyn	30	16.6	44	28.9
New York State other than New York City	14	7.7	12	7.9
Other than New York State	31	17.1	20	13.2
Do not know	102	56.4	54	35.5
Total	181	100.0	152	100.0

* Chi-square, 4df = 30.48; $p = 0.001$; chi-square, 3df = 15.78; $p = 0.001$ (excluding "do not know").

TABLE IX. Subjective Impressions of Second-Year Medical Students of the Quality of Postgraduate Medical Training Programs in Brooklyn Compared to Elsewhere, SUNY HSCB, 1978 and 1986*

	1978		1986	
	No.	%	No.	%
Much worse	9	5.0	6	4.0
Worse	42	23.2	21	13.8
As good as	67	37.0	73	48.0
Better than	16	8.8	23	15.1
Much better than	7	3.9	17	11.2
No answer	40	22.1	12	7.9
Total	181	100.0	152	100.0

* Chi-square, 5df = 26.03; $p = 0.001$; chi-square, 4df = 13.28; $p = 0.001$ (excluding no answer).

TABLE X. Projected Practice Locations, Second-Year Medical Students, SUNY HSCB, 1978 and 1986*

	1978		1986	
	No.	%	No.	%
Brooklyn	6	3.3	22	14.5
New York City other than Brooklyn	20	11.1	30	19.7
New York State other than New York City	22	12.2	20	13.2
Other than New York State	37	20.4	32	21.1
Do not know	96	53.1	48	31.6
Total	181	100.0	152	100.1

* Chi-square, 4df = 25.27; $p = 0.001$; chi-square, 3df = 9.79; $p = 0.02$ (excluding "do not know").

TABLE XI. Projected Character of Practice Location Site, Second-Year Medical Students, SUNY HSCB, 1978 and 1986*

	1978		1986	
	No.	%	No.	%
Very large city	59	32.6	70	46.1
City of 100,000	29	16.0	25	16.5
City of 50–100,000	22	12.2	13	8.6
Small town	7	3.9	7	4.6
Rural area	8	4.4	5	3.3
Do not know	56	31.0	32	21.1
Total	181	100.2	152	100.2

* Chi-square, 5df = 8.32; $p = 0.14$; chi-square, 4df = 4.14; $p = 0.39$ (excluding "do not know").

TABLE XII. Geographic Choices for Practice of Second-Year Medical Students, SUNY HSCB, 1986

Area	No.	%
Northeast	120	79.0
West coast	5	3.3
Arizona, Colorado, and adjacent areas	2	1.3
Midwest	3	2.0
South	4	2.6
Do not know	18	11.9
Total	152	100.1

and noise were listed as deterrents by a significantly greater proportion of students in 1986 compared to 1978. In addition, 18.1% of respondents in 1986 said there were no deterrents to living in Brooklyn, compared to 6.2% who gave this response in 1978 (Table XIV).

Students were asked an identical question about deterrents to living in Manhattan (Table XV). Crime, danger, crowds, pollution, and the quality of schools were seen as less of problems in 1986 compared to 1978. However, significantly higher proportions of 1986 respondents saw bad environment, inadequate public services, housing/rent costs, and noise as deterrents compared to their 1978 counterparts. In 1978, only 8.3% found no deterrents to living in Manhattan, whereas in 1986, 21.1% did.

Students were questioned about the deterrents to living in any large city (Table XVI). Among 1986 respondents, a significantly lesser proportion saw crime as a deterrent compared to respondents in 1978. A significantly higher proportion of 1986 respondents saw bad environment, expense, inadequate public services, and noise as deterrents compared to 1978 respondents. In 1986, 31.0% saw no deterrents, whereas only 10.8% saw none in 1978.

Crime Victims. Students were asked if they knew of anyone who had been the victim of a crime while that person had been involved in a medical-center-related activity. In 1978, 135 students (74.6%) knew of another medical student who had been the victim of a crime, whereas in 1986, only 58 (38.2%) knew of one. In 1978, five respondents (2.8%) said that they had been the victim of a crime. In 1986, six times more, 31 (20.4%), said they had been victims (Table XVII).

New York Tax Structure. The majority of students thought that tax rates for New York City were very high or high (Table XVIII). Chi-square analysis demonstrated no significant difference between the answers obtained in 1978 and 1986. However, the 1978 and 1986 responses, with regard to New York State taxes, were statistically significant. In 1978, 52.5% stated that the state's taxes were very high, whereas in 1986, 36.8% gave this response. In 1978, 33.1% said the state's taxes were high, while in 1986, 49.3% said they were. A small percentage in both 1978 and 1986 said that the city and state taxes were low (Table XVIII).

DISCUSSION

The borough of Brooklyn consisted of predominantly

TABLE XIII. Projected Specialty Choice of Second-Year Medical Students, SUNY HSCB, 1978 and 1986*

	1978		1986	
Specialty	No.	%	No.	%
Internal medicine	59	32.6	55	36.2
Pediatrics	14	7.7	10	6.6
Surgery	18	9.9	29	19.1
Obstetrics-gynecology	7	3.9	3	2.0
Other	20	11.1	8	5.3
Do not know	63	34.9	47	30.9
Total	181	100.1	152	100.1

* Chi-square, 5df = 10.00; $p = 0.08$; chi-square, 4df = 9.40; $p = 0.05$ (excluding "do not know").

TABLE XIV. Second-Year Medical Students' Opinions About Deterrents to Living in Brooklyn, SUNY HSCB, 1978 and 1986

	1978		1986		
Deterrents	No. of Responses*	% of Respondents	No. of Responses†	% of Respondents	Probability
Crime	74	51.0	47	40.5	0.09
Danger	47	32.4	32	27.6	0.40
Crowded	34	23.3	30	25.9	0.65
Dirty	31	21.4	19	16.4	0.31
Bad environment	30	20.7	65	56.0	0.01
Pollution	18	12.4	16	13.8	0.74
Schools	18	12.4	11	9.5	0.45
Expensive	14	9.7	20	17.2	0.07
Inadequate public services	10	6.9	20	17.2	0.01
High tax rate	7	4.8	9	7.8	0.33
Housing/high rent	6	4.1	14	12.1	0.02
Noise	5	3.5	16	13.8	0.01
None	9	6.2	21	18.1	0.01
Total	303		320		

* Number of respondents = 145.
† Number of respondents = 116.

TABLE XV. Second-Year Medical Students' Opinions About Deterrents to Living in Manhattan, SUNY HSCB, 1978 and 1986

	1978		1986		
Deterrents	No. of Responses*	% of Respondents	No. of Responses†	% of Respondents	Probability
Crime	53	39.8	37	32.5	0.23
Danger	29	21.8	17	14.9	0.17
Crowded	55	41.4	43	37.7	0.56
Dirty	17	12.8	16	14.0	0.77
Bad environment	12	9.0	39	34.2	0.01
Pollution	25	18.8	15	13.2	0.01
Schools	15	11.3	9	7.9	0.37
Expensive	51	38.3	52	45.6	0.25
Inadequate public services	6	4.5	21	18.4	0.01
High tax rate	6	4.5	10	8.8	0.18
Housing/high rent	10	7.5	19	16.7	0.03
Noise	4	3.0	20	17.5	0.01
None	11	8.3	24	21.1	0.01
Total	294		322		

* Number of respondents = 133.
† Number of respondents = 114.

TABLE XVI. Second-Year Medical Students' Opinions About Deterrents to Living in Any Large American City, SUNY HSCB, 1978 and 1986

	1978		1986		
Deterrents	No. of Responses*	% of Respondents	No. of Responses†	% of Respondents	Probability
Crime	52	55.9	24	35.3	0.01
Danger	15	16.1	12	17.6	0.80
Crowded	32	34.4	20	29.4	0.50
Dirty	15	16.1	9	13.2	0.61
Bad environment	12	12.9	29	42.6	0.01
Pollution	15	16.1	11	16.2	0.99
Schools	8	8.6	7	10.3	0.72
Expensive	18	19.4	23	33.8	0.04
Inadequate public services	3	3.2	13	19.1	0.01
High tax rate	4	4.3	7	10.3	0.14
Housing/high rent	8	8.6	9	13.2	0.35
Noise	3	3.2	10	14.7	0.01
None	10	10.8	21	31.0	0.01
Total	195		195		

* Number of respondents = 93.
† Number of respondents = 68.

TABLE XVII. Second-Year Medical Students' Knowledge of Local Crimes, SUNY HSCB, 1978 and 1986*

	1978		1986	
Crime Victim	No.	%	No.	%
Self	5	2.8	31	20.4
Another medical student	135	74.6	58	38.2
Another non-medical student or employee	16	8.8	16	10.6
Do not know	25	13.8	47	31.0
Total	181	100.0	152	100.2

* Chi-square, 3df = 54.11; $p = 0.001$; chi-square, 2df = 41.40; $p = 0.001$ (excluding "do not know").

working class and middle class populations for the first half of the century. During the 1960s many of the borough's neighborhoods began to undergo demographic changes, while the borough as a whole lost businesses and industries. The exodus of businesses from New York City as a whole and a middle class immigration to new suburbs peaked during the 1970s, when the city administration suffered a severe fiscal crisis.[7] However, the doom and gloom that hung over the city did not continue into the 1980s due to a variety of factors. The city emerged from the fiscal crisis and for most of the 1980s has had a balanced budget and even surpluses in certain years. Although some industries moved out, service industries moved in and more than replaced the jobs lost by the earlier exodus. A construction boom has taken place in the central borough of Manhattan for most of the 1980s, resulting in the construction not only of new office towers, but also of cooperative apartments, condominiums, and rental housing.

During the late 1970s it was popularly held that Brooklyn was moving inexorably into decay. For example, by 1978, Bushwick, a large section of northern Brooklyn, had half of its population on welfare. In 1960, 77% of the residents had been working class and middle class.[8] Many sections of Brooklyn had abandoned and burned-out buildings, youth gangs, crime, and numerous social problems. The white, middle class neighborhood to which SUNY HSCB relocated in 1955 had become a poor, black, inner-city area by 1975. Brooklyn's decline was seen by many as progressive in the late 1970s.

TABLE XVIII. Second-Year Medical Students' Opinion About the Tax Structure in New York City and New York State, SUNY HSCB, 1978 and 1986

	New York City*				New York State†			
	1978		1986		1978		1986	
Tax Level	No.	%	No.	%	No.	%	No.	%
Very high	104	57.5	76	50.7	95	52.5	56	36.8
High	53	29.3	57	38.0	60	33.1	75	49.3
Average	7	3.9	6	4.0	13	7.2	10	6.6
Very low‡	1	0.6	—	—	—	—	—	—
No answer	16	8.8	11	7.3	13	7.2	11	7.2
Total	181	100.1	150	100.0	181	100.0	152	99.9

* Chi-square, 3df = 2.80; $p = 0.42$; chi-square, 2df = 2.53; $p = 0.28$ (excluding no answer).

† Chi-square, 3df = 9.85; $p = 0.02$; chi-square, 2df = 9.85; $p = 0.01$ (excluding no answer).

‡ Excluded from chi-square analysis.

The 1980s have witnessed a marked change in many aspects of the city's life. New York has experienced an economic boom for most of the 1980s which has significantly altered the prevalent negative attitudes of the 1970s. Brooklyn has experienced significant redevelopment in a number of geographic areas. New office towers are being built in the civic center of the borough, and urban renewal has been going on for a number of years in many areas. Gentrification of some areas has been progressive, particularly in Park Slope, Carroll Gardens, Bedford-Stuyvesant, Cobble Hill, and Boerum Hill. Some minority individuals have economically moved into middle class status and have worked to improve the quality of their neighborhoods. This is evidenced in the area in which the medical center is located. Many one- and two-family homes have been renovated and improved by minority owners.

Few medical students at SUNY HSCB have had the opportunity to work with the numerous community groups in Brooklyn that have been striving for a number of years to solve a broad spectrum of problems, including health-related ones. Thus, these students are cut off from the positive forces at work for social, economic, and health care improvements. The opinions of medical students in the late 1970s in Brooklyn were shaped by what they saw and experienced: progressively decaying neighborhoods, burned-out and abandoned buildings, marked declines in city services, and the absence of urban renewal. During the 1980s, decay has been halted in many geographic areas, renewal is visible, and the level of certain city services has improved. The physical infrastructure of the city, including roads, parks, and subway cars and public buses, has also been appreciably improved. Some of the negative perceptions of the 1970s have been replaced by more postive ones. This is not fully reflected in the student responses over time, which indicate an increase in the perception of several deterrents to living in large cities.

The 1978 and 1986 second-year students surveyed did not significantly differ in terms of their demographic characteristics. There are, however, more women enrolled in the entering medical school classes now. In the 1986 class surveyed, 36.8% of respondents were women, compared to 27.1% in 1978. However, attitudes were similar between male and female medical students in both classes.

Although the differences are not statistically significant (Tables IV, VI), fewer students in the 1986 groups lived and attended college in New York City than in the 1978 group. In 1986, a higher proportion of students had attended college outside of New York State—30.3% compared to 21.0% in 1978 (Table VI). In both years, the vast majority of students lived in Brooklyn at the time of the survey.

In both years, fewer than a third of the students said they knew Brooklyn "extremely well" (Table VII). However, 65.2% in 1978 and 61.8% in 1986 said they knew the borough either "extremely well" or "fairly well." Thus, the majority of students have a good level of familiarity with Brooklyn.

An important finding in the 1986 survey is the more than five-fold increase in the number of students who stated that they planned to do their postgraduate training in Brooklyn (Table VIII). Also significant was that 28.9% in

1986 compared to 16.6% in 1987 stated that they planned to seek their training in New York City other than Brooklyn. Another important difference between the two groups is that 56.4% of those in 1978 either did not know their plans or else gave no answer, compared to only 35.5% in 1986. Thus, a higher proportion of 1986 students knew their future plans compared to those surveyed eight years earlier. The significant changes in 1986 for opting for postgraduate training in both Brooklyn and other parts of New York City may have several causes. The improved economic climate of both the borough and the city may be one. There is also a perception among students that matching for positions outside of New York City has become increasingly competitive. This is based in part on knowledge of the number of annual medical school graduates in the country, which had steadily increased for many years until 1985.[9] Some faculty have voiced the opinion that many SUNY HSCB students undervalue their standing and as a result set their sights too low and too close to home with regard to matching programs. Religiously observant students often try to match at local institutions that will enable them and their families to live in neighborhoods situated in either Brooklyn or elsewhere in New York City. A lower proportion of 1986 respondents were married (9.9%), compared to 14.4% in 1978 (Table III). Thus, spousal, social and employment considerations do not seem to figure in the significant changes documented in 1986.

Perceptions of the quality of postgraduate training programs in Brooklyn clearly play a role in the choices students make. In 1978, 49.7% of students stated that such programs were "as good as," "better than," or "much better than" programs elsewhere. In 1986, 74.3% replied in this manner (Table IX). The 1986 students differ from their 1978 counterparts in that significantly more of them assign a high value to the Brooklyn-based training programs. In 1986, only 17.8% said that the Brooklyn-based programs were "worse" or "much worse" than elsewhere. In 1978, 27.2% gave this response, and 22.1% had no answer. Only 7.9% of the 1986 respondents gave no answer. All those data indicate that significantly more 1986 students thought highly of Brooklyn-based training programs than had their 1978 counterparts.

A significant difference was also observed in projected practice location. In 1986, 14.5% said they planned to practice in Brooklyn, compared to only 3.3% who so replied in 1978. Far more also opted in 1986 for other locations in New York City than had in 1978 (Table X).

In 1978, although 49.7% of respondents thought well of training programs in Brooklyn, only 3.3% planned to practice in the borough. By contrast, in 1986, 74.3% gave a high rating to programs in Brooklyn, and 14.5% said they planned to practice in the borough. These data show an increased interest in practicing in Brooklyn and New York City. In previous years, choice of location for postgraduate study did not correlate with future practice location. This is supported by data from the alumni files of the medical school and the *Medical Directory of the State of New York*. On average, 1.5% of the graduates of years 1969 through 1975 were still in Brooklyn in 1979.[6]

There were no significant differences between the two classes with regard to population size of practice location site and specialty choice (Tables XI, XII).

The vast majority of 1986 students planned to practice in the Northeast (79.0%) compared to the West Coast (3.3%) (Table XII).

Deterrents to living in New York City and Brooklyn still exist, but student opinion about them has changed to some extent over time. Fewer 1986 students listed crime as a deterrent than did 1978 students. Bad environment, inadequate public services, and housing/rental costs were targeted as deterrents by a significantly higher proportion of 1986 students than 1978 students for both Brooklyn, Manhattan, and large cities in general. A higher proportion of 1986 students (18.1%) saw no deterrents to living in Brooklyn, while 21.1% saw none to living in Manhattan. These proportions are much higher than those recorded among 1978 students. Different attitudes may account in part for all these differences. The improved economic and physical environment of many areas of the city may also play a role.

Significantly lower proportions of 1986 students responded that there were no deterrents to living in Brooklyn, Manhattan, or any large American city (Tables XIV, XV, XVI). This may reflect the adaptation over time to many urban problems and the acceptance of these problems by a generation that has been aware of them from an early age.

While fewer 1986 students (38.2%) knew of another medical student who had been a crime victim, compared to 74.6% in 1978, more, 20.4%, compared to 2.8% in 1978, had been victims themselves. The greatly improved security services in and around the medical center implemented during the 1980s may have influenced these findings. Neighborhood improvements may also play a role.

There were no significant differences between the two groups with regard to how they perceived New York City taxes, yet a higher proportion of 1978 students thought New York State's taxes were very high than did 1986 respondents (Table XVIII).

A comparison of the survey data from 1978 and 1986 shows that significant differences exist between the two groups of students. Significantly higher proportions of the 1986 students expressed an interest in doing their postgraduate training in Brooklyn and elsewhere in New York City, gave a high rating to the quality of Brooklyn-based postgraduate training programs, and expressed an interest in practicing in Brooklyn. Concerns about crime and danger in both Brooklyn and New York City were less in 1986 than they had been in 1978. The reasons for these findings include positive changes in both New York City and Brooklyn, and changes in student values, expectations, and perceptions.

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Calcium entry blockers and human platelet aggregation

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ABSTRACT. It has been hypothesized that calcium entry blocking agents inhibit platelet aggregation by decreasing calcium flux across the thrombocyte membrane. Previous studies have given equivocal results, demonstrating decreased platelet aggregation in vitro, but only at concentrations of calcium blockers far above the therapeutic range. Nevertheless, bleeding time is mildly prolonged in patients taking these drugs. We have attempted to resolve this paradox by determining the effect of diltiazem and verapamil, in therapeutic concentrations, on collagen and adenosine diphosphate (ADP)-induced platelet aggregation in vitro. Both diltiazem (180 ng/mL and 360 ng/mL) and verapamil (300 ng/mL, 500 ng/mL, and 1,000 ng/mL) produced significant decreases in collagen-induced platelet aggregation. While this effect was statistically significant, its absolute magnitude was unlikely to have clinical significance. Neither drug produced a significant decrease in ADP-induced platelet aggregation. These results suggest that the effect of calcium entry blockers on platelet aggregation should not be a serious concern for surgical patients whose platelet function is not otherwise compromised.

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Fourteen and a half million prescriptions for calcium entry blocking agents were filled in the United States alone in 1984, for the treatment of angina pectoris and hypertension.[1] Calcium flux across the platelet membrane, together with internal redistribution of stored intracellular calcium, is thought to be essential for the aggregation of human platelets.[2-6] If so, drug-induced inhibition of calcium flux by calcium entry blockers should inhibit thrombocyte aggregation. Two such blockers, verapamil and nifedipine, have been shown to inhibit platelet aggregation induced by epinephrine, adenosine diphosphate (ADP), thrombin, and collagen, but at plasma concentrations far in excess of their therapeutic range.[5,7-14] Nevertheless, therapeutic doses of nifedipine appear to cause a slight increase in template bleeding time in vivo.[12]

In contradistinction to previous in vitro studies, we demonstrate here that two calcium entry blockers, diltiazem and verapamil, do inhibit collagen-induced platelet aggregation by 9-10% at therapeutic concentration. While total aggregation induced by collagen decreased, the time necesary to effect maximum aggregation also decreased, thus demonstrating an expedited but reduced response. Aggregation induced by ADP was not altered, presumably because this process is not dependent on calcium flux.[5,13]

In 1962, Grette[2] postulated the need for calcium during platelet aggregation. Subsequent studies demonstrated calcium ion uptake during aggregation induced by epinephrine or collagen, and it has been shown that flux of intracellular calcium across the cell membrane is followed by reuptake of extracellular calcium during thrombin-induced aggregation.[3] These findings are consistent with the view that calcium is required for all cellular secretory processes.[15] Aggregation induced by ADP may not depend on calcium flux, but may depend on redistribution of intracellular calcium. If so, calcium entry blockers would be expected to inhibit aggregation induced by thrombin, epinephrine, or collagen, but not ADP.

The calcium entry blocker verapamil inhibits platelet aggregation at plasma concentrations of 4×10^{-4}M or 4×10^{-6}M, which are greatly in excess of the therapeutic range.[9] Similarly, nifedipine inhibits in vitro aggregation only at concentrations beyond therapeutic range, but, paradoxically, nifedipine does appear to increase template bleeding time in vivo.[12] This ability of calcium entry blockers to alter platelet activity in vivo, while apparently having no effect at therapeutic concentrations in vitro, creates ambiguity regarding the therapeutic benefits and potential toxicities of these drugs.

METHODS

After obtaining approval of the university's Human Research Committee and informed consent from each of the subjects, 27 mL of whole blood was drawn from each of 12 healthy, fasting, nonsmoking volunteers who were taking no medication or alcohol (seven men, five women; mean age, 36 years). The blood was immediately and gently mixed with 3 mL of 0.11M sodium citrate in a plastic tube.

Platelet-rich plasma (PRP) and platelet-poor plasma (PPP) were then prepared as previously described.[16] Using Born's turbidimetric technique[17] in a Platelet Aggregation Profiler III (Bio Data Corp), platelet aggregation was determined in response to ADP (2×10^{-5}M final concentration) and collagen (0.19 mg/

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mL final concentration), measuring total aggregation as the percent of maximum.

Aliquots of PRP were then incubated at room temperature in sealed siliconized tubes for 15–20 minutes with low therapeutic, high therapeutic, and twice high therapeutic concentrations of each drug (verapamil, 300 ng/mL, 500 ng/mL, and 1,000 ng/mL; diltiazem, 120 ng/mL, 180 ng/mL, and 360 ng/mL).[18,19] Aggregation was determined again. In addition to determining total aggregation, we measured time in seconds between the introduction of collagen and the beginning of aggregation (lag time), which is the time during which platelet membrane permeability increases, leading to the release phenomenon. Results were analyzed by one-way analysis of variance for repeated measures. Where significant F-ratios were found ($p < 0.02$), individual comparisons were made by paired t-test and were considered significant at $p < 0.05$, one-tail.

RESULTS

Collagen-induced platelet aggregation was moderately inhibited at all concentrations of verapamil and at the two highest concentrations of diltiazem (see Table I). ADP aggregation was not significantly affected by either drug at any concentration. Time to aggregation in the collagen curve was significantly decreased by both drugs at all concentrations, except at the lowest concentration of diltiazem. Thus, while therapeutic concentrations of these drugs inhibit the total amount of thrombocyte aggregation by 9–10%, they decrease the time necessary to achieve that amount of aggregation.

DISCUSSION

Primary hemostasis is activated when circulating platelets come into contact with the collagen sub-basement layer of disrupted blood vessels. Arachidonic acid in the platelet membrane is converted into thromboxane A2, which causes an increase in platelet membrane permeability and the release of endogenous ADP and other vasoactive substances. Concurrent with this release phenomenon is calcium flux and intraplatelet calcium redistribution, which causes contraction of thrombasthenin (the platelet smooth muscle component), platelet shape change, and forcible transport of intracellular vacuoles and extrusion of their contents into the peri-platelet spaces.[20,21] Platelets then adhere to each other, forming clumps which precipitate to form a hemostatic plug.

These findings are compatible with the 11.8% increased bleeding time found in patients using calcium entry blockers.[12] Prior in vitro studies may have failed to demonstrate an inhibitory effect of calcium entry blockers in therapeutic concentrations because of faulty technique or failure to incubate PRP with the drugs for a sufficient period of time. While the in vitro change in platelet aggregation found here and the half-minute increase in bleeding time of patients taking these drugs are unlikely to cause clinically significant problems in patients whose platelet function is not otherwise impaired, the ability of members of this drug group to alter hemostasis merits consideration. Specifically, if a surgical patient's platelet function has been impaired by drugs such as aspirin or non-steroidal anti-inflammatory agents, or by disease processes that affect platelet function, the use of calcium entry blockers may further extend bleeding time and exacerbate blood loss.

TABLE I. Collagen-Induced Platelet Aggregation and ADP Aggregation

Drug and Concentration	% ADP Agg (± SEM)[*]	% Collagen Agg (± SEM)	Lag Time in Sec (± SEM)
Controls	84 (± 1.8)	81 (± 2.4)	68 (± 4.6)
Diltiazem			
120 ng/mL	83 (± 1.3)[†]	78 (± 2.4)[†]	59 (± 6.5)[†]
180 ng/mL	83 (± 1.5)[†]	75 (± 1.6)[‡]	45 (± 4.1)[‡]
360 ng/mL	82 (± 2.5)[†]	75 (± 1.5)[‡]	54 (± 3.7)[‡]
Verapamil			
300 ng/mL	85 (± 2.1)[†]	76 (± 1.9)[‡]	56 (± 3.8)[‡]
500 ng/mL	85 (± 1.3)[†]	76 (± 2.0)[§]	56 (± 4.3)[‡]
1,000 ng/mL	80 (± 3.3)[†]	73 (± 3.2)[§]	60 (± 4.7)[§]

[*] SEM = standard error of the mean.
[†] Not significant.
[‡] $p < 0.01$.
[§] $p < 0.05$.

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Systemic effects of ophthalmic medication in the elderly

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Ophthalmic drops are in widespread use among the elderly. These medications can have serious systemic effects, especially in elderly patients, and these effects should be considered in the differential diagnosis of iatrogenic diseases. A review of the literature suggests that the problem has not been specifically looked at in the geriatric population, though most of the patients studied were elderly.

After instillation of an ophthalmic medication into the conjunctival sac, most of the drug, except a small amount which overflows or is swallowed, directly enters into the circulation, being absorbed through conjunctival capillaries, the nasal mucosa, as well as the pharynx. The quantity absorbed can be so large as to produce adverse effects. In addition, there is the possibility of drug interactions with other medications that the patient may be taking. Polypharmacy is very common in the elderly.

The adverse effects of ophthalmic medications can be described according to the predominant system involved, though often more than one system may be affected at the same time.

CARDIOVASCULAR SYSTEM

Cardiovascular effects depend on the type of drug being used. Beta-blockers such as timolol, which is used to treat glaucoma, may cause bradycardia, hypotension, and even syncope (Table I). The National Registry of Drug-Induced Ocular Side Effects has recorded some 1,900 possible adverse reactions to ophthalmic timolol, of which 63% were considered systemic side effects.[1]

McMahon et al[2] describe a 71-year-old woman who had an episode of syncope that occurred shortly after instillation of timolol ophthalmic solution; the medication eventually had to be discontinued. They also found that adverse effects affecting one or more systems occurred in 38 (23%) of 165 patients (age not specified) when timolol was added to their glaucoma therapy. Because of these adverse effects, it became necessary to discontinue this drug in 15 (9%) of the patients studied. Eight patients (4.8%) developed bradycardia, palpitation, hypotension, and syncope with ocular timolol. Severe bradycardia has been induced in elderly patients taking ophthalmic timolol by the addition of quinidine.[3] A bradycardia of similar intensity may be precipitated by the instillation of timolol drops to patients who are already taking digoxin and/or oral beta-blockers. Ophthalmic beta-blockers can also precipitate cardiac failure in the elderly patient with compromised cardiac function. Britman[4] describes an episode of congestive heart failure in a 72-year-old woman that was precipitated by the addition of timolol ophthalmic solution to her glaucoma medication regimen.

Epinephrine used in the treatment of glaucoma significantly increases the incidence of extra systoles.[5] Occasionally, serious adverse effects such as palpitation, hypertension, trembling, paleness, and excessive perspiration (Table I) can occur following the use of adrenergic ophthalmic solutions.[6]

An adrenergic ophthalmic solution such as phenylephrine, which is frequently used as a mydriatic, can be detrimental to patients with borderline cardiac status. Solosko and Smith[7] described three cases of arterial hypertension following instillation of phenylephrine 10% ophthalmic solution in their patients, one of whom was aged 69. The risk of hypertensive reaction, as described by Kim et al,[8] may be greater when phenylephrine is used in patients with autonomic neuropathy, such as patients with diabetes mellitus whose sympathetic nervous systems are partially denervated. Myocardial infarction can also be precipitated by these drugs. Fraunfelder and Scafidi[9] described 33 cases, as reported (although not proven) to the National Registry of Drug-Induced Ocular Side Effects, of adverse reactions with the use of phenylephrine 10%. Of these patients, 60% were aged 65 or older. Within this same group of patients, an impressive 40% were reported to have sustained myocardial infarction associated with the use of phenylephrine ophthalmic solution. Based on data in the National Registry and on phenylephrine package inserts, it was recommended by the authors that phenylephrine solution of only 2.5% or weaker be used in the elderly patient.

RESPIRATORY SYSTEM

Adverse respiratory effects such as bronchospasm can be produced in susceptible individuals with ophthalmic solutions of beta-blockers. Charan and Lakshminarayan[10] described a 69-year-old patient in whom acute bronchospasm developed after he received timolol eye drops, 0.5% solution, one drop twice daily in each eye. Such respiratory effects are less marked with a more selective ophthalmic beta-blocker such as betaxolol.[11] Cholinergic drugs

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TABLE I. Summary of Adverse Effects*

Drug	Organ System	Responses
Beta-blockers (eg, timolol)	Cardiovascular	Bradycardia, hypotension, syncope, palpitation, congestive heart failure
	Respiratory	Bronchospasm
	Neurologic	Mental confusion, depression, fatigue, lightheadedness, hallucinations, memory impairment, sexual dysfunction
	Miscellaneous	Hyperkalemia
Adrenergics (eg, epinephrine, phenylephrine)	Cardiovascular	Extrasystoles, palpitation, hypertension, myocardial infarction
	Miscellaneous	Trembling, paleness, sweating
Cholinergic/ Anticholinesterases (eg, pilocarpine, echothiophate)	Respiratory	Bronchospasm
	Gastrointestinal	Salivation, nausea, vomiting, diarrhea, abdominal pain, tenesmus
	Miscellaneous	Lacrimation, sweating
Anticholinergic (eg, atropine)	Neurologic	Ataxia, nystagmus, restlessness, mental confusion, hallucination, violent and aggressive behavior
	Miscellaneous	Insomnia, photophobia, urinary retention

* This table lists the most prevalent but not all of the adverse effects associated with the use of these ophthalmic solutions.

such as pilocarpine and anticholinesterase agents such as echothiophate iodide can also precipitate similar bronchospasms (Table I).

GASTROINTESTINAL SYSTEM

Adverse gastrointestinal effects, mainly produced by cholinergic eye drops, include salivation, nausea, vomiting, diarrhea and abdominal pain, and tenesmus, along with lacrimation and sweating. Epstein and Kaufman[12] report such an occurrence of toxicity in a 75-year-old man.

NEUROLOGIC SYSTEM

Adverse neurologic effects may be produced by anticholinergic medication such as atropine. These central nervous system disturbances manifest themselves as ataxia, nystagmus, restlessness, mental confusion, hallucinations, violent and aggressive behavior, insomnia, and photophobia (Table I). Though reported in younger age groups,[13] these reactions can occur at any age, particularly when stronger solutions are used. Summers and Reich[14] have shown that postcataractectomy delirium has been attributed to anticholinergic toxicity.

Anticholinergic drugs should be used with extra caution in the elderly. Our experience (unpublished) indicates that giving anticholinergic eye drops to the elderly who were on antiparkinsonian agents such as benztropine or amantadine can precipitate psychosis. In addition to other adverse reactions, these drops can cause urinary retention, particularly in patients with an enlarged prostate. Davison[15] emphasizes the hazards of anticholinergic agents in the elderly; adverse effects include blurred vision, urinary retention, and toxic psychosis.

Mental confusion, depression, fatigue, lightheadedness, hallucinations, and memory impairment also can be produced by ophthalmic beta-blockers. McMahon et al[2] found that of 165 patients studied, 17 (10.3%) experienced neurologic adverse effects with timolol, which resulted in its being discontinued in six patients (3.6%). In addition, Verkijk[16] describes a 70-year-old man with myasthenia gravis in whom the addition of timolol ophthalmic solution resulted in worsening of his disease state.

MISCELLANEOUS

Sexual dysfunction following the use of timolol in 25 patients ranging in age from 26 to 64 years has been reported to the National Registry of Drug-Induced Ocular Side Effects.[17] These reports include impotence (18 cases), decreased libido (nine cases), and decreased ejaculate volume (one case).

Hyperkalemia has also been caused by the use of timolol ophthalmic solution in a 72-year-old man, as reported by Swenson.[18] Beta-blocker-induced hyperkalemia occurs infrequently and is more likely to be seen in patients with several predisposing factors that lead to changes in potassium homeostatis. However, as shown by Swenson's report, this change can occur in patients with no obvious abnormalities in potassium metabolism.

CONCLUSIONS

This review highlights the importance for all health care providers to be aware of the significant systemic absorption of ophthalmic medications, the possible role these drugs play in the dysfunction of various organ systems, and their interactions with other medication. Therefore, it is essential to consider the patient's medical status and medication profile before prescribing any of these agents. All ophthalmic medications, if possible, should be used in minimum concentration to achieve the desired therapeutic effect, and the patient, particularly if elderly, should be carefully monitored for manifestations of systemic toxicity.

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UNTOWARD EFFECTS OF THE NEWER DRUGS

Prior to the current era of medical science the physician was largely dependent upon naturally occurring botanicals and minerals for his dispensable therapeutic agents. Bromides were employed for convulsions, colchicum for gouty arthritis, quinine for malaria and other fevers, iron salts for anemia, iodine for thyroid disorders, foxglove for heart disease, opium for pain, mercury for syphilis, ergot for contraction of the uterus, caffein for diuresis, and a variety of herbs and minerals for purgation. Each therapeutic agent was effective in selected instances; sometimes the efficiency was high, at other times it was low. Most of these preparations are still in use and continue to accomplish great therapeutic good. On the other hand, and without exception, new and improved drugs, which have come largely from the chemical laboratory, have been introduced either to enhance or to replace each of the above-noted agents. No well-informed physician will deny the value of these newer drugs. Some small loss has accompanied a tremendous gain, however, and the untoward reactions of certain of the preparations in a percentage of the patients receiving accepted therapeutic amounts has led to serious or even fatal results.

It is only human to minimize the untoward reactions of a new therapeutic substance in the enthusiasm of discovering and subjecting it to clinical trial. It has been observed also that the full significance of an untoward reaction may not be appreciated until after months or even years of clinical trial have elapsed. . . .

In most instances the side-effects are inconvenient and not serious and subside with cessation of the use of the drug. On the other hand, irreparable and mortal changes have followed the use of some of the substances in an occasional instance. Prophylactic and precautionary measures should be observed with each drug that has been shown to be potentially harmful. Careful watch should be maintained in each patient even though there is no visible evidence of toxicity. To know when to stop a drug is frequently as important as to know when to prescribe it. Finally, it is the duty of every physician who is prescribing either new or established preparations to observe carefully untoward reactions and to report them either to the manufacturer or in a medical publication. As the number of new drugs increases, and we know that this will be a fact, continued vigilance should be practiced.

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(*NY State J Med* 1948; 48:280-286)

A history of the Onondaga Sanatorium for the Treatment of Tuberculosis

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Whether tuberculosis existed in central New York in the pre-Columbian era or in the prehistoric American Indian population has not been clarified. Study of skeletal remains of prehistoric Iroquois Indians from Livingston County of about 1200 AD, from Monroe County from the Owasco era, 500–1200 AD, and from the pre-Columbian era in Seneca County have led to mixed conclusions. Evidence that tuberculosis was present in ancient Egypt has been established.[1,2]

Until the 20th century, tuberculosis was the major cause of death in Syracuse, NY (Table I). In 1882, Robert Koch[3] identified the tubercle bacillus as the cause of tuberculosis, and in 1891 he developed antigenic material which produced an allergic response in infected subjects.[4] At first, bovine tuberculosis was considered identical to human tuberculosis, and it remained for Theobold Smith[5,6] to conclusively differentiate bovine tubercle bacilli from the human bacilli. As the implications of Koch's discovery were realized in this country, an educational beginning was made about the disease under the aegis of such people as Hermann M. Biggs, William Osler, and Edward L. Trudeau. This led to active measures for control of the disease. This article reviews the beginning of the tuberculosis control program in the county of Onondaga in New York State as knowledge about the disease expanded.

The word *sanatorium* comes from the Latin *sanatorius*, meaning an establishment providing therapy by physical agents, such as heliotherapy, rest, exercise, and rehabilitation. Edward Livingston Trudeau,[7] himself a victim of tuberculosis, moved to the Adirondacks and opened a cottage sanatorium in 1884, the first in this country. Syracuse, in Onondaga County, had become something of a medical center by that time, with a medical school (the Geneva Medical College, founded in 1834, transferred to Syracuse in 1872), a board of health with medical representation since 1882, a department of vital statistics since 1873, two general hospitals since shortly after the Civil War, an infectious disease hospital since 1875, and a county home since 1827.

In 1903, Dr Frederick W. Smith, the Syracuse city health officer, stated: "I wish to recommend the building and maintenance of a hospital for indigent consumptives."[8(p45)] In 1908 the first tuberculosis clinic in upstate New York opened in Syracuse under the auspices of the Bureau of Health with Dr H. Burton Doust in charge and Dr David M. Totman as health officer.[9(p36)] That year Totman reported 173 deaths from tuberculosis (death rate in Syracuse 150/100,000 [Fig 1]). Dr C. Floyd Burrows, physician in charge of the City Hospital for Communicable Diseases, recommended "a more energetic crusade against tuberculosis in Syracuse and the establishment of a municipal hospital."[9(p166)] On June 7, 1909, the Onondaga County Board of Supervisors passed a resolution to "investigate the matter of the establishment of a county hospital for tuberculosis as permitted by Chapter 341 of the laws of 1909."[10] Various locations were proposed. Dr Burrows recommended building the hospital[9(p117)] and various other sites were proposed. Finally, the transaction was completed for the purchase of a 137-acre tract from Daniel and Flora Strong and Clarissa M. Harrison and Mary E. Weiting Johnson near Hopper's Glen on Onondaga Hill. This action was approved by the county board of supervisors in 1913.[11]

Onondaga Hill is centrally located within the county and had been the site of the original county courthouse from 1802 to 1830. In 1825 the Onondaga County Board of Supervisors had purchased from Josiah Brown 145 acres of land near the courthouse for the site of the county home, which opened in 1827 to care for the county "wards," as indigents were called in those days. Indigents included some who were sick and could not care for themselves, some with "incurable diseases," some who were retarded, and some who were "insane." There were even some children and pregnant women.[12]

As yet, there were no hospitals in the community, nor would there be for over a quarter of a century. There were, however, persons needing special care, and as specialized facilities for such care became available, transfers were arranged. But one oldtimer remembered that "lungers," as those with phthisis were called, were put out in some tents that looked like a "row of chicken coops."[13] Review of the rosters of the day does not show admission to the county home of persons with tuberculosis, or "consumption" as it would likely have been called.[12] Yet due to the growing awareness of the need for a tuberculosis control

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TABLE I. Number of Deaths by Year During the Decade 1890–1899, Syracuse, NY

Year	Consumption	Pneumonia	Typhoid Fever	Diphtheria	Scarlet Fever	Cholera Infantum
1890	201	111	29	37	11	119
1891	228	104	40	18	2	123
1892	247	122	33	52	1	103
1893	234	152	34	97	9	92
1894	220	109	35	54	92	92
1895	180	80	31	22	19	92
1896	181	115	78	33	24	71
1897	346	155	24	21	24	72
1898	253	134	47	41	13	104
1899	136	71	20	43	18	91

Source: Annual Reports of the Bureau of Health, Syracuse, NY, 1899, p 30.

program, and the attempt to do something about the "lungers" at the county home, the selection of Onondaga Hill would seem to have been a logical choice.

THE TUBERCULOSIS CONTROL PROGRAM AND THE ONONDAGA SANATORIUM

The county appropriated $37,000 for land and $174,000 for the construction of a 150-bed tuberculosis hospital. The "Special Tuberculosis Hospital Committee" was directed to procure plans and consult with Taylor and Bonta, architects from Syracuse who had been associated with Scopes and Feustmann, architects of Saranac Lake.[11] Plans were prepared for the proposed tuberculosis hospital.[14] Three years passed before the sanatorium opened; the cost mounted to $600,000, and only 78 beds were ready. In October 1916 the first patients were admitted, and within a year 108 beds occupied the space planned for 78 and "many more were seeking admission who could not be received."[14-16] One who witnessed the official opening on September 2, 1916, declared that, "It was an auspicious occasion with a delegation of County Supervisors, State Officials, prominent Doctors and citizens, and members of the press forming a parade from the Court House."[17] (The magnificent new courthouse in downtown Syracuse, the fourth and present structure, had been dedicated December 30, 1906.)

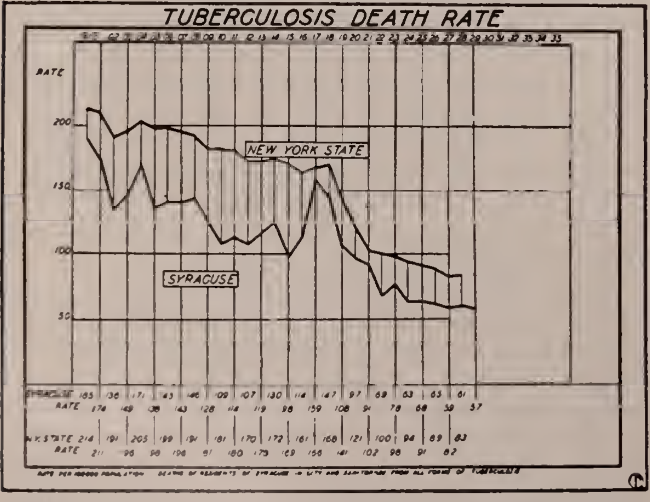

FIGURE 1. Tuberculosis death rate, New York State and Syracuse, 1900–1929. "The considerable decrease in deaths from tuberculosis in Syracuse in 1929 is due to enlargement of the sanatorium." Annual Report of the Department of Health, Syracuse, NY, 1929, p 17.

There were two sanatorium buildings, a powerhouse with steam generator and laundry, water and sewer mains, a water tower, an underground service tunnel, and access roads, all located on the 137-acre tract just outside the city. Some dairy products, meat, eggs and poultry, and other food products were still supplied by the nearby county farm when the sanatorium closed in 1959.

The x-ray equipment, known as a "Campbell machine," operated on DC current and required 2.5 seconds' exposure at a focal distance of three feet. Glass plates were used, for which storage was a problem. An account of one of the early x-ray technicians has been preserved.[15] This was not the first x-ray used for the purpose; Dr H. Burton Doust had been using x-rays in the tuberculosis clinic since 1909.[17] Dr Harry J. Brayton was appointed superintendent of the new sanatorium.

Besides Dr Brayton, who was joined by Dr Vernon M. Parkinson, the staff comprised two registered nurses and two practical nurses. (The first class of nurses in central New York graduated from the Hospital of the Good Shepherd in Syracuse in 1889.) There was a matron, who also supervised the kitchen. There was no laboratory technician at the sanatorium, laboratory tests being performed by one of the doctors. Patients, when well enough, were trained to do their own urine and sputum tests.[15] Dr William H. May was the city bacteriologist (from the annual reports of the Syracuse Bureau of Health).

Energetic programs of bovine tuberculin testing were carried on by F. E. Englehardt, PhD, who reported inspection of 4,126 cattle on 230 farms in 1900 with 1.26% positive tuberculin reactors, a reduction from 5.20% the year before. He made this statement in 1902: "There is unquestionably no city in the United States where the tuberculin test has been so thoroughly enforced for four years as in Syracuse."[8(p51)] In 1908 Dr Joseph C. Palmer, Medical Inspector of Public Schools, reported nine tubercular children, three with active bone and joint disease.[9(p60)]

In 1917 Dr H. Burton Doust joined the United States Medical Corps. Dr Frederich H. Knoff took over the tuberculosis clinic. The influenza epidemic struck. Draftees were then being examined and some were found to have tuberculosis.[18-20] In 1918 a women's auxiliary to the sanatorium was formed, the first in the county, and by 1945 it was one of the largest in the state.[21] The following year (1919) the Onondaga County Tuberculosis and Health Association was founded. Drs Burrows, Doust, and Totman pressed for programs for prevention and treatment of

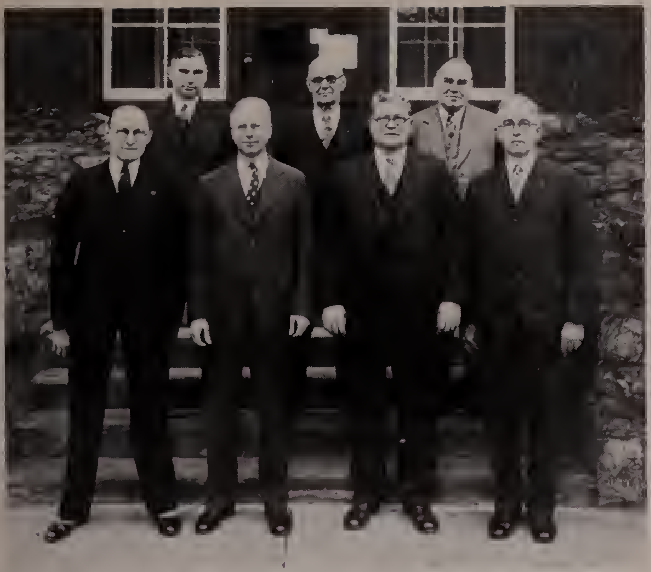

FIGURE 2. Dr Harry J. Brayton and the Board of Managers. Front row, l to r: B. B. Given, H. Burton Doust, E. L. Edgerton, and Dr Brayton. Back row: Brooks McCuen, Gardner Chamberlain, and Edwin A. Kaye (photograph by Doust Studio, ca. 1925).

childhood tuberculosis. As early as 1911, through the assistance of the anti-tuberculosis league, milk and eggs were distributed to needy families free of charge.[22] Until the Onondaga Sanatorium opened, Dr Doust sent to Ray Brook Tuberculosis Hospital as many infectious cases of tuberculosis as could be accepted there (Syracuse Bureau of Health reports, 1908–1916, Tuberculosis Clinic Reports). A Children's Preventorium was established at Camp Hillcrest. In 1920 the Children's Pavilion was constructed at the sanatorium. A school program for the Children's Pavilion was initiated through the Syracuse School District, Mr Willard W. Cooley being the first teacher.

Dr Brayton and the board of managers (Fig 2) worked with the board of supervisors and other agencies to improve the sanatorium. In 1921 over 1,200 evergreen and hardwood seedlings were set out on the grounds, thus developing the grounds into a park-like setting, in contrast with the barren hilltop shown in early photographs. In 1929 a new infirmary building was opened (Fig 3). This brought the total bed capacity of the sanatorium to 255, making it the third largest such institution in upstate New York. The new building provided individual rooms, some with access to the open porches (Fig 4). It had a new laboratory, pharmacy, dental and outpatient clinics, a small operating suite, a new x-ray department, and space for a new, expanded occupational therapy department (Fig 5). A new chapel was provided for worship, leaving the former auditorium for entertainment and classes.

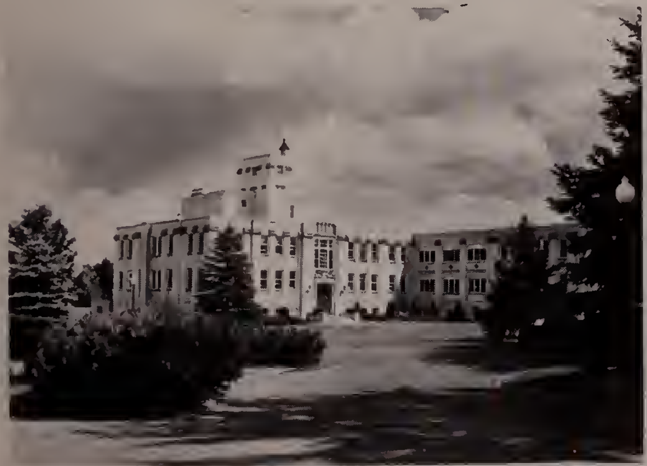

FIGURE 3. Onondaga Sanatorium for the Treatment of Tuberculosis, Infirmary Building, opened 1929 (photograph ca. 1955).

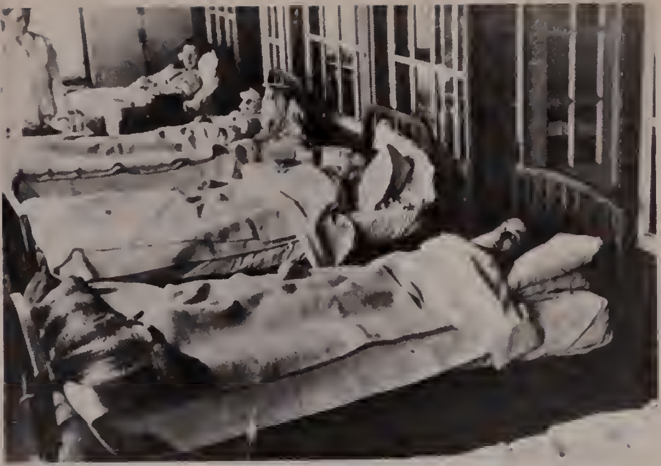

FIGURE 4. Open porch, 1942. Standing, upper left, Mrs Ruth Donnelly, RN. Mrs Marion Borsi was Superintendent of Nurses (photograph taken with consent of the patients, reproduced courtesty of Illustration Services, Community General Hospital, Syracuse, NY).

Principles of treatment included isolation of active cases, heliotherapy, and fresh air, good nutrition, and strict bed rest on open porches. In a recent conversation with an octogenarian, this was related: "It was pretty monotonous and boring. In the winter, snow would sift over my blankets and my breath would be frosty. In the daytime you might be allowed up in a lounge chair to read." Reference is also made to the accounts of Thomas Conway and Howard Straub.[15,16]

A typical course of therapy in the sanatorium "for the cure" lasted for months. In 1940, Dr Brayton reported an average stay of 19 months.[23(p9)] By 1944 the average stay was reduced to nine months.[24(p7)] With improvement, as gauged by such signs as reduction in fever and pulse rate, cessation of hemoptysis, thinning of sputum and reduction

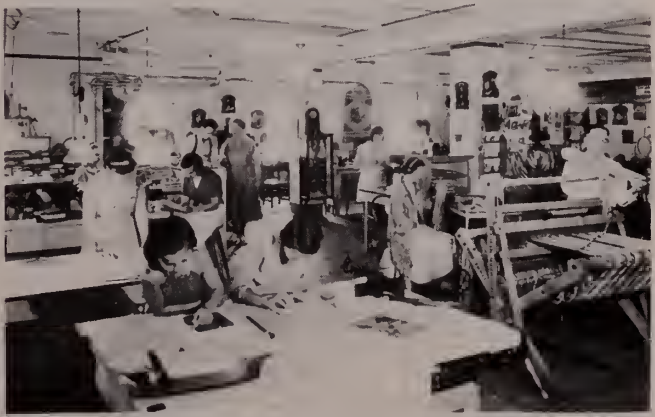

FIGURE 5. Scene in the Occupational Therapy Department, Onondaga Sanatorium for the Treatment of Tuberculosis, ca. 1933.

in bacillary count, drop in sedimentation rate, and improving chest x-ray, gradually increasing activity would be instituted, with bathroom privileges, then dining room privileges, and progression to graduated out-of-door exercises and some work assignments.

Mr Willard W. Cooley's education program involved individual tutoring as well as classes, some older patients taking advantage of the education offered to qualify for diplomas. As reported by Dr Brayton in the November 1924 issue of *The Temp Stick*, the sanatorium publication, an open-air school was attended "by over forty."[14] An October 30, 1941, newspaper featured a picture of a nine-year-old child on a hyperextension frame for treatment of tuberculosis of the spine (Pott's Disease). The child was out of doors in the sun, exposed almost fully, and being tutored by Mr Cooley[25] (Fig 6).

By the 1930s, the use of artificial pneumothorax became an accepted method of treatment (Fig 7) and certain adjuncts were introduced to supplement collapse therapy: intrapleural pneumolysis of adhesions, artificial pneumoperitoneum and/or phrenic nerve resection to elevate and/or paralyze the diaphragm.[26] Each time before "refills," fluoroscopy was repeated to determine the degree of lung collapse, subjecting patient and physician to heavy doses of radiation and the hazard of electric shock. A number of physicians of that era incurred permanent radiation burns. With direct current, polarity had to be corrected or else there would be hissing and sparking in the fluoroscopy room.[15(pp5-9)]

There ensued a decline in the death rate from tuberculosis in the late 1880s, which continued into the 20th century (Fig 1). Tuberculous enteritis had become a rarity, though the work of Wilensky and others raised questions about the etiology of a certain granuloma of the intestine as yet unknown.[27-30] Dr Edgar M. Medlar, in his necropsy studies in New York City in the mid-1940s, found that tuberculous infection was present in 35% of persons ten to 19 years of age, 65% in persons aged 30 to 39, and more than 85% in persons above age 60. In his comparative study of 17,196 necropsy protocols, he concluded that the incidence of tuberculous lesions was approximately the same from 1940 to 1945 as it was from 1916 to 1920.[31]

FIGURE 6. Willard W. Cooley tutoring a nine-year-old child with Pott's disease being treated on an extension frame in the Children's Pavilion at Onondaga Sanatorium. Photograph was taken in 1941 when 78 patients, ranging in age from six years to 50 years, were enrolled (reprinted with permission from *Syracuse-Post Standard*[25]).

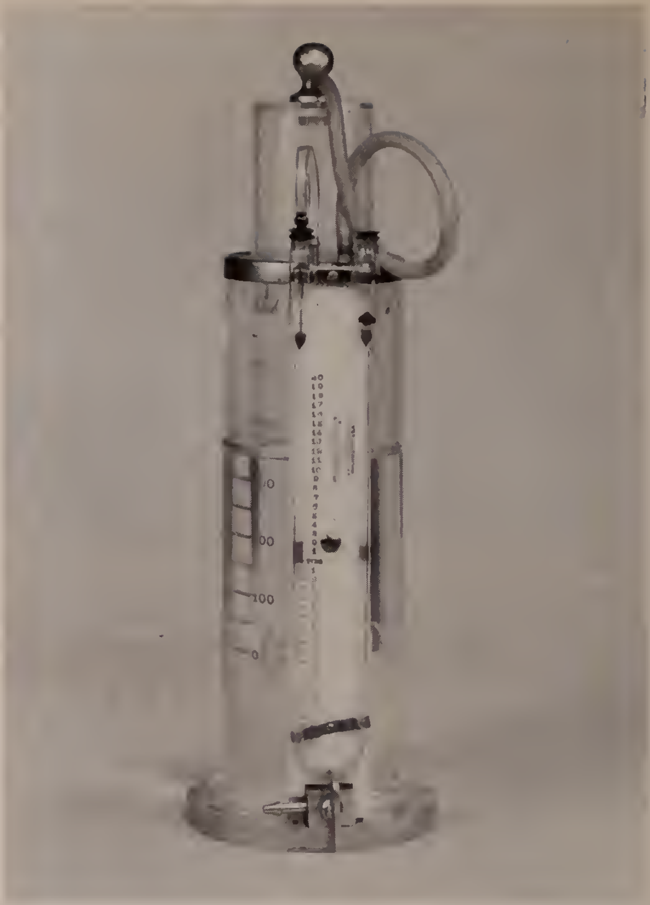

FIGURE 7. Dr Norman Bethune's Pneumo Apparatus. Made by G. P. Pilling & Son Co, Philadelphia. Loaned by Ronald A. Miller, MD. Inner cylinder suspended in water, graduated in centimeters; mercury manometer recorded intrathoracic pressure (photograph: Medical Illustration Services, Community General Hospital, Syracuse, NY).

State mental hospitals, when surveyed for tuberculosis in 1942, were found to have an incidence rate varying between 3.7% and 8.4%, and a mortality rate of 600/100,000 as compared with 50/100,000 in New York State. Sixty percent of patients with dementia praecox died of tuberculosis.[32] As late as 1952, Dr Medlar warned of the "undetected seedbed of tuberculosis."[33]

Dr Edward E. Godfrey, Jr, who was New York State Commissioner of Health from 1930 to 1947, had the following statement printed on his official letterhead: "Within Natural Limitations Any Community Can Determine Its Own Death Rate." Such a prediction, shared by few public health workers of his time, began to be realized when progressive urbanization permitted concentration of forces, both official and private, to watch over community health.[34] A growing awareness of sanitation, pasteurization of milk supplies, eradication of tubercular herds,[8(p51)] and more wholesome nutrition[22] were among the factors contributing to a lowering death rate. Extrapulmonary tuberculosis and childhood tuberculosis diminished with the widespread adoption in veterinary medicine of the Mantoux test[8(p51)] and the slaughtering of tubercular cattle.

It had long been known that bovine tuberculosis could be transmitted to man. Efforts to control bovine tuberculosis took two different courses. Von Behring's work was quoted in which he claimed to be able to immunize cattle and protect them against tuberculosis.[35] Uhlenhuth et al,[36] however, reported no positive results with attempts to immunize cattle with large doses of attenuated bovine bacilli. Consequently such efforts were abandoned in this country, and tubercular cattle were destroyed.

Initially known as the phenomenon of Koch, the hypersensitivity reaction to tuberculin in tuberculous animals and humans was the most extensively investigated phenomenon of allergy to bacteria and bacterial products.[37] Several methods for testing were utilized, but the intracutaneous method proposed by Mantoux in 1908 was finally universally adopted and methods for production of old tuberculin (OT) standardized. In the 1930s, Dr Florence B. Seibert[38] of the Phipps Institute in Philadelphia developed a purified protein derivative (PPD) from culture filtrates of the human strain of *Mycobacterium tuberculosis*, which eventually replaced OT to a large extent. Her method became the standard for the production of PPD for other mycobacteria, valuable in diagnostic testing in instances of infection with atypical mycobacteria.

Although attempts to raise the natural resistance of animals against tubercle bacilli were tried[35] and abandoned,[36] they did reveal information of an immunologic nature. In 1908, Bacillus of Calmette and Guerin (BCG),[39,40] an attenuated bovine tubercle bacillus carried through many successive generations of culturing until virulence was lost, was shown to be a reliable method of producing some degree of acquired specific immunity. It was first used in this country around 1920. Dr Brayton introduced its use at Onondaga Sanatorium, vaccinating medical and nursing students (Fig 8).

State orthopedic clinics were established and, until 1930, skeletal tuberculosis was treated in central New York by nonoperative methods. In 1935, operations were performed in central New York for patients with skeletal tuberculosis, first at nearby Jefferson County Sanatorium, then in 1943, at Onondaga Sanatorium.[41]

PULMONARY SURGERY

In 1940, thoracic surgeons began consulting at Onondaga Sanatorium. Ethan Flagg Butler, known for his work treating chest casualties and empyema during World War I, and for his continuing work in chest surgery, was one of the first such consultants.[42] Thoracoplasty had been introduced for permanently collapsing a tuberculous segment of infected lung, and by the late 1940s pulmonary resectional surgery was first undertaken.[43] The tuberculosis hospitals offered an early training arena for developing the specialty of thoracic surgery. Dr Medlar, along with thoracic surgeons, reported on excisional surgery.[44] In the 24th annual report of Onondaga Sanatorium in 1940, Dr Eugene W. Bogardus reported: "Plans had been under way for the surgical treatment of certain patients through the facilities of the State Tuberculosis Hospital at Ithaca, New York. The total cost per patient per day for such service is $2.50."[23(p17)]

Dr Harry J. Brayton, the first superintendent of Onondaga Sanatorium, went abroad in 1937, crossing the Atlantic to Europe and England on the dirigible *von Hindenberg*, returning on the *Queen Mary*. Soon thereafter his health failed, and more responsibility fell to Dr Anthony Gandia, who was appointed acting superintendent. After Dr Brayton's death in 1939, Dr Eugene Bogardus was appointed superintendent. Bogardus was a native of Dewitt, NY, educated in Syracuse and a graduate of the Syracuse University College of Medicine. He had previously been with the Westchester County Department of Health and the New York State Department· of Health.[23(p3)] He served as superintendent for four years before resigning to become head of the medical department of *Reader's Digest* in Pleasantville, NY.[24(p3)]

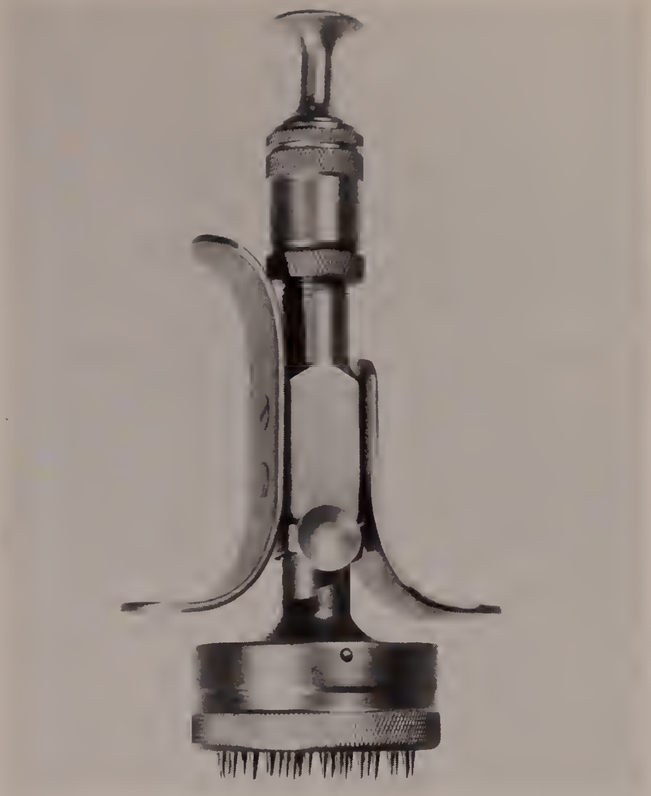

FIGURE 8. BCG "gun" used for vaccination. Loaned by Ronald A. Miller, MD (photograph: Illustration Services, Community General Hospital, Syracuse, NY).

Syracuse was recognized as a leading community in public health. In the spring of 1922, the Milbank Memorial Fund financed programs for health administration in New York State. One was in a rural county (Cattaraugus) and one in a large metropolitan area (New York City). Syracuse was chosen as the site for its study of a medium-sized city. In Syracuse, the demonstration study began in January 1923 and continued through 1930. The fund spent $683,335.00 on this demonstration in Syracuse. Chapter VII in C. E. A. Winslow's account of the demonstration study deals with tuberculosis.[45] During that time the appropriation for the health department nearly doubled, and more than trebled for school health services. The position of health officer became full-time; Dr George C. Ruhland became the health commissioner in 1928. Among leaders in the medical community at that time were Drs O. W. H. Mitchell, H. B. Doust, A. C. Silverman, O. D. Chapman, and H. G. Weiskotten. Syracuse had a recognized laboratory service, and public health nursing was established with Miss Agnes J. Martin as Director.

Upon the resignation of Dr Bogardus, Dr Bernard T. Brown, another native of Onondaga County and a graduate of Syracuse University School of Medicine who, like many victims of the disease, had become interested in pulmonary problems and tuberculosis, was appointed superintendent. He served in that capacity from 1944 until the sanatorium closed.[24(p16)]

THE ONONDAGA SANATORIUM BECOMES A STATE TUBERCULOSIS HOSPITAL

One of Brown's first reports to the board of managers in 1944 referring to the war years stated that the preceding year had been difficult in the field of hospital management, but that the sanatorium had been fortunate in securing the services of Dr Clara Hale Gregory[24(p16)] (Fig 9). Brown and the sanatorium's board of managers became convinced that it would be best for the state to take over the sanatorium. The local health department and county board of supervisors worked out the transfer[24(p4)] and on April 1, 1948, the sanatorium became a state tuberculosis hospital. New York State assumed responsibility for the annual budget and the costs of upgrading the facilities, with the provision that when and if they were no longer used for the care and treatment of tuberculosis, the whole should revert to the county.

Plans were made for expanding the infirmary (Fig 5) to include a surgical unit. For reasons that will later become obvious, such plans never were implemented. Two new staff homes were built on the grounds. The medical staff was enlarged, and teaching programs were augmented in connection with the College of Medicine at Syracuse University and the new Veterans Administration Hospital. The nursing staff was expanded under the leadership of Mabel Liscombe followed by Beatrice Latremore. Dr Brown was made acting director until appointed director through the state civil service.

THE EFFECT OF THE WAR

Disabilities among Civil War military personnel resulted in the building of such places as the Bath Hospital in Steuben County and Mt McGregor Hospital in Saratoga County, the latter eventually being taken over by the Metropolitan Insurance Company. Following World War II, veterans administration hospitals were opened at many of the medical centers including Syracuse.

A review of health department records concerning World War II recruitment reveals a 2% rejection rate for conditions of the heart and lungs. As a result, several young men were admitted to the Onondaga Sanatorium, one of whom having been found to have minimal tuberculosis of the lungs was soon put to work because of the shortage of staff. Military and government services recruited medical, nursing, and technical personnel, and one of the first losses for the Onondaga Sanatorium occurred when Miss Mary Drescher, its first full-time laboratory technician, left for military service.[16]

After the war New York State tuberculosis hospitals and sanatoria, along with medical services of the armed forces and veterans administration hospitals and the support of six pharmaceutical companies, entered into cooperative studies and research programs.[46] The Onondaga State Tuberculosis Sanatorium, being near the medical center and the veterans hospital, was in a favorable situation to participate in the early studies of new agents being tested for their effectiveness in the treatment of tuberculosis.[47-49]

FIGURE 9. Clara Hale Gregory. Photograph taken while she was a member of the medical staff of Onondaga Sanatorium, 1944–1959 (photograph by C. Basta).

ANTITUBERCULOSIS DRUG THERAPY

Trudeau and his associates had experimented with antitoxin serum.[50] The capacity of certain saprophytic organisms to inhibit the growth of *Mycobacterium tuberculosis* had been recognized.[46(pp7-13)] In the long search for an effective agent in the treatment of tuberculosis, the discovery of streptomycin by a team headed by Waksman at Rutgers was the most encouraging. Working in the Department of Soil Microbiology at the New Jersey Agriculture Experimental Station of Rutgers University, Waksman found that cultures of soil-inhabiting organisms named *Streptomyces griseus* inhibited the growth of certain bacteria.[51] Clinical studies by Hinshaw and Feldman at the Mayo Clinic and McDermott and Muschenheim at Cornell University Medical College in New York supported the conclusion that streptomycin could be used in the treatment of human tuberculosis.[46(p545)]

Problems arose such as the emergence of drug-resistant organisms, increasing frequency of atypical strains of mycobacteria, and the development of toxicity to the new antituberculosis drugs.[47] Then there were administrative problems such as budgets, and patients' noncompliance with the various regimens of treatment. Workers in animal husbandry, veterinary medicine, and pathology (Dr William H. Feldman of the Mayo Clinic and the University of Minnesota, for example, was a Doctor of Veterinary Medicine) cooperated with workers in human medicine and pathology and shared information.

FORMATION OF NATIONAL HEALTH AND EDUCATION ORGANIZATIONS

The campaign against tuberculosis at the turn of the century was confined to relatively few professionals. Public health professionalism itself was invented out of a need for health education, and health organizations from the need for coordinated effort. The American Public Health Association had met for its first meeting in 1872.[52(p248)] By the last quarter of the 19th century, a sound basis had been created for further development of public health in this country. The term "health education" was first officially adopted in 1919, and by 1922 workers in public health agencies concerned with education formed a separate section of the American Public Health Association.[52(pp395-396)] With respect to tuberculosis, there was a growing realization that public and professional education was vital in any effort to conquer the "white plague." Onondaga Sanatorium entered into educational programs with the medical college, as noted by Dr Bogardus in his 1940 annual report: "The Sanatorium has continued to give instruction to medical students concerning various types of chest conditions."[23(p18)]

The American Sanatorium Association, later named the American Trudeau Society, was started in 1902. The National Association for the Study and Prevention of Tuberculosis, which later became the National Tuberculosis Association, was formed in 1904. Its first president was Dr Edward L. Trudeau, and among its founders were the illustrious William Osler, Hermann M. Biggs, Lawrence F. Flick, Adolphus Knopf, William H. Welch, and lay members, including Homer Folks and Samuel Gompers.[52(p389)]

At the turn of the century Dr Totman's first attempts at public health education had been directed toward the county legislative body.[10] Afterwards, through the efforts of Drs Totman, Brayton, and Doust, and interested citizens of the antituberculosis league,[22] the Tuberculosis and Health Association of Onondaga County was founded in 1919.

As time went by, a number of young physicians joined the staff to gain experience in working with tuberculosis. Physicians serving as medical missionaries and others from overseas became members of the staff for shorter or longer terms. In cooperation with Dean Edith H. Smith of Syracuse University School of Nursing, a teaching syllabus for nurses was developed.[53] With the opening of the Syracuse VA Hospital and its tuberculosis unit, medical students, interns, and residents received their tuberculosis training there, and one of the physicians of the sanatorium was appointed attending physician.

Dr Brown developed a series of talks for a closed circuit radio program for patients and visiting family members[24(p16)] which, preserved in the sanatorium's archives, gives a running account of activities. In 1949 he told his radio audience: "Dr. Beck arrived from Biggs Hospital, and after lunch Dr. Brown and Dr. Beck left by car for Ray Brook, where the interhospital conference will take place. We arrived in Saranac Lake at 7:00 PM, ate dinner at Tuffy's Grill, then proceeded to Ray Brook. Dr. Medlar was there ahead of us. We reported our arrival to Dr. Bray. . . ." On May 24, 1949, he reported, "Dr. Lincoln of Ithaca called and stated they had x-rayed a child in Cortland whose mother (E.L.) is now a patient in Onondaga Sanatorium, and found that this child has a miliary type of tuberculosis. He agreed to hospitalize the child at Biggs Hospital in Ithaca."

THE SANATORIUM AND THE COMMUNITY

The Onondaga Sanatorium, although located on the edge of the city, was also situated in a large agricultural region. Outpatient clinics were operated in 21 villages throughout the county.[23(p18)] In addition to bovine tuberculosis, other diseases of the farm were recognized. Skin-test antigens including histoplasmin, coccidioidin and blastomycin became available for cases showing x-ray evidence of granulomatous disease when evidence of tuberculosis was lacking. Interest in nontuberculous cases developed and resulted in a member of the staff's eventually participating in a symposium at the College of Medicine concerning "farmer's lung,"[54] which attracted participants from a number of states. The so-called atypical mycobacteria came to light as a result of more refined culture techniques. In the last 15 months of its operation, five patients at Onondaga Sanatorium were found to have infections caused by atypical organisms.[55,56]

The sanatorium was within a short distance of the Onondaga Indian Reservation. A number of American Indians were admitted. Interest was created concerning the natural history of diseases among American Indians, particularly tuberculosis.[1,57] The problem of the incidence of alcoholism among patients with tuberculosis, often leading to both legal and public health complications, led to the formation of a chapter of Alcoholics Anonymous. Dr Brown's radio script of June 24, 1949, includes a note on the subject, "Alcoholism in Relation to Tuberculosis." Occasionally a tuberculous inmate of the local penitentiary would have to be admitted to Onondaga Sanatorium. On several occasions the matter was discussed with the hospital directors by members of the state health department. Dr Brown was a member of the Advisory Committee on Alcoholism and Rehabilitation of the County, copies of minutes of which are in the sanatorium archives. One of the medical staff became a board member of the Rescue Mission, an interfaith agency founded in 1886 whose purpose was to minister to the needs of the less fortunate, among whom were alcoholics.

Training the handicapped to return to normal employment in industry led to the formation of the Onondaga Workshop for the Handicapped, Inc, an independent agency operated under a local board of directors with the advice and guidance of the Onondaga Health Association, the Community Chest, and the Easter Seal Organization. Dr Brown was one of the original sponsors, and copies of that organization's minutes are in the sanatorium archives.

THE SANATORIUM'S CLOSING

During the sanatorium's 43 years of operation (1916–1959), the incidence and death rate from tuberculosis fell. By 1940 the incidence of childhood tuberculous disease was sufficiently reduced to allow closing the Children's Pavilion.[23(p17)] By the late 1950s the census in the state tuberculosis hospitals declined. The total capacity was 4,022, but the average daily occupancy during the year April 1956–March 1957 was 2,996 (73%). The Hermann M. Biggs Hospital in Ithaca was the first to close, and in November 1957 hearings began concerning the closing of Onondaga Sanatorium. It was apparent that its days of usefulness were past. The treatment of the disease no longer required long periods of hospitalization. Case finding was a function of health departments, and outpatient clinics were adequate for follow-up. Antituberculosis drug therapy was generally effective in rendering active cases noninfectious. Thoracic surgery, no longer frequently indicated, was being done in general hospitals.

When Dr Brown started the task of closing the sanatorium, 170 patients and 200 employees remained. Those patients needing further hospital care were transferred to other state tuberculosis hospitals: Mt Morris in Livingston County, Homer Folks Hospital in Otsego County, and Ray Brook in Essex County. Some employees transferred to county employment, some to other state hospitals, and some to private employment. Dr Brown and his successor continued to operate a tuberculosis clinic for the state health department, which covered Cortland, Cayuga, and Onondaga counties, until 1968 when its function was taken over by the Syracuse Tuberculosis Clinic of the Onondaga County Health Department.[58]

In July 1958 the Onondaga County Board of Supervisors passed a resolution to apply to the State Commissioner of Health for reconveyance of the sanatorium lands to Onondaga County, of which 43 acres of forest land was to be deeded to the newly formed Community Hospital Corporation. Buildings and grounds were returned to the County of Onondaga in accordance with resolution number 194 of the board of supervisors.[59,60]

In May 1959 the Onondaga Sanatorium closed its doors as a state-operated tuberculosis hospital, the last of the patients having been transferred or discharged. The property remained under state jurisdiction for three months, leaving a sufficient labor force to inventory equipment and transfer records and maintain the facility. Medical and other confidential records and reports were transferred to other facilities; active medical records followed the patients, outpatient records remained with the Onondaga State Chest Clinic.

Continuity of outpatient services was maintained by Dr Brown, a small office force, two x-ray technicians, and an administrator. Equipment for operating the clinic as well as field clinics was retained. It remained for his successor to direct the clinic from 1963 to 1968. Final disposition of pertinent records and equipment was carried out in accordance with directives from the state health department.[58,61] Such records as are considered of archival value and not confidential in nature come under the jurisdiction of the Public Officers Law and permission from the state education department is required for disposition.[62,63] Patient records and x-ray files and equipment were turned over to the Syracuse Bureau of Tuberculosis, which is a continuum of the tuberculosis clinic opened in 1908.[9(p36)]

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FROM THE LIBRARY

THE UNDETECTED SEEDBED IN TUBERCULOSIS

Discussion. Foster Murray, M.D., *Brooklyn.*—Almost thirty-five years ago, Dr. Lawrason Brown of Saranac Lake coined the term "a good chronic." He was referring to the old chronic fibrocavernous case with constant or intermittent positive sputum who, in spite of extensive damage to the lung, lived on for many years. The term, "a good chronic," was intended as a prognostic one and was applied to the individual. It certainly had no epidemiologic significance other than an adverse one. The outlook, while very good in the individual case, was bad from an epidemiologic viewpoint. Such a patient continued to live comfortably, and in some cases even happily, but was a source of continued infection of others and, as such, left a long trail of victims in his wake.

It is to this group that Dr. Medlar on the basis of a thorough and erudite study has so wisely directed our attention. It is in this group, patients over forty years of age preponderantly of the male sex, that there lies a vast reservoir of dynamic infection. Here are the individuals usually considered by their friends and even their physicians to be cases of chronic bronchitis, asthma, or bronchiectasis, or anything other than pulmonary tuberculosis. Little is ever done to explore deeper into the origin of their complaints. In the meantime they are infecting all with whom they come in contact. They are the analogs of "Typhoid Mary," the bacillus carrier.

Let us profit, therefore, by Dr. Medlar's timely warning and in our mass surveys direct more of our attention, if possible, to this particular age and sex, namely, the male over forty years of age. Let us also provide extended facilities for this patient group through the greater utilization of the domiciliary type of institution. The crying need here is not so much for therapy but for providing a place for the isolation of such cases, a place where such patients may live in reasonable comfort but out of contact with the world.

E.M. MEDLAR, MD
(*NY State J Med* 1952; 52:1987-1989)

Physician discipline and professional conduct

Daniel F. O'Keeffe, MD, Gerard L. Conway, JD

Perhaps more than at any other time in the history of medicine, today there are ever increasing calls in the United States for improvements in the system by which members of the medical profession are disciplined. The focus of public attention on professional medical conduct is not new but the intensity and, indeed, in many cases the stridency, of the exhortations calling for the development of a new and more efficient system have reached unprecedented levels.

The public attention that is being focused on physician discipline reflects the existence of active consumerism in American society. This heightened level of scrutiny, however, is also in part the result of an attempt by some to divert attention from the actual causes of the current liability insurance crisis that exists both in New York State and throughout the country.

The link between medical malpractice and physician discipline is not a new phenomenon. However, it is for the most part a misconception and in many instances an intentional distortion. There is an area of overlap between malpractice and discipline. However, there is no evidence that the current liability crisis has been caused by deficiencies in the discipline system. The consequence of erroneously assuming a relationship between the two is serious because it obfuscates the cause of the liability problem and will inevitably delay the implementation of the basic civil justice reforms necessary to restore equity. Quite simply, the charge of "inadequate discipline" is a "red herring" that is frequently made to thwart the efforts of those who are seriously working toward meaningful reform in the present legal system. Allegations of medical malpractice are often made against the most competent physicians, particularly those in high-risk specialties, while the true miscreant can quite possibly escape appropriate civil accountability. Medical liability is a separate and distinct issue from physician discipline and professional conduct. To understand the differences, one must first look at each system individually. This article focuses on the discipline system.

HISTORICAL PERSPECTIVE

It is useful to examine briefly the process of physician licensure, regulation, and discipline from a historical perspective. The earliest item of colonial legislation affecting physicians in New York State was chapter 1120 of the Colonial Law of 1760.[1] Prior to that the practice of medicine was virtually unregulated and, quite literally, anyone could practice medicine. With the enactment of chapter 1120, however, there appeared for the first time a requirement of an examination. It is rather interesting to note that the earliest examining authorities were men of "law" rather than medicine, including "His Majesty's Counsel, Supreme Court Judges, the King's Attorney General, and the Mayor of New York City."

Chapter 1120 did not include any specific educational requirements for qualification for the licensure examination, but in March 1792 the law was amended to require three years of preceptorship with a "reputable physician" or two years' preceptorship if one had earlier obtained a doctor of medicine degree.[2] By 1792 a medical graduate also had to complete successfully an examination, and pass an "evaluation" by three "reputable" physicians—in effect, the first Board of Medical Examiners in New York State.[2]

The early regulation of the profession was clearly focused on licensure and prelicensure preparation. The conduct of physicians once licensed was not given much attention, and in the early 19th century the law still lacked any procedures for disciplining physicians after they had been licensed. It was not until 1806 that the first significant steps were taken to address the issue of postlicensure professional conduct. In that year the state legislature allowed physicians to organize medical societies for the purpose of regulating the practice of medicine in the state.[3] The purpose was to insure, in the public interest, that unqualified individuals would not practice medicine. Both the state and county medical societies were empowered to license, with the state medical society having the power to override actions taken by a county society. Importantly, the role given to the profession involved oversight after admission and not just the formulation of the admission process itself. Once again, however, with respect to the enforcement of conduct and practice standards for existing licensees, the law was virtually devoid of penalties. Without sanctions, the law was quite difficult to enforce and rather easy to circumvent.

In 1827, an even greater recognition of a need for the centralized regulation of the medical profession came when the profession was given control of the practice of medicine in New York.[4] Licensing authority was exclu-

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sively vested in the state medical society when in 1849 even the right of a county medical society to license was revoked. With very few exceptions the law of 1806, with its recognition of the preeminent role of organized medicine, governed the licensing of physicians until almost the end of the 19th century.

A significant change occurred, however, with the new Medical Practice Act of 1890.[5] This law shifted ultimate power from the profession to the state and specifically to the Board of Regents. The regents were empowered to and did appoint three boards including the Medical Society of the State of New York, the Homeopathic Society, and the Eclectic Society. Thus, organized medicine remained involved but essentially a shift to more direct state control occurred. State licenses were required by the act and were issued only after examination by medical examiners. In 1891, as a result of this new law, the first formal system of state-controlled licensing and written examination became operative in New York State, and in the following year the Medical Society of the State of New York gave the first examination.[5,6] A requirement of five years of "reputable practice" in lieu of educational requirements was allowed in 1893. Once again, however, the 1890 law focused primarily on licensure and there were still no strong, formal disciplinary procedures.

THE ADVENT OF PHYSICIAN DISCIPLINE

It was not until well into the 20th century that there arose a system for physician discipline with real power that permitted the imposition of meaningful sanctions. In 1935 the education law was amended to give the Board of Regents broad power with respect to sanctions for misconduct.[7] Most of the penalties that exist today were enacted at that time. The procedures that were authorized in 1935 continued with relatively little change until the enactment of chapter 109 of the Laws of 1975, which embodied major system changes.*

The structure established at that time, a little more than ten years ago, continues to serve as the core of the system now operating in New York State. Perhaps the most visible change brought about was the establishment in 1975 of the State Board for Professional Medical Conduct within the Department of Health. This largely removed the conduct enforcement function, as opposed to that of initial examination and licensure, from the State Education Department. Today professional discipline for physicians is regulated primarily by section 230 of the Public Health Law.[9] The education law continues to embody the substantive dimension of professional misconduct. For example, it proscribes certain conduct deemed unprofessional, including practicing fraudulently with gross negligence on a particular occasion, or negligence on more than one occasion.

Obviously, practicing while one's ability is impaired by alcohol, drugs, or physical or mental disability is improper, as is willfully harassing, abusing, or intimidating a patient, making or filing a false report, or failing to file a report that is required by law. Ordering excessive tests or treatment and promoting the sale of services, goods, appliances, or drugs in a manner that exploits the patient are all also clearly delineated in the education law as improper practices. Abandoning or neglecting a patient in need of immediate care is clearly misconduct.

While standards continue to be set in the education law the process by which adherence to these standards is investigated and adjudicated and deviations punished is set forth in the Public Health Law. Section 230 of that law established the Office of Professional Medical Conduct (OPMC) and the State Board for Professional Medical Conduct as the principal enforcement arms of the state in this endeavor. This section authorizes the investigation of complaints of medical misconduct, and describes the procedures by which such investigations take place. In effect, the OPMC is charged with initially investigating all allegations of misconduct. If warranted, an investigative or screening committee of the State Board for Professional Medical Conduct becomes involved and reviews the staff report on the case. A medical consultant and OPMC staff are generally present for this review by a committee of the board. If the committee finds evidence of impending harm to patients it may recommend that the Commissioner of Health take summary action. Otherwise, if it finds cause it will recommend that OPMC formally prosecute the case in an administrative hearing or that it issue an administrative warning. If the Director of OPMC concurs with a recommendation for an administrative warning, the Executive Secretary of the Board of Professional Medical Conduct issues the warning, which remains confidential. If the director concurs with a recommendation to press charges, he or she will refer the matter to a Hearing Committee of the State Board for Professional Medical Conduct. The Board then appoints a committee to hear the formal charges of medical misconduct. An administrative law judge presides. The committee may recommend anything from dismissal of the charge to revocation of the physician's license. The Commissioner of Health then reviews the report and either concurs with its recommendations, makes further recommendations, or dismisses the case. Finally, the Education Department reenters the process and the Board of Regents reviews the commissioner's report and makes the final determination.

PHYSICIAN DISCIPLINE IN THE 1980s

Several changes in this process were enacted in July 1986.[9] The number of members on a Committee on Professional Misconduct was reduced to three and now consists of two physicians and one lay member. Additional due process protections for physicians were added and require that any licensee who is investigated by OPMC must be given a copy of the findings, conclusions, and recommendations of the investigating committee. This section also attempts to reduce the number of postponements that have been granted because lawyers have to appear in other courts. Thus, the law now specifically raises the status of an appearance before the Office of Professional Medical Conduct to a level equal to that of an appearance in state supreme court. The July 1986 law also specified those situations in which a physician's practice may be monitored by another physician. This monitoring can be initiated only after an investigation and review by the Director of the Office of Professional Medical Conduct in consultation with a Committee on Professional Medical Conduct and only where the committee finds that there is reason to believe that the licensee is unable to practice medicine with reasonable skill and safety to patients. County medical societies are given a role in the monitoring process.

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* That law sets forth *inter alia* provisions governing proceedings in cases of professional medical misconduct, including penalties. The law also vests the Department of Health with authority in investigating and adjudicating professional medical conduct under Article 2, Title II-A of the Public Health Law.

It is quite apparent from a review of the statutory provisions that govern physician professional conduct and discipline that the role of organized medicine in the process is quite minimal. Aside from the county society's monitoring role just mentioned, medical societies are involved only to the extent that they can make recommendations or "nominations" of members of the profession to serve where such members are required to be physicians. Thus, while physicians are involved and, indeed, rather extensively so, it is an involvement largely ancillary to an essentially governmental process. Further, it is an involvement which tends to be individualized and not organizational. One important exception is the Committee for Physicians' Health, wherein a committee of the Medical Society of the State of New York is expressly recognized and authorized in the law. This particular involvement is less punitive than it is rehabilitative. Its focus is to identify health problems that impair a physician's ability and, hopefully, to help the physician overcome his or her disability. On the whole, however, physician organizations are regularly criticized for not disciplining their members when the truth is that organizationally they have consistently been denied a meaningful participation in that function.

The law requires reporting to the state by physicians and by physician organizations where misconduct is reasonably suspected. However, any action involving or affecting licensure must be state action. In sum, where medical organizations once had genuine authority in the process, it is fair to say that now where organized medicine is mentioned in the law at all, it is almost always within the context of a "duty" of the society rather than a "power." Thus, for example, section 230 of the Public Health Law requires that if a medical society investigation reveals reasonable evidence of professional misconduct, there is an absolute duty of the society to report to the Office of Professional Medical Conduct.[9] If no investigation is initiated by the county society on a complaint it has received, it must advise a complainant that he or she may report directly to the Office of Professional Medical Conduct.

Although many believe the law to be ambiguous, the Department of Health has stated that the society must report a complaint to the Office of Professional Medical Conduct even if it does not investigate or after investigation finds no reasonable evidence of professional misconduct. Individual physicians also have the absolute duty to report professional misconduct under section 230 of the Public Health Law if the physician has reason to believe that professional misconduct may have occurred. If in doubt, the physician may utilize several options. The advice of the Board of Medical Examiners on the issue may be requested in writing. If advice is given, the physician must follow it. If the physician in question is a member of a hospital staff, referral to the appropriate committee of that hospital is an acceptable course even though the problem may have nothing to do with hospital activities. If the physician is a member of a county society, referral to the appropriate county medical society is an acceptable alternative. As noted earlier, there is a special exemption from this mandatory reporting requirement for the Medical Society of the State of New York's Committee for Physicians' Health. Deliberations of medical audit, tissue committees, and mortality and morbidity committees are also specifically exempt.

CONCLUSIONS

The present statutory discipline process minimizes the role of physicians by deemphasizing if not totally eliminating the organizational dimensions of the physician's participation. This deemphasis is a system deficiency. It is clear that peer review (physicians' review of physicians) is in fact far more extensive and real in terms of the day-to-day operational reality of medical practice than it is in any other profession. There are immeasurable medical review entities that operate within many medical practice contexts. Tissue committees, utilization review committees within public systems of insurance as well as private systems, quality assurance programs, morbidity and mortality committees, peer review within the context of our malpractice insurance programs, the role of physicians in the initial physician examination process, specialty accreditation evaluation, hospital privilege determination—these are just a few examples. Although it is obvious to even the most casual observer, it is worth repeating. The practitioners of medicine are reviewed more regularly and from more different perspectives than are the members of any other group or profession. Physicians themselves, furthermore, are willing participants in this process and indeed are at the very heart of it.

Physicians have repeatedly demonstrated that they will diligently and aggressively perform the peer review function while at the same time bringing to it an expertise and knowledge which enhances the quality of the process, which is clearly a benefit to society as a whole. Despite this record of achievement in the peer review process, organized medicine continues to be denied a truly meaningful role within the primary area of conduct control, namely the state's discipline system, and the status of a physician's professional license under the law of the state. Physicians must continue their efforts to achieve this role under our law. They have the resources and expertise to enhance the process of professional discipline immeasurably. Even more importantly, they have the responsibility to be involved in that process.

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Importance of testing for sexually transmitted chlamydial disease in the pediatric emergency room

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Chlamydia trachomatis is often reported as a sexually transmitted disease in adolescents and young adults.[1-5] It should also be considered a sexually transmitted disease (STD) when found in the anal or genital areas of children.[6-8] The following cases illustrate the importance of screening for chlamydial disease in adolescent or pediatric patients in whom any sexually transmitted disease is suspected.

CASE REPORTS

Case 1. A 9-year-old boy was brought to the emergency room by his mother. She had seen her other son, age 8, spitting and running out of the older child's room. On questioning, the mother discovered that the older boy had forced the younger one to perform fellatio. When the mother questioned the older sibling about his behavior, he revealed that his father had forced him into multiple acts of fellatio, and that his father had also committed rectal sodomy. The father's sexual advances had begun one year prior to the emergency room visit, and correlated with a deterioration in the boy's school performance and the onset of severe behavior problems at school.

A general physical examination was entirely normal, revealing no skin, mucous membrane, joint, perineal or perianal abnormalities. Cultures for gonorrhea were obtained from the throat, rectum, and urethra, and chlamydia fluorescent monoclonal antibody (Microtrak) slides were prepared from rectal and urethral samples. The child was then admitted to the hospital in order to assure his protection during a sexual abuse investigation. Child Protective Services had been notified before the patient left the emergency room.

The boy's behavior on the ward was markedly abnormal, in that he was continually acting out and was rebellious despite the best efforts of staff to control him. After the initial interview, he was not cooperative in talking further about the alleged sexual abuse. Gonorrhea cultures were negative. However, the chlamydia Microtrak test from the rectum was positive. Treatment was instituted with doxycycline, 100 mg orally twice a day, for seven days in the hospital. Thereafter, a repeated rectal Microtrak test was negative for chlamydia.

The child was transferred to a children's psychiatric ward for further evaluation and treatment. A warrant was issued for the father's arrest.

Case 2. A 14-year-old girl presented in the emergency room with a history of migratory arthralgia and arthritis. Three days prior to admission she first noted swelling and tenderness in her right wrist. These symptoms in the wrist diminished, but the following day, new tenderness followed by swelling and decreased range of motion were noted in the left wrist and elbow. Finally, on the day of admission, a severe, painful swelling of the right ankle occurred, coincidental with diminished symptoms in other affected joints. Because she could not walk without severe pain, she decided to seek medical attention. The patient reported no fever, chills, or sweats and had taken no medications.

On examination, the patient was afebrile with considerable distress due to her pain. There was limitation of motion and tenderness to palpation of the left elbow and of both wrists. Only mild swelling without redness or heat was detectable in those joints. The right ankle was grossly swollen, hot, reddened, and very tender, with decreased range of motion. The rest of the physical examination, including pharynx and abdomen, was within normal limits. There were no skin, conjunctival, or oropharyngeal abnormalities. A pelvic examination was within normal limits. Initially, only gonorrheal cultures were obtained in the emergency room.

Because of the suggestive symptomatology and a history of sexual activity, the admitting diagnosis was gonorrheal arthritis. The patient was therefore begun on intravenous penicillin and responded with a clearing of gross symptomatology within 48 hours. A vaginal examination was repeated on the second day of admission in order to obtain a cervical specimen for fluorescent monoclonal antibody testing for *C trachomatis* (Microtrak) and a wet mount for trichomonas.

No trichomonads, clue cells, or yeast hyphae were seen on the saline and potassium hydroxide slides. Blood and cervical cultures were negative for *Neisseria gonorrhoeae*, but the clinical course of rapid resolution supported the admitting diagnosis. The patient received seven days of intravenous penicillin therapy in the hospital. On the fifth hospital day, the chlamydia test was reported as positive. The patient was begun on doxycycline, 100 mg orally twice a day, for a seven-day course to be completed at home. A return visit for test of cure was scheduled for ten days following discharge; subsequent chlamydia testing was negative.

DISCUSSION

If physicians follow Centers for Disease Control recommendations, they will automatically overtreat for chlamydia in adolescents who present with clinical cervicitis, urethritis, pelvic inflammatory disease, or epididymitis.[9] Hopefully, clinicians also try to document these infections, but it may be argued that treatment is sufficient. However, these two cases demonstrate the importance of extending chlamydial testing.

We believe that in all alleged or suspected rape or sexual abuse cases, chlamydial testing should be mandatory. Sites for testing should include the vagina in female children with a Sexual Maturity Rating (SMR-Tanner Stage) of 1, and the cervix in adolescent females with SMR 2 or greater. In addition, rectal testing should be done in both male and female children.

The forms of testing that may be done vary with circumstances and test availability. Given a choice, the "gold standard" of chlamydial culture is to be

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preferred. However, if chlamydial culture is not readily available, then immunologic tests, which are now readily available, should be adequate, as long as they are performed by experienced technicians.

For the past two years, we have made extensive use of the Microtrak fluorescent monoclonal antibody slide test for *C trachomatis* (Syva Corp, Palo Alto, CA). A cervical smear is taken, in a fashion similar to a Pap smear, and fixed with acetone. The slides can then be delivered to any pathological lab facility that has a fluorescent microscope for staining and reading. The clinician must have confidence in the technicians or pathologists who read Microtrak slides, particularly when rectal samples are taken, since there are cross-fluorescing reactions with staphylococci, micrococci, and certain gonorrheal subforms. We believe an experienced viewer, however, can readily identify these false positives.[10-15]

Because there is evidence that the fluorescent monoclonal antibody test (Microtrak) is positive in some cases when chlamydial culture is negative, and vice-versa,[11-16] we recommend that both cultures and slide testing for chlamydia be done in cases of sexual abuse, if both tests are obtainable.

An alternative testing method, which is becoming more common and may eventually replace Microtrak in offices and emergency rooms because of ease of performance, is the enzyme-linked immunosorbent assay (ELISA). Current varieties (eg, Chlamydiazyme, Abbott Labs, Chicago, IL) take four hours for color change and require special equipment, but more rapid tests, akin to current color-change tests for streptococcus (eg, Vivid, Seradyn Inc, Indianapolis, IN) are just now becoming available. Both immunologic tests are very sensitive and specific in comparison to cultures in most studies, with positive predictive values in excess of 85% and negative predictive values in excess of 95%.[11-16]

Chlamydia can be contracted in the rectum and vagina at birth,[17,18] but it is not known at present how long it can be carried asymptomatically. Current evidence suggests that the *C trachomatis* colonization contracted in the newborn period ends by age 1½ years.[17] However, there is evidence that chlamydia may be carried as a commensal organism in the vagina for at least three years when acquired later in childhood or at birth.[18,19] A long-term chlamydial vaginal carrier state as a result of neonatal infection is rare in comparison to transmission of the organism to older children as a result of sexual abuse. Therefore, unless further research indicates otherwise, *Chlamydia trachomatis* should be considered a sexually transmitted disease if found in the rectum, vagina, or urethra of children beyond the age of 1½ years, and should be presumed to be evidence of sexual abuse until proven otherwise.[6]

The finding of chlamydia in the rectum in case 1 served as significant corroborating evidence of sexual abuse. If gonorrheal cultures alone had been done, this evidence would not have been forthcoming, while the patient would have been at unknown future risk from the rectal chlamydial infection.

Similarly, in case 2, the typical history and response to penicillin established the diagnosis of disseminated gonorrhea, despite negative gonorrheal cultures.[20] However, had chlamydial testing not been done, the unwary clinician might have failed to treat for that organism, which is not eradicated by penicillin. The patient would subsequently have been at risk for spreading this sexually transmitted disease and for developing pelvic inflammatory disease and its serious sequelae.[21-23] In addition, the definite proof of an STD provided by a positive test for chlamydia was helpful in convincing the patient of the seriousness of her situation.

Chlamydial testing should certainly be done on any patient in whom the possibility of gonorrheal disease is entertained for any reason, since the organisms are found together in up to 30% of cases tested.[24] Chlamydial testing should be done routinely, we believe, in every sexually active adolescent female who receives a pelvic examination in the emergency room or physician's office. Even the asymptomatic cervical carrier rate is high enough in all segments of the general population (5–25%) to justify routine screening.[1-5] Failure to test for chlamydia with either a culture of the cervix, Microtrak cervical smear, or chlamydia ELISA test may constitute a grave clinical error. In fact, we believe that chlamydial screening must join gonorrheal testing as a routine part of the periodic examination of all sexually active females, and that chlamydial infections should become a reportable disease in New York State. Unless all physicians are more aggressive in finding and following up cases and possible contacts, there is little hope of ending the current chlamydial epidemic among young people, with its resulting huge costs from pelvic disease and infertility.[21-23]

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Bilateral lipomas of the diaphragm

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Lipomas of the diaphragm are extremely rare entities which, in the past, were often difficult to classify based on either clinical or roentgenographic information.[1-3] We report a case of bilateral tumors, both of which were identified serendipitously on an upper gastrointestinal series with fluoroscopy and then confirmed and diagnosed on computed tomographic (CT) scans. To our knowledge, the combination of these fluoroscopic and CT findings has not been previously documented.

CASE REPORT

A 43-year-old man with a five-month history of intermittent pain and internal bleeding from an anal fissure was scheduled for a fistulectomy. A preoperative upper gastrointestinal and small bowel series was ordered to exclude the possibility of Crohn disease. Fluoroscopy revealed bilateral round, homogeneous masses that projected above the abdomen with an extrapleural configuration.

A lateral chest radiograph (Fig 1) confirmed the posterior, inferior location of the mass densities apparently arising from the diaphragm. Fluoroscopically, both masses moved in unison with the diaphragm, without any pulsation, configurational change, or internal calcification. The patient denied any history of trauma, chest pain, or pulmonary symptoms.

A CT scan (Fig 2) of the lower thorax and upper abdomen demonstrated both pleural-based lesions which had a similar appearance and uniform hypodensity. Both masses abutted the posterior rib cage and were smooth-contoured, lacking any internal calcifications or gaseous content. Neither mass enhanced significantly with intravenous contrast, and their density (−120 to −130 Hounsfield units) was consistent with homogeneous fat content of diaphragmatic lipomas.

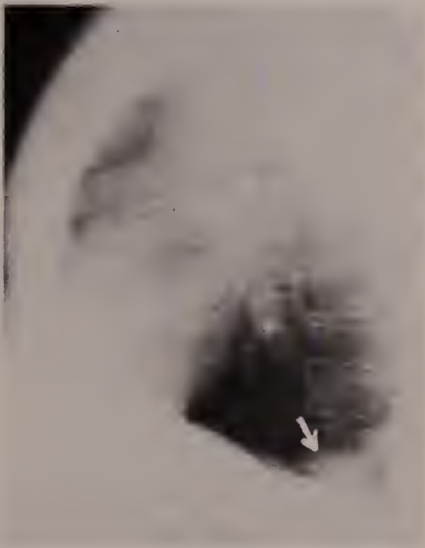

FIGURE 1. Two extrapleural masses (arrow) superimposed along posterior hemidiaphragms on lateral chest film.

DISCUSSION

Lipomas of the diaphragm are encapsulated, soft fatty tumors, which typically present as sessile or pedunculated projections from the diaphragm. Although they are usually round and convex upward, they occasionally have an hourglass shape with abdominal extension through a diaphragmatic isthmus.[4]

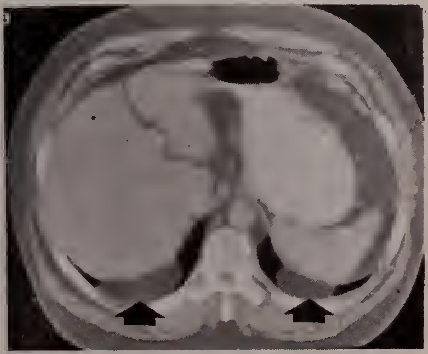

FIGURE 2. Bilateral hypodense masses (arrow) along posterior lung bases on axial CT scan.

Intrathoracic lipomas are equally common in men and women, but occur frequently in obese patients. They have been reported twice as often on the left side, and the majority are posterolateral in location.[5] Lipomas grow slowly or remain stationary, with reported sizes ranging from 1 cm to almost 10 cm in diameter.[6] Although congenital intrathoracic lipomas were hypothesized to exist,[7] none along the diaphragm has been reported in patients younger than 14 years, whereas most are discovered in middle or old age due to their typically asymptomatic nature.[4]

In the past, conventional roentgenographic analysis served only to identify the mass, and thus thoracoscopy or surgical exploration was usually indicated to exclude malignancy, post-traumatic hematoma, and diaphragmatic herniation, versus localized eventration. Barium studies or nuclear scans can exclude herniation or heterotopic growth of abdominal viscera. Ultrasonography can delineate the echolucent lumen of diaphragmatic cysts and hematomas, the membranous layers (of pleura and peritoneum) covering hernias, or the muscular and membranous contour of localized eventrations.[8]

More recently, CT images have provided diagnostic specificity, thus avoiding further workup or surgical intervention.[9] Tissue characteristics of lipomas, including homogeneity and fat density in the −95 to −135 HU range, can be confirmed by this modality. Differentiation from herniated omentum may be more difficult, but discontinuity with abdominal fat can be traced on reconstruction images.

Magnetic resonance imaging (MRI) also readily aids in identifying lipomas, which have a high signal intensity similar to that of subcutaneous fat. MRI's multiplanar capability is of benefit in defining the exact extent and origin of

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these lesions. However, CT is generally the modality of choice, since MRI examinations are more expensive and time consuming.[10]

Finally, there may be a concern about malignant degeneration of lipomas, so that any CT or MR findings may be subject to future revision. Such degeneration has been reported for extrathoracic lipomas, albeit rarely.[11] Hourglass-shaped lipomas may resemble the often undulating contour of liposarcomas. However, there is no documented example of a diaphragmatic liposarcoma, although this lesion has been found in the mediastinum, but more commonly in the retroperitoneum.[5]

Thus, computed tomography preceded by fluoroscopy yields the diagnosis of intrathoracic lipoma with high specificity. Standard chest films may not delineate a low-lying lipoma, but abdominal radiographs and CT scans should be more definitive. Such benign tumors can be monitored by serial conventional abdominal or chest films, although they generally remain stable in size and have not been reported to degenerate into malignant lesions.

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Obstructive sleep apnea in syringomyelia-syringobulbia

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Neurologic disorders affecting the brainstem have been reported as rare causes of obstructive sleep apnea syndrome (OSAS).[1-6] To our knowledge, only one previous case of syringomyelia-syringobulbia as a cause of OSAS has been reported.[7] We recently studied a second case in which tracheostomy reversed debilitating symptoms, which were previously thought to be irreversible sequelae of the neurologic disorder.

CASE REPORT

A 43-year-old man presented in 1981 with a complaint of progressive daytime sleepiness of eight months' duration, resulting in the loss of his job. He regularly fell asleep within minutes of sitting down, even during a conversation or while eating. He was little refreshed following a night's sleep or a nap, but could be aroused easily and was not confused. He reported several episodes of hypnagogic hallucinations, but denied sleep paralysis or symptoms of cataplexy. His wife stated that he had begun to snore heavily, sometimes with long pauses in his breathing. In addition, he noted a three-year history of pain, numbness, and weakness in the proximal right arm.

Physical examination was remarkable for obesity (weight, 114.5 kg; height, 182 cm) and for scars from cigarette burns on both hands. During the neurologic examination, the patient repeatedly fell asleep when asked to close his eyes. While awake, his mental status was normal. Speech was well-articulated except for a lisp. There was a sympathetic pseudoptosis of the left eyelid, and the left pupil was 1.5 mm smaller than the right; sweating was equal on both sides of the face. There was no facial weakness. The palate elevated fully and symmetrically, phonation was normal, and the gag reflex was easily elicited on both sides. There was no atrophy of the tongue, but there was weakness for protrusion into the left cheek. There was no difficulty in swallowing liquids or solids. Sensory testing over the face revealed marked deficits for only pinprick and cold sensibilities over all three divisions of the right trigeminus externally. The mucosa of the right cheek was relatively insentient to pinprick, but the remainder of the intraoral structures tested normally to pinprick, touch, and pressure.

Examination of the trunk and limbs revealed bilateral atrophy of the shoulder girdle. There was marked bilateral weakness for arm elevation, the right more than the left; a lesser degree of weakness was present in the arms with a rough proximal-to-distal gradient. The biceps and triceps tendon jerks were absent. Strength was intact in the legs, which demonstrated hyperreflexia and equivocal plantar responses. Pinprick was not appreciated on the left from C_2 to C_5, and on the right from C_2 to C_6 with partial loss extending to C_8. Cold sensibility was impaired on the right from C_2 to C_6; all other modalities were intact. The patient stood with stooped shoulders, his arms hanging by his sides, and his neck partially flexed. Gait and balance were normal.

The initial electroencephalogram (EEG) showed onset of sleep within one minute and transition from stage II to REM within two minutes. Metrizamide myelography indicated a cord of normal dimensions, while delayed imaging with computed tomography (CT) revealed contrast material within a syrinx in the cord at C_2 level. The intracranial CT was normal. Pulmonary function tests showed a mildly restrictive pattern (vital capacity = 3.16 L, or 69% of predicted).

The patient was placed on methylphenidate, and his condition stabilized on a dose of 20 mg three times a day with moderate improvement in his hypersomnolence. During the next six months, however, his performance declined. Somnolence recurred to the same degree, and his behavior became irrascible; snoring was noted to become louder and more irregular. A CT scan was repeated and again was compatible with syringomyelia; evidence of extension of the syrinx into the brainstem (syringobulbia) was now present (Figs 1,2). Arterial blood gas testing during wakefulness yielded a Pco_2 of 48 mm Hg (6.4 kPa), and further testing re-

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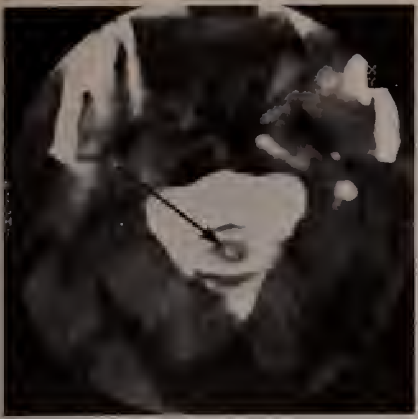

FIGURE 1. CT scan of the cervical spine performed six hours after intrathecal injection of metrizamide dye. The arrow crosses the body of the second cervical vertebra and the subarachnoid space filled with dye (both white), and the arrow head points at the dye-filled syrinx (white) inside the spinal cord (dark).

vealed a diminished respiratory drive to inhaled CO_2: $\Delta\dot{V}_E/\Delta P_{CO_2} = 1.10$ L/m/mm Hg (8.25 L/m/kPa) by rebreathing method.[8] A polysomnographic study was performed after the patient had discontinued all medications. It showed sleep-onset REM and periods of obstructive apnea in all sleep stages (Fig 3), with a calculated apnea index of 37/hour.

Polysomnography was repeated following nasotracheal intubation. No apneas or significant desaturations occurred during spontaneous sleep (stages I and II). Induction of deeper sleep with a small dose of intravenous diazepam also failed to elicit any respiratory defects. On the basis of these findings, tracheostomy was performed. There was an immediate improvement in the hypersomnolence. Repeat studies performed two months later demonstrated a return of resting P_{CO_2} and CO_2 responsiveness towards normal ($P_{CO_2} = 43.8$ mm Hg [5.8 kPa]; $\Delta\dot{V}_E/\Delta P_{CO_2} = 1.78$ L/m/mm Hg [13.35 L/m/kPa]). Subsequently, a CT scan of the pharynx performed while the patient was drowsy, in the supine position, revealed asymmetry and narrowing of the oropharynx with soft tissue fullness and prolapse on the left (Fig 4). Repeat polysomnography showed no apneas, respiratory periodicity, or significant desaturation. Three years later, with the tracheostomy still in place, the patient continues to do well. There has been no progression of his neurologic deficits, and he denies hypersomnolence or other symptoms of OSAS.

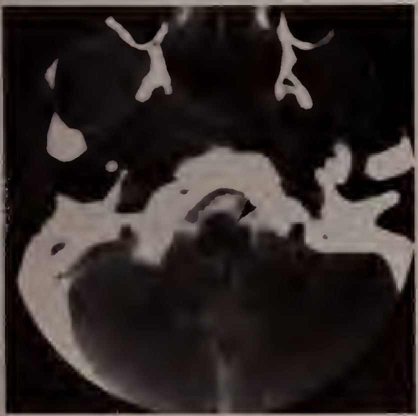

FIGURE 2. CT scan of the lower brainstem with metrizamide dye. The arrowheads bracket the medulla which is distorted, atrophic, and mottled. These features indicate the presence of a syrinx extending at least to this level of the brainstem.

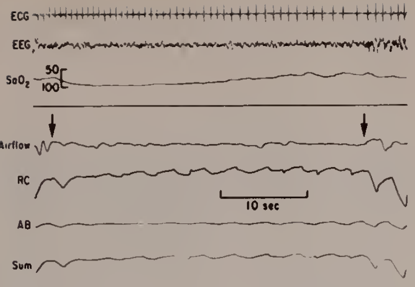

FIGURE 3. Representative polysomnographic tracing. An obstructive apnea is delineated by the vertical arrows on the airflow (nasal thermistor) tracing. The apnea is characterized by the loss of airflow despite continuing evidence of respiratory effort, as recorded by the ribcage (RC) and abdominal (AB) channels of a respiratory inductive plethysmograph. Paradoxic movements of RC and AB are also seen. A marked decrease in Sa_{O_2} occurred. The electrocardiographic (ECG) channel exhibits sinus bradycardia during the latter part of the apnea.

DISCUSSION

There is widespread agreement that OSAS occurs when adequate tone of the muscles controlling upper airway patency is not maintained during inspiration.[9] Although structural abnormality (eg, narrowed pharyngeal cross-section) is frequently demonstrated,[10] dysfunction of the upper airway muscles remains an essential element of the disorder.[11] No neurologic disease is evident in most cases of OSAS. However, some diseases involving brainstem respiratory centers have repeatedly been associated with OSAS. These include poliomyelitis[1-2] (frequently many years after the initial infection), brainstem infarct,[3] Shy-Drager syndrome,[4] and olivopontocerebellar degeneration.[5] In addition, diseases that affect the cranial nerves, such as amyotrophic lateral sclerosis, may also cause OSAS.[6]

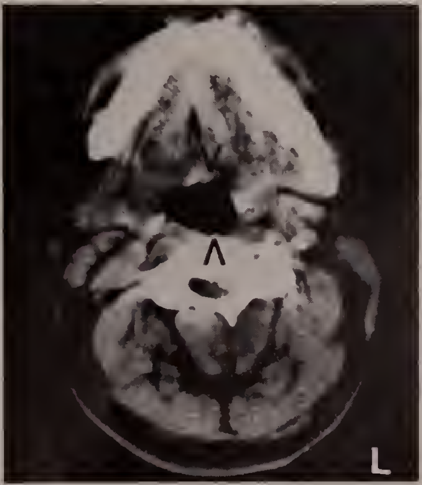

FIGURE 4. CT scan of the oropharynx. Arrowheads indicate midline. There is marked asymmetry, with narrowing of the lumen due to prolapse on the left.

Syringobulbia can lead to similar defects of the lower cranial nerves. Muscles of the tongue, pharynx, and vocal cords may be involved.[12-14] In addition, an extensive syrinx may encroach on the medullary respiratory centers. Abnormalities of respiratory control, such as decreased responsiveness to inhaled CO_2, primary alveolar hypoventilation, and central sleep apnea syndrome, have been reported in such patients.[15-17]

Despite the potential involvement of muscles of the upper airway and of the respiratory control centers, obstructive sleep apnea is rarely reported in patients with syringomyelia-syringobulbia. In a case reported by Adelman and coworkers,[5] OSAS developed in a patient with known syringomyelia-syringobulbia only after neurosurgical decompression of the syrinx. It was therefore more likely a consequence of the surgery itself. In the case reported by Haponik et al,[7] OSAS may be ascribed to the documented syringobulbia. Neurologic findings were similar to those found in the patient described here, including deviation of the tongue with little other cranial nerve abnormality. Apneic episodes were completely abolished by introduction of a nasopharyngeal airway in the case reported by Haponik et al and by endotracheal intubation in this patient. Both patients exhibited a good clinical response to tracheostomy.

This case illustrates the importance of accurately distinguishing obstructive from central sleep apnea in brainstem neurologic disease. Because this patient had daytime hypercapnia, it was initially assumed that the medullary syrinx had damaged brainstem respiratory centers and caused central sleep apnea and a primary alveolar hypoventilation syndrome. Therapeutic results in such disorders generally have been poor.[18] However, the neurologic findings of genioglossal weakness suggested the possibility of airway occlusion and led to the polysomnographic confirmation of OSAS and abolition of the disorder by endotracheal intubation.

The precise mechanism through which syringobulbia caused OSAS in this patient is unclear. Dysfunction of the innervation to the muscles of the upper airway, or destruction of parts of the brainstem respiratory centers or their connections, are possible explanations, and either are supported by the occurrence of OSAS in other neurologic diseases. The presence of decreased hypercapnic ventilatory response and elevated P_{CO_2} initially, with improvement following tracheostomy, suggests the former etiology. The sequence of events might have been as follows: prolapse of the tongue at night, due to destruction of lower cranial nerve nuclei or their central connections, causing OSAS and nocturnal hypercapnia, leading to decreased ventilatory response to CO_2 and daytime hypercapnia. Removal of the nocturnal obstruction via tracheostomy allowed gradual return of respiratory center responsiveness to CO_2 and eucapnia.[19]

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FROM THE LIBRARY

THE ACQUISITION OF CLINICAL WISDOM

In the course of a recent address before the Canadian Medical Association Dr. William Osler made the following pertinent comments: "Medicine is a most difficult art to acquire. All the college can do is to teach the student principles, based on facts in science, and give him good methods of work. These simply start him in the right direction, they do not make him a good practitioner—that is his own affair. To master the art requires sustained effort, like the bird's flight, which depends upon the incessant action of the wings, but this sustained effort is so hard that many give up the struggle in despair. And yet it is only by persistent, intelligent study of disease upon a methodical plan of examination that a man gradually learns to correlate his daily lessons with the facts of his previous experience and with that of his fellows, and so acquires clinical wisdom. Nowadays it is really not a hard matter for a well-trained man to keep abreast of the best work of the day. He need not be very scientific, so long as he has a true appreciation of the dependence of his 'art on science,' for, in a way, it is true that a good doctor may have practice and no theory, art and no science. To keep up a familiarity with the use of instruments of precision is an all-important help in his art, and I am profoundly convinced that as much space should be given to the clinical laboratory as to the dispensary. One great difficulty is that while waiting for the years to bring the inevitable yoke, a young fellow gets stale and loses that practiced familiarity with technique which gives confidence.

"I wish the older practitioners would remember how important it is to encourage and utilize the young men who settle near them. In every large practice there are a dozen or more cases requiring skilled aid in the diagnosis, and this the general practioner can have at hand. It is his duty, and failing to do so he acts in a most illiberal and unjust way to himself and to the profession at large. Not only may the older man, if he has soft arteries in his gray cortex, pick up many points from the young fellow, but there is much clinical wisdom afloat in each parish which is now wasted or dies with the old doctor, because he and the young men have never been on friendly terms.

"In the fight which we have to wage incessantly against ignorance and quackery among the masses and follies of all sorts among the classes, *diagnosis*, not *drugging*, is our chief weapon of offense. Lack of systematic personal training in the methods of the recognition of disease leads to the misapplication of remedies, to long courses of treatment when treatment is useless, and so directly to that lack of confidence in our methods which is apt to place us in the eyes of the public on a level with empirics and quacks."

(*NY State J Med* 1902; 2:277)

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

The myth called Medicare

TO THE EDITOR: John T. Prior's salient commentary, "The myth called Medicare,"[1] is a most important contribution which should be read by all physicians, politicians, sociologists, health planners, and medical journalists in particular, and by the wider public in general.

His incisive critique is the more forceful in coming from within the medical profession, by one whose credentials are unimpeachable, showing keen appreciation of the broad social as well as medical implications inherent in the situation.

Let it not go unnoticed that here, as in other writings, Dr Prior shows by his concerns and by his language that the compassionate physician is not necessarily one who is engaged in providing primary care.

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Teaching in ambulatory care settings

TO THE EDITOR: The response of Dr Wallace et al to the recommendations of the New York State Commission on Graduate Medical Education concerning teaching in ambulatory care settings[1] is subject to debate by family physicians. The article suggests that internists must be trained to provide primary care, yet the definition of primary care is not included in the article.

I propose the following definition to help clarify some issues. Primary care is the delivery of health care to individuals regardless of age, sex, or nature of complaint. Therefore, the only physicians who practice primary care (and thus serve as gatekeepers) are family physicians. Similarly, the statement by Wallace et al (which did not provide any reference) that internal medicine is "the major primary care specialty" in New York State is simply not true. It just happens to be true that there are more internists in New York than family physicians.[2]

Wallace et al further fail to address some of the most fundamental tenets of primary care. The first is continuity of care, which is structured into family practice but not internal medicine programs. Lack of continuity often results in situations such as those well depicted by Deutzman[3] wherein patients with complex medical problems who see several physicians are unable to obtain information about their own problems because nobody acts as "home base." The end result can be a frustrated patient who loathes physicians. Are internists willing to accept cheerfully the responsibility of coordinating health care? Possibly not.[4]

Another important part of primary care that is sadly under-addressed by Wallace et al is the concept of behavioral medicine. Presently, only psychiatry and family medicine offer formal psychosocial training during residency. Yet it is well known that much of patient care is related to psychosocial issues. Surely one cannot learn enough about these issues without a behaviorist's being incorporated into a training program.

I ask whether increasing outpatient training time to 25% will enable internists to perform minor surgery and outpatient gynecological exams, and reliably diagnose psychiatric disease any better than when ambulatory training constituted 10% of residency training. If not, and a depressed woman has to see three specialists (internist, gynecologist, and psychiatrist) for her health care instead of one family physician, then primary care is not being practiced at all.

I am not suggesting that any particular discipline of medicine is any better than any other one. I submit, however, that although it is important for internal medicine residency training programs to increase their outpatient training time, this does not necessarily qualify their graduates as primary care physicians.

A plausible way to increase the number of primary care physicians in New York State is to start by changing the education of medical students. As educators, we have to answer certain questions that have been ignored, such as, Why do medical schools in New York City not offer mandatory third-year clerkships in family medicine? Why is such an emphasis always placed on the rare "pearls" in medicine rather than the more common complaints so often seen in primary care, particularly psychosocial ones? What is a bedside manner? What is family medicine? Perhaps after these questions are addressed, we can change the low ratio of family physicians to specialists which at the present time is making it impossible for patients who want primary care to get it, particularly in New York State.

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In reply. Dr Korpi's letter suggests an either/or posture about the growth of primary care practitioners in New York, which does not reflect the content or intent of our article. Our response to the *Report of the New York State Commission on Graduate Medical Education* accepted the definition of primary care as used in that report, as "comprehensive, continuous, personalized medical care . . . ."[1] Primary care internal medicine exists and has been extensively discussed and defined. Primary care training tracks in internal medicine have been developed in many residency programs and are funded, as are family medicine programs, under Title VII. Dr Korpi's suggestion that a primary care physician can only be a family physician who practices surgery and obstetrics/gynecology disenfranchises not only general internists but family practitioners who do not practice obstetrics or surgery.

The commission report stated that 22.9% of practitioners in New York in 1982 were internists and 5.6% were in family practice.[1] The exact number of internists practicing general internal medicine cannot be extracted from the data. Even if they represented only 40–50% of the total, however, there are clearly more internists than family physicians providing ongoing medical care to the population. To assume that none of the internists in practice provide continuity of care to patients is to do a disservice to the medical profession and has no basis in fact.

We clearly acknowledged the need and desire to move internal medicine residency training into a greater primary care focus and to incorporate a larger emphasis on behavioral medicine, preventive medicine, and on office skills relevant to patients' needs. Federally funded primary care tracks have produced superb primary care internists. Divisions of general medicine have been developed, a new journal of general internal medicine is being published, and non-federally-funded training programs are beginning to shift to a greater primary care focus. In order to more fully achieve the latter we need to meet the needs identified in our article. In particular, house staff must be freed from the extraordinary inpatient service burden which they carry, a burden not placed on family medicine training programs in most institutions.

Family medicine and primary care internal medicine training share similar goals for the acquisition of primary care skills by their trainees. We agree with Dr Korpi that exposure of medical students to primary care experiences is clearly appropriate as we attempt to shift their career choices to meet society's needs.

National attention has been focused on these issues in internal medicine at meetings of the American College of Physicians, the Association of Program Directors in Internal Medicine, the Association of Professors of Medicine, and the American Board of Internal Medicine, among others.

We are much more optimistic than Dr Korpi that these disciplines in primary care are being defined and that with adequate support, training programs in both internal medicine and family practice can serve our society with increasing effectiveness.

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1. *Report of the New York State Commission on Graduate Medical Education.* Albany, NY, State of New York Department of Health, 1986.

Drug treatment and HIV seropositivity

TO THE EDITOR: The second most frequent behavior associated with the prevalence of the acquired immunodeficiency syndrome (AIDS) continues to be parenteral drug abuse.[1] Intravenous (IV) drug abuse is particularly predictive of AIDS cases among women and children in the United States.[1] This is especially true among ethnic/racial minorities.[2,3] Consequently, interventions that discourage the IV drug abuse-associated transmission of the human immunodeficiency virus (HIV) would have significant public health implications for geographic regions where intravenous drug abuse is prevalent.

Drug treatment has frequently been presented as an intervention which might reduce HIV transmission associated with parenteral drug abuse.[4,5] However, few epidemiologic investigations among intravenous drug abusers (IVDAs) have focused on this question. To examine the role of drug treatment on HIV infection, 360 IVDAs, who were either currently enrolled in or applying for admission to drug treatment programs, agreed to participate in an institutional review board approved study in January 1986 in New York City.

The overall HIV seroprevalence rate was 54% and the mean duration of treatment enrollment for the study population was 23 ± 1.4 months. HIV seropositive patients had a mean duration of enrollment of 20.3 ± 1.7 months, while seronegative patients had a mean enrollment of 28.4 ± 2.8 months. This difference was statistically significant ($p < 0.05$) and remained so even after controlling for patient age and duration of drug use. Patients enrolled for less than one year were 1.5 times more likely to be HIV seropositive than those enrolled for more than two years (see Table I). While blacks and Hispanics had longer periods of drug treatment enrollment (27.2 ± 2.3 and 20.6 ± 1.9 months, respectively) than their white counterparts (15.4 ± 2.1 months), the seroprevalence rate among blacks and Hispanics (55% and 58%, respectively) was greater than the HIV infection rate in whites (48%). This difference in HIV infection among these ethnic groups was not statistically significant. Even so, seropositive patients within each racial group had shorter periods of treatment enrollment than those who were seronegative.

TABLE I. HIV Seropositivity by Duration of Treatment Enrollment

	Total Tested	Seropositive N (%)
Less than one year	159	97 (61)
One to two years	72	40 (56)
More than two years	129	65 (50)

These results would support the utilization of drug treatment as a valuable intervention in reducing the risk of HIV exposure in IVDAs. Given the pivotal role that IVDAs play in the prevalence of AIDS in women, children, and ethnic minorities, the public health significance of drug treatment cannot be underestimated.

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Aneurysm of the membranous ventricular septum

TO THE EDITOR: Since Laennec's[1] original description of an aneurysm of the membranous ventricular septum, numerous case reports and reviews have appeared in the literature.[2-3] Steinberg[4] in 1957 was the first to demonstrate a septal aneurysm by angiography. Edelstein and Charms[5] were the first workers to suggest that aneurysms of the membranous portion of the interventricular septum may be related to spontaneous closure of ventricular septal defects.

It is now recognized that closure of ventricular defects that involve the membranous septum may occur through diminution in their size as the heart grows, by fibrosis around the circumference of the defect, by formation of an aneurysm of the membranous septum, or by septal leaflet adhesion of the tricuspid valve which obliterates the shunt by forming a pocket or by plastering completely over the defect.[6]

We report an aneurysm of the membranous septum in an 89-year-old woman which produced no symptoms and was recognized only at autopsy.

Case Report. An 89-year-old woman was admitted to the hospital because of an injury to the left eye caused by a fall. The patient had a history of hypertension, but was otherwise in good health. Her blood pressure was 140/90 mm Hg, and her heart rate was 60/min and regular. There was a corneal abrasion of the left eye. Bilateral carotid artery bruits were noted. The lungs were clear to percussion and auscultation. There was a grade III/VI ejection, systolic murmur over the aortic area. The liver, spleen, and kidneys were not palpated, and there was no peripheral edema.

The electrocardiogram showed nonspecific ST-T wave changes. The chest film revealed a pneumonic infiltrate in the right lower lobe. The patient was treated for pneumonia with various antibiotics. Myocardial infarction and pulmonary embolus were not suspected. The patient died suddenly eight days after admission.

The autopsy revealed bronchopneumonia and pulmonary emboli. There was calcific degeneration of the aortic valve with mild stenosis and calcification of the mitral anulus. There was a recent subendocardial infarction of the anterior apical wall and ventricular septum, not near the membranous septum. There was a 3.3 × 2 × 1.0 cm aneurysm of the membranous part of the interventricular septum which bulged towards the right ventricle. The tricuspid leaflet was also adherent to the ventricular septal aneurysm (Fig 1). No interventricular septal defect was patent.

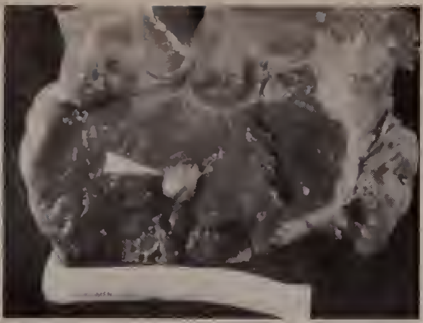

FIGURE 1. The aneurysm can be seen protruding into the right ventricle.

Discussion. The membranous septum is the thinnest part of the interventricular septum. It separates the left ventricle from the right ventricle as well as the right atrium behind the tricuspid valve. Aneurysms of the membranous septum are variable in size and may project into the right atrium or the right ventricle.

The lesions seldom produce significant clinical manifestations. Occasionally aneurysms of the membranous septum are very large and these can produce hemodynamic disturbances. They may even rupture and cause death or they can be the site of thrombus formation or endocarditis. They can also cause obstruction of the right or left ventricular outflow tract. Various rhythm abnormalities and conduction disturbances have been attributed to this defect. This is not surprising considering the close relationship of the membranous septum with the bundle of His.[7]

There are usually no physical signs that suggest the presence of this condition.[8] However, as the aneurysm obstructs the right ventricular outflow tract, a systolic ejection murmur may be heard in the pulmonic area. Aneurysms of the membranous septum can be diagnosed by a selective left ventricular angiogram which shows the outpouching below the aortic root.[4] M-mode and 2-D echocardiograms can also be used to follow patients with this condition.[9] Aneurysm formation may be advantageous for the patient with a ventricular septal defect, since it is one of the mechanisms for decreasing the size of the defect.

Nugent and co-workers[10] studied the natural history of ventricular septal defects in 119 patients. On follow-up angiograms, which were performed an average of 5.4 years after the initial reports, 26 patients were found to have developed aneurysms. Aneurysms were rare in infants. Twelve percent occurred in children under the age of two, 45–50% in children aged 2–11, and 62% in those over age 12. This suggests an evolutionary process which begins in childhood and continues into adult life.

Vidne and co-workers[11] reviewed their surgical experience with patients with interventricular septal defect who also had an aneurysm of the membranous septum. They recommended surgical closure of the defect and excision of the aneurysm as the treatment of choice. However, many pediatric cardiologists would disagree with such a recommendation.

In the patient presented here, the ventricular septal defect was closed by two mechanisms: tricuspid valve adhesion of the defect, and a septal aneurysm. The patient was completely asymptomatic and lived to be 89 years of age. Thus, surgical intervention does not appear to be warranted in cases of aneurysm of the membranous septum.

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New York State neurologists not represented

TO THE EDITOR: Recently a study was undertaken by the Medical Society of the State of New York (MSSNY) to survey the adequacy of workers' compensation fees and to establish a rational measurement of the effort, time, and cost expended by physicians providing services. A questionnaire was mailed to all specialists in the state of New York *except* neurologists. Neurologists were not consulted because there is no society of neurologists in New York State at this time.

There are many other areas concerning the practice of medicine where physician input is needed, such as Medicare, Medicaid, insurance companies, HMOs, PPOs, IPAs, and so on, but no input has been forthcoming from neurologists. Obviously, there is a need for neurologists, especially those in private practice, to band together if they wish to be heard. This is a call to all neurologists who agree. Please write to me with your suggestions. If there is sufficient interest, an organizational meeting could be called at a later date, perhaps to coincide with the annual meeting of the MSSNY.

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Influenza, pneumococcal, and tetanus vaccine recommendations for diabetics and other high-risk individuals

Influenza

Each year, thousands of people are hospitalized and many die needlessly from the complications of influenza. People unable to cope well because of their advanced age or underlying health problems are especially at risk and more likely to require hospitalization. Excess mortality results directly from influenza, pneumonia and the exacerbation of chronic diseases. In epidemic years, approximately 40,000 excess deaths occur nationwide. Unfortunately, only about 20% of those considered to be at the highest risk are vaccinated annually.

Pneumococcal pneumonia

Pneumococcal pneumonia accounts for about 25% of all cases of pneumonia. Mortality from pneumonia is highest among patients with underlying medical conditions, such as cardiopulmonary disease and diabetes. In some high-risk patients, mortality has been reported to occur in as many as 40% of cases of bacteremic disease and 55% of meningitis cases. These rates occur despite treatment with antibiotics. While nationwide estimates on pneumococcal disease range from 150,000 to 500,000 cases each year, only about 10% of the target population has been immunized. In New York State in 1986, there were over 5,000 pneumonia-related deaths. Many of these fatalities could have been prevented by appropriate use of pneumococcal vaccine for people at risk.

Tetanus

From 1982 to 1986, 371 cases of tetanus were reported in the United States. During the same period, 20 cases of tetanus were reported in New York State with two deaths. Nationwide, people 50 years and older accounted for over 70% of the cases. More than half of infected people 60 years and older died from the disease. This is not surprising since more than half of people in this age group are not adequately protected.

Recommendations

The Immunization Practices Advisory Committee (ACIP) specifically recommends vaccinating people with diabetes mellitus against influenza and pneumococcal disease. Tetanus-diphtheria toxoid is recommended for all adults every ten years. Health care providers can play a critical role in disease prevention by recommending annual influenza vaccination, one lifetime dose of pneumococcal vaccine, and tetanus-diphtheria toxoid every ten years as a part of routine, preventive health care for adults with diabetes.

Health care providers are also urged to recommend annual influenza shots for those six months of age or over who are at high risk for influenza for reasons other than chronic metabolic diseases such as diabetes. Those at greatest risk of influenza-related complications include people with chronic disorders of the cardiovascular or pulmonary systems requiring regular medical follow-up or hospitalization during the preceding year and residents of nursing homes and other chronic care facilities.

People for whom influenza vaccine is recommended who are at moderate risk of the complications of influenza include otherwise healthy individuals 65 years of age or more, adults and children who have required regular medical follow-up or hospitalization during the preceding year for renal dysfunctions, anemia or immunosuppression, and individuals six months through 18 years of age who are receiving long-term aspirin therapy and may be at risk of developing Reye syndrome following influenza infection.

Employees capable of nosocomial transmission of influenza to high-risk individuals in a health care facility should also receive influenza vaccine each year.

Vaccine acceptance among people with diabetes can be increased by stressing the following:

- Influenza vaccine does not cause flu. The most frequent side effect is soreness at the injection site.
- Unlike the swine influenza vaccine of 1976, subsequent vaccines have not been associated with an increased frequency of Guillain-Barre syndrome.

Pneumococcal vaccine and influenza vaccine can be given at the same time at different sites without an increase in side effects. Pneumococcal vaccine should be given only once to those at high risk for pneumococcal disease. In addition to people with diabetes, individuals at high risk for pneumococcal disease include those with chronic cardiovascular and pulmonary disease, splenic dysfunction or anatomic asplenia, Hodgkins disease, multiple myeloma, cirrhosis, alcoholism, renal failure, cerebal spinal fluid leaks, and other conditions associated with immunosuppression. Otherwise healthy adults 65 years of age and older are also considered at high risk for pneumococcal disease. If you have questions regarding immunization recommendations for diabetics or seek additional information concerning prevention and control of influenza, pneumococcal disease, and tetanus, call the Immunization Program at (518) 473-4437, or the Diabetes Control Program at (518) 474-1515.

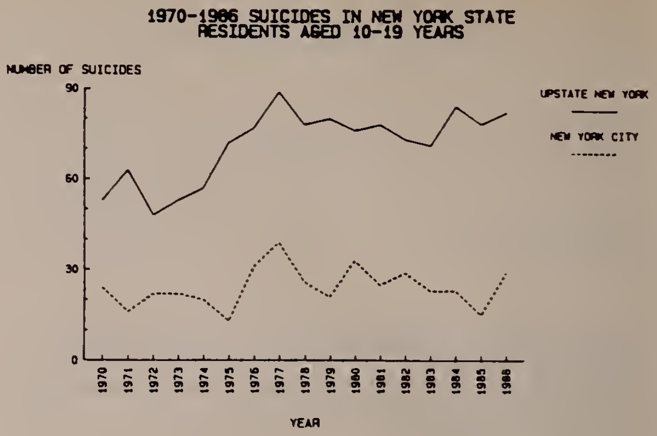
Suicide

It is commonly believed that suicide is underreported as a cause of death.[1] Some true suicides may be purposefully or inadvertently classified as accidents of undetermined cause. Nonetheless, in the 30 years that automated vital records have been kept, approximately 1% of deaths in upstate New York have been recorded as suicides on the basis of death certificate information. During the last ten years, the number of suicides has ranged from a low of 971 in 1985 to a high of 1,137 in 1986. The 1986 figure represents 1.17% of the total number of deaths. In New York City in 1986, there were 576 resident suicides (or 0.78% of all deaths). In recent years, there has been a downward trend in the number of reported New York City suicides. The greatest number (716) was reached in 1976. Suicide is frequently found among the ten leading causes of death in both upstate and New York City.

In recent years, the number of suicides in the upstate area has been almost double the number occurring in New York City. This contrasts with the fact that New York City accounts for about four times as many homicides. Although according to one source,[2] women are three times as likely to attempt suicide as are men, the number of male suicide deaths for 1986 in upstate New York was over three times as high as that for women. The ratio of male to female deaths has also been increasing in recent years. For the last seven years in upstate New York, the ratio has been greater than 3 to 1; the ratio had been between 2 and 3 to 1 in the previous 11 years. In New York City, the male/female ratio is about 2.5 to 1.

The predominate method of suicide for upstate males (50% in 1986) involved the use of firearms. Hanging, strangulation, and suffocation comprised the next largest category of suicides. Among upstate females, the greatest percentage of suicides (about 30%) was accomplished by self-inflicted poisoning by analgesics, barbiturates, or other drugs. The second method of choice was firearms. In New York City in 1986, the greatest number of male suicides was by hanging, strangulation, and suffocation; the greatest number of female suicides was by jumping from a high place. Firearms and jumping were almost equal as a second method of choice for males while the second and third greatest methods for females were hanging and poisoning by drugs.

Teenage suicide appears to be increasing in upstate New York (see graph).



The US surgeon general has stated an objective that the rate of suicide among people 15 to 24 should be below 11.0 per 100,000 population by 1990. In 1980, the upstate New York rate for this 15–24 age group was 9.2 per 100,000. In 1985, the upstate rate was 10.3 for this age group. In New York City, the corresponding figures for 1980 and 1985 were 9.4 and 6.0 respectively. Projections for 1990 were made using the Box-Jenkins method of time-series analysis with rates from 1970 to 1985. The resulting forecast for upstate New York was 10.2 deaths per 100,000 population and a rate for New York City of 7.2 per 100,000. Both of these rates fall within the national target for 1990. In upstate New York, suicides committed in 1986 represent 15.5% of the deaths in the 18–19 age group, 10.1% in the 15–17 age group, and 4.7% in the 10–14 age group. In seven of the last eight years, more than 10% of the deaths were due to suicide among 18–19 year olds. For the 15–17 age group, more than 10% of the deaths were suicides in three of the last four years.

In 1986 in upstate New York, suicide accounted for more than 10% of the deaths in ages 15 to 34. The percentage of deaths due to suicide increases with age, reaching a peak of 15.7% in the 25–29 age group, and decreases with age thereafter. Similar patterns are observed in New York City.

The Bureau of Biostatistics maintains a database in which usual industry and occupation are added to the statistical information from the death certificate. This database now contains more than 260,000 death records of residents of upstate New York who were aged 18–74 at the time of death (1980–1985). A look at the main industry classifications revealed that an excessive proportion of deaths due to suicide was found in the agriculture, construction, and business and repair service industries. Lower suicide levels were found in the personal services and public administration industries. Higher suicide levels were found among students, the unemployed, farmers, and precision production, craft and repair occupations. Lower suicide levels were found among housewives and service occupations.

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BOOK REVIEWS

THE DEMOGRAPHICS OF PHYSICIAN SUPPLY: TRENDS AND PROJECTIONS

Phillip R. Kletke, PHD, William D. Marder, Anne B. Silberger. 62 pp, illustrated. Chicago, American Medical Association, 1987. $18.00 (paperback)

The growing supply of physicians in the US is rapidly becoming the focus of much concern. Previous experience in Europe has shown that physician surpluses can lead to both underemployment and unemployment. Such developments in the US would be tragic for the young men and women who invest so much time, effort, and money into becoming physicians.

Kletke, Marder, and Silberger, of the American Medical Association's Department of Manpower and Demographic Studies, have produced a well-researched volume that presents numerous useful details about the physician supply in the United States. The book is divided into six chapters, "Long-term Trends in Physician Supply," "Trends in Medical Education," "Trends in the Characteristics of Physicians and Their Practices," "Physician Supply: Technical Considerations," "Projection of the Physician Population, 1985–2000," and "Future Research Directions."

Each chapter contains a useful opening summary, and excellent figures and charts are presented throughout. Chapter 5 presents data on physician numbers under several scenarios. These include restricting the entry of alien foreign medical graduates, reducing the number of US foreign medical graduate entrants, and doing both. The third option if adopted would bring about a decline in the physician-to-population ratio which by the year 2000 would be similar to what it was in 1985. However, whichever of these options is projected, physician numbers will not appreciably decline until 2000.

Many hold that physician-population ratios are not valid standards for manpower planning. Physician workloads and disease prevalence and physician use data are viewed as more valid standards. Yet all other variables remaining roughly equal, physician-population ratios can provide good insights into the levels of physician supply.

There are a number of options for dealing with the projected physician oversupply in the United States. One can do nothing, and allow competition among physicians to bring down costs. This option may in fact emerge if a consensus cannot develop around other options. It will not alter the level of supply, only its costs in terms of reimbursement. Another option is to extend the prospective payment system of reimbursement to physician practices. This option might contain costs, but it would have little impact on the oversupply problem. Inducing physicians to move into primary care and rural areas is not really viable as an option, although it has been put forward by some. The surplus is already broadly spread out in most specialty areas, and most rural communities that can sustain viable practices already have sufficient numbers of physicians.

The number of graduates of American medical schools could be reduced by voluntary attrition of class size in individual schools and by the elimination of federal financial support for medical schools and medical students. This is not likely to occur any time soon as it finds little favor with the medical schools for a wide variety of reasons. The only option around which a consensus is forming is entry restrictions on foreign medical graduates. These restrictions are to some degree already underway because of the implementation of the new FMGEMS examination for certification for internship and residency training. Far fewer foreign medical graduates are passing this examination than its predecessor. Further restriction of foreign medical graduates could come about through legislation barring the use of Medicare funds for supporting the training of foreign medical graduates. There is movement in this direction at present, although clearly those hospitals that depend on foreign medical graduates to staff their internships and residencies oppose this initiative.

The physician oversupply that is now emerging in the US did not develop in a vacuum but through the collective efforts of government, private bodies, and organized medicine itself, motivated by an erroneous perception of a physician shortage in the 1960s. Most specialty societies that undertook manpower-need studies in the 1960s and early 1970s called for more of their kind. So did government, and the Carnegie Foundation Report of 1970 that urged a 50% increase in the number of physicians. In addition, the application pressures of "baby boomers" and the high proportion of them who wanted to study medicine influenced public policy in the direction of expanding existing medical schools and establishing new ones during the late 1960s and the 1970s.

The implications of a physician surplus are serious for both physicians and society at large. As time goes on it will become one of the leading if not the leading public policy issue in medical care. This publication provides an excellent overview of the subject and should be read by all physicians concerned with the future of their profession.

PASCAL JAMES IMPERATO, MD
Editor

HEALTH CARE AND ITS COSTS: CAN THE U.S. AFFORD ADEQUATE HEALTH CARE?

Edited by Dr Carl J. Schramm. 301 pp, illustrated. New York, W. W. Norton & Co, 1987. $18.95 (hardcover)

Jack Kent Cooke, owner of the Washington Redskins, when asked why he fired his football coach, George Allen, said, "I gave him an unlimited budget and he exceeded it." Donald R. Cohodes reports a similar phenomenon in his excellent chapter, "The Loss of Innocence: Health Care Under Siege," in this volume under review. He says, "By the mid-1980's, in business's and government's eyes, the health care system had exceeded its unlimited budget." The continuing growth in costs for the medical care of patients, despite the rigid restrictions placed on physicians, hospitals, and community care, and charges by Medicare and the other third party insurance payors, has caused thoughtful and concerned physicians, health economists, and others to question if the United States really has enough money and resources for adequate health care.

This volume, a well-edited account of interesting papers presented in November 1986 at the American Assembly, focuses on health care and costs. It poses the question, "Can the United States afford adequate health care?" It is noteworthy that of the $425 billion spent on health care in 1985, only 19% constituted expenditures for physicians' services. But the direct costs of physicians' services are only the tip of the iceberg. Another 50–60% of health care costs is under the physicians' control and power. This is why "bashing" of physicians ranks high as a public policy in certain cost-containment circles.

The United States can afford to provide adequate health care. But this goal requires more attention to and study of innovative and equitable forms of financing, a shift of emphasis from chronic and long-term care to more formal home care and community-based services, better access to health care, and the maintenance of adequate quality of care.

This excellent book can be recommended to physicians and laymen concerned with improving health care and containing its costs. It will help concerned Americans to come of age in regard to the complex issues of providing good health care. In this process, physicians must be able to furnish quality care and to serve as patient advocates in a sick world which longs for cost containment, but which is unwilling to come to grips with the major problems involved in adequate health care, so well documented in this book.

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FINANCING HEALTH CARE: ECONOMIC EFFICIENCY AND EQUITY

By Steven R. Eastaugh. 720 pp, illustrated. Dover, Mass, Auburn House Publishing Co, 1987. $17.95 (paperback)

Basically, this book considers the multitude of ramifications related to maintaining the delicate balance between two philosophic approaches to health care. One altruistic theory views health care as a social good, while the other opts for a winning profit and loss statement in favor of health care as a consumer good. In today's situation, it would appear that physicians, hospitals, and other health care providers are entering the uneasy and uncomfortable world of business more rapidly than otherwise deemed prudent.

The author states that the US health care system faces revolutionary reformulations in four major areas: access, efficiency, effectiveness, and quality of life. However, the author notes that the ancient Chinese proverb, "May you live in interesting times," implies that a crisis situation offers opportunity as well as danger. Therefore, the rapidly evolving US health care scene could lead either to comprehensive care or to a calamity.

In 24 chapters profusely packed with references, charts, and illustrations, the author covers the following burgeoning issues: generic economic concerns; managerial concerns for buyers and providers; quality assurance and productivity control; health manpower education; efficiency, effectiveness, and cost benefit; diversification, multihospital systems, and access to capital; and future policy trends. Thought-provoking quotations appear at the beginning of each chapter. At the end of each chapter is an informative list of references. A subject and author index for the entire book is also useful.

Acronyms abound in the book and include initials well known to some, such as HMOs, PPOs, DRGs, CMPs, S/HMOs, PPS, CEOs, FTEs and SOI. (For those unfamiliar with the initials they mean: health maintenance organization; preferred provider organization; diagnosis related group; competitive medical plan; social/HMO; prospective payment system; chief executive officer; full-time equivalent; severity of illness.) However, there are also less easily identifiable initials such as HBO (health benefits organization), an alternate delivery system, and WTP (willingness to pay), to measure how much consumers would pay for specific health care services. In addition, the SPGs (strategic product-line groupings) which classify health care services into specific "products" can be used to identify the "cash cows" that account for the greatest "net contribution margin to profitability" in the approach to health care delivery as a business. A telling example comments that "one Miami hospital closed 16 beds and opened a 'Sniffles and Sneezes' center—day care for sick children. Working parents can leave their children for 12 hours at a cost of $20 and receive a pediatrician's exam for an additional $10."

Health workers have always maintained that their services cannot be equated to the productivity concepts of industrial assembly line managers. That myth is shattered by a quote from Peter F. Drucker, a respected managerial consultant:

> Thinking that the facility cannot improve productivity substantially is the principal affliction of the health care industry. Productivity is the first test of management's competence. One should get the greatest output for the least input effort, better balancing all factors of service delivery to achieve the most with the smallest resource effort.

It should be obvious by now that this book is a storehouse of information for those who are involved with the business aspects of medical and health care: administrators, service providers, policy decision makers, consumers, and health economists. Additionally, it should be obvious that this book is not material for bedtime reading unless you are seeking a soporific. Those readers willing to make the effort to apply their attention to the material will be amply rewarded with information, ideas, suggestions, and examples.

For those of you who have been upset and distressed by the prospect of the intrusion of black-hearted Simon Legrees onto the pristine white aseptic floors of health care facilities, take heart in the sanguine words of Will Rogers, also quoted in this book: "An economist can tell you the prices of everything, but the value of nothing."

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LIFE-DEATH DECISIONS IN HEALTH CARE

By Lesley F. Degner, PhD, and Janet I. Beaton, PhD. 159 pp. New York, Hemisphere Publishing Corp, 1987. $39.95 (hardcover)

Lesley Degner and Janet Beaton describe for the readers the myriad factors that become interrelated in the process of making life-death decisions in health care. Both authors are experienced in dealing with the effects of life-death decisions, as they have extensively practiced in both oncology and maternal-child health nursing. Though their background is concentrated in nursing, their focus in this text is on the development of a clear and concise format which assists any health care professional and the lay reader as well in establishing an understanding and an appreciation of the components of life-death decisions. Their work forms a frame of reference for anyone who must be involved in or who must cope with the complex and emotional issues that interplay in making such crucial decisions.

From the outset this text focuses on the major factors that are considered in making the decision, such as the role of the institution; persons included in or excluded from the decision itself; information considered when selecting a treatment option; and the impact the decision has on those who are part of the process. The authors describe the major factors that affect the process and then illustrate each tenet with clinical vignettes. These examples are numerous and at times detract from the content of the text, though they provide supportive illustration.

The authors further describe components of the health care system and how each component operates within lines of formal authority and responsibility. As a backdrop, Degner and Beaton describe the roles of personnel, technology, and treatment as well as the limitations of each. This information offers the reader a framework for understanding the dilemmas the provision of health care creates and the forces that influence the determination of treatment alternatives and patient care outcomes.

Two case studies are provided to further illustrate points previously developed, and finally, recommendations are made for enhancement of the decision making process. The recommendations are designed to respond to the major issues confronting health care providers, ie, utilization of a patient advocate in the decision-making process; communication of information to patients and families which contains a clearly developed analysis of treatment risk versus benefit; and clarification of resuscitation guidelines to reflect individual patient needs, not merely a "code/no code" approach.

In closing the authors include an audit format which has been used to analyze the efficacy of life-death decisions in the clinical setting. The criteria for the evaluation of the decisions seem to hold the greatest promise of assistance to health care professionals as guidelines in finalizing a current decision or auditing previous decisions.

This text offers valuable information which is of increasing importance to a wide range of readers including patients, families, health care professionals, and students as well as volunteers who offer supporting services in the hospital or home environment.

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ETHICS IN EMERGENCY MEDICINE

By Kenneth V. Iserson, MD, Arthur B. Sanders, MD, Deborah R. Mathieu, PHD, Allen E. Buchanan, PHD. 285 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1986. $39.50 (softcover)

This book is directed at a broad audience that is not limited by any means to those specializing in or dealing with emergency services. The centripetal force of the emergency room in modern medical practice provides a powerful focus for ethical discussions which should be of equal interest to students of medicine, law, ethics, and philosophy. The authors have done an excellent job. The book is a "good read," the jargon of the ethicist—no more or less bothersome than that of any specialty—is not intrusive, and the compilation of ethical statements of various agencies and an extensive bibliography are also very helpful. The volume would serve equally well as a text for a course or for the practitioner's library.

The essence of emergency medicine is action, and the philosopher William James aptly identified the minimal requirement for placing an act into the universe of ethical dilemma: "An act has no ethical quality whatever unless it be chosen out of several all equally possible." The authors of this fine contribution to the literature of medicine succeed admirably well in demonstrating that principle. The bulk of the book is organized into a series of cases chosen to illuminate an ethical principle or a difficult area of emergency practice. The possible alternative actions by the treating physician are listed and each is discussed from the perspective of whether it succeeds in fulfilling the ethical responsibility of the physician as fiduciary. The multidisciplinary approach which the authors bring to the discussion reveals the field of medical ethics to be a specialty unto itself, as subtle and difficult as any of the fields of organized medicine yet applicable to all of them. The authors emphasize that the principles are relative, their application turning on the definition of how serious the emergency is and how it is complicated by pertinent law. The relativistic nature of ethical practice is brought into high relief by cases concerning consent (p 69) and notification (p 92), in which a dramatic worsening in the patient's status is in the one case justification for proceeding against the patient's expressed desire and in the other not.

In many of the chapters, key points are restated in tabular form and the tables taken together serve as a primer. It would have been useful, therefore, to rely on this technique in every section. I think it would also have been useful to have more frequent legal commentary, since the practitioner in these litigious times is concerned as much with whether acts of medical care are lawful as whether they are ethical. The instances in the text when legal issues are discussed and the interplay of law and philosophy exposed are fascinating and we can hope that the second edition will be expanded in this direction. Nonetheless, Iserson and colleagues have provided us with an extremely interesting and useful text which is an important addition to the field.

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EVALUATING PREVENTIVE CARE. REPORT ON A WORKSHOP

By Louise B. Russell. 107 pp. Washington, DC, Brookings Books, 1987. $22.95 (cloth)

One of the so-called new directions in medical care these days is preventive medicine. It originally appears to have been stimulated by the search for ways to reduce the high costs of today's medical care. However, as studies were reported it was discovered that preventive medicine only rarely reduces medical expenditures. Rather, it is now considered to offer better health *at additional cost*. If preventive care is not an answer to rising health care costs, we need to consider the question of whether the health gain is worth the cost.

This slim volume (107 pages) is another in the Brookings Institution series. It is the report of a workshop assembled to look at the evidence for the potential of prevention in improving the health of older people.

Six areas are considered: drug therapy for hypertension, smoking, exercise, dietary calcium, alcohol use, and obesity. Twenty-seven scientists with expertise in these areas, led by Louise Russell, who has considerable expertise in this field, were asked to decide if the current evidence about the effects of prevention is enough to warrant more careful, sophisticated attempts to evaluate the cost effectiveness of change. One of the problems was the difficulty in determining the appropriate method of cost analysis, which is simply a set of techniques for determining the effects of a proposed change and the costs required to achieve those effects.

Rather than concentrating only on health care costs, the conference also considered health effects, ie, the sum of the years of life brought about by the intervention and the improvements in health during the years remaining, minus any deterioration caused by the side effects of the intervention.

It is readily evident that the effects of preventive medical care are not as simple as they may seem, and while many do improve health, they rarely reduce expenditures. The participants concluded that the questions of smoking, hypertension therapy, and perhaps exercise were ready for further evaluation, while the issues of dietary calcium, alcohol use, and obesity did not warrant more study at this time.

As a practicing physician I found the book easy to read. The appendix gives a very useful guide to cost effectiveness evaluations which may be applied to a wide range of health practices, and the book's index was complete and quite helpful.

This is not a book that should be on every physician's self. However, as interest continues to accumulate about preventive medical care, this short workshop report will become more popular, and, of course, for those with any interest in this area of medical care, it is an important addition to the material presently available.

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FROM THE LIBRARY

CURRENT EDITORIAL COMMENT

Uterine Cancer Following Radiotherapeutic Menopause. The ability to foretell the occurrence of cancer in an individual would be of assistance in protecting him from the disease by prophylactic measures or in attacking it earlier than would be the case without this anticipation. While heredity and the constitution of the patient probably contain factors predisposing to cancer they furnish as yet no help in the management of an individual. In cancer of the uterus there is, in the behavior of menstruation, possibly a more direct signal. Cancer of the corpus is definitely more frequent in women whose menopause occurs late in life. Also, according to Corscaden, *et al.* excessive bleeding before the menopause indicated a definite predisposition. In a study of 1,000 patients treated by the radiotherapeutic menopause for benign uterine conditions and followed for an average of seven years, there occurred three and a half times as many cancers of the uterus as occur in the same number of women followed for the same length of time in the general population. This means that about 9 percent of the women studied were destined to contract cancer. Three fifths of these were in the corpus and two fifths in the cervix making the corpus cases about six times as frequent as they normally should be. That the radiation was a factor is made unlikely by evidence that radiation with similar technics in other parts of the body has not caused cancer. Further evidence that the abnormal behavior of the endometrium was the predisposing condition is offered by Randall who found an excess of cancer among a similar group of women who had received no radiation.

This predisposition to cancer of the uterus does not permit promiscuous prophylactic removal of the uterus but does suggest that prophylaxis against subsequent cancer of the uterus be a definite factor to be considered in any plan of therapy for benign uterine bleeding with or without fibromyoma of the uterus.

(*NY State J Med* 1946; 46:2138)

NEWS BRIEFS

Are healthcare workers following AIDS safety guidelines?

Emergency room nurses and acupuncturists in Los Angeles are only "haphazardly" following Centers for Disease Control (CDC) guidelines for needle use and disposal, according to a survey performed by the AIDS Virus Education and Research Institute (AVERI: San Francisco, CA). AVERI is an educational organization which serves health care providers, the business community, and the lay public.

Failure to observe CDC safety recommendations can place health professionals at unnecessary risk of exposure to the AIDS virus. Most of the hospitals that participated in the survey probably have established specific guidelines for the handling and disposal of needles. Although 60% of the emergency room nurses surveyed were aware of such hospital policies, two thirds of this group could not recall the specific guidelines.

Accidental needlesticks of fingers and hands while disposing of used needles are one of the main routes for acquiring the AIDS virus for nurses. The CDC strongly advises against recapping needles. Nevertheless, half the nurses questioned routinely put the needle sheath back on the needle. In addition, many nurses polled failed to wear gloves—a simple and effective method of protection against contact with bodily fluids. One hundred percent of nurses did acknowledge wearing gloves when cleaning or wiping wounds. The survey revealed that some patients have requested that nurses wear gloves when examining them.

New high blood pressure medication

Lisinopril (trade name, Prinivil™; Merck Sharp & Dohme, West Point, PA) is a new, long-acting, high blood pressure medication that recently received approval from the US Food and Drug Administration. Indicated for treatment of all degrees of hypertension, lisinopril need only be taken once a day.

This antihypertensive agent is the third in the class of drugs called angiotensin-converting enzyme (ACE) inhibitors. ACE inhibitors act on the renin-angiotension-aldosterone system. The inhibitor binds with the angiotensin-converting enzyme, preventing it from catalyzing the conversion of angiotensin I to angiotensin II. Plasma levels of angiotensin II, a strong vasoconstrictor, decrease, which helps to reduce blood pressure.

In clinical trials, lisinopril was shown to be effective in reducing both systolic and diastolic blood pressure in patients with mild to moderate hypertension. The drug was also generally well tolerated. The most common side effects associated with lisinopril therapy were dizziness (6.3%), headache (5.3%), fatigue (3.3%), diarrhea (3.2%), upper respiratory symptoms (3.0%), and cough (2.9%). ACE inhibitors can also cause swelling of the face, extremities, lips, tongue, and throat tissues.

Lisinopril can be combined with other antihypertensive agents. Excessive hypotension can occur in patients on diuretics who begin lisinopril therapy. The manufacturer recommends an initial dose of 10 mg once a day for patients with uncomplicated hypertension who are not on diuretic agents. Physicians should adjust the dosage according to each individual's response. The usual dosage range is 20–40 mg per day, given in a single dose.

New meningitis vaccine approved

The US Food and Drug Administration has granted approval for marketing for ProHIBiT™ (Connaught Laboratories, Inc, Swiftwater, PA), a vaccine effective against infection by *Hemophilus influenzae* type b, or Hib. Previous vaccines against Hib produced consistent levels of antibody protection only in children two years of age or older. ProHIBiT is effective in children as young as 18 months, and clinical trials are underway to determine its efficacy in infants as young as two months of age.

Failure of earlier Hib vaccines in children less than two years of age is attributed to the young child's immature immune system and poor antibody response and to the weak antigenicity of Hib. The new vaccine takes advantage of conjugate technology to elicit a strong antibody response: scientists link noninfective portions of Hib to the diphtheria vaccine, which is a strong antigen and yields a protective response in infants as young as two months of age. This conjugate vaccine tricks the infant's immune system into producing antibodies against both the diphtheria and Hib antigens.

Hib is responsible for 20,000 to 30,000 cases of systemic illness each year in the US. Hib bacteria are present in respiratory secretions, and thus routes of transmission include coughing, sneezing, and close facial contact. Bacterial meningitis develops in more than half of all children who become infected with Hib. From 25% to 30% of the children who survive suffer permanent brain damage, including hearing loss, partial blindness, mental retardation, behavioral problems, or seizures.

Although ampicillin and chloramphenicol are generally effective against Hib meningitis, strains resistant to these antibiotics have been increasing. To date, more than one of every five infections is resistant to ampicillin.

ProHIBiT is indicated for immunization of children 18 months to five years of age. A single 0.5-mL dose provides adequate immunity. There is no need for booster vaccinations. ProHIBiT will replace HibVAX™, Connaught's polysaccharide vaccine now in use for children 24 months and older.

FDA approves new inactivated poliovirus vaccine

A new, more potent, and more effective inactivated (Salk) poliovirus vaccine recently received FDA approval. The new vaccine (Connaught Laboratories, Ltd, Ontario, Canada), requires only three primary immunizations, compared to the four for the earlier vaccine. Whereas the older version of the Salk vaccine was made using monkey kidney cell cultures, the new vaccine is produced using human cells.

The American Academy of Pediatrics and the National Academy of Sciences recommend the oral (Sabin) live po-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

liovirus vaccine for most immunization programs. The Sabin vaccine induces immunity against intestinal infection, is well tolerated by patients, and can prevent the spread of wild polioviruses. The new inactivated (Salk) vaccine is recommended for use among the following groups: unimmunized adults at increased risk of exposure to poliovirus, such as travelers to certain regions of the world and persons having contact with children who receive the oral polio vaccine, and children with suppressed immune systems.

The new vaccine may contain small amounts of streptomycin and neomycin. Therefore, persons who have anaphylactic reactions to these drugs should not receive the enhanced inactivated vaccine.

Prevalence of diabetes rises in United States

The estimated prevalence of self-reported diabetes in the United States has risen from 0.9% in 1958 to 2.6% in 1985, based on data from the National Health Interview Survey (*Am J Public Health* 1987; 77:1549–1550). Recent findings show that only about half of people with diabetes are aware of their disorder. This statement is based on data from the second National Health and Nutrition Examination survey, conducted by the National Center for Health Statistics, which included a two-hour, 75-g oral glucose tolerance test administered to a group of adults aged 20–74 years who had no history of diabetes. "While approximately 3.4 per cent of the US population age 20–74 years have been diagnosed as having diabetes, an additional 3.2–3.4 per cent meet criteria for the disease but have not been diagnosed . . . . Thus diabetes appears to be twice as prevalent in the United States as rates estimated from medical history surveys."

Certain trends are consistent for both diagnosed and undiagnosed diabetes. For example, the disease is more prevalent among blacks than whites, the prevalence increases with increasing age and with greater degrees of overweight, and women are more likely than men to have diabetes at younger ages.

The authors of this report find it "remarkable that in community studies over the past 40 years and now in this recent national study, the same ratio of about half diagnosed and half undiagnosed diabetes has been found." They contrast this situation with that of hypertension. Awareness of hypertension has increased over the years, and the authors attribute much of that to specific high blood pressure awareness and screening programs. There is no comparable success to report for diabetes.

The authors attribute the large number of undiagnosed cases of diabetes in the US to the asymptomatic nature of the disease in many patients. "The high prevalence of undiagnosed diabetes places many people unknowingly at increased risk of heart disease, blindness, renal failure, and inadequate circulation and sensation in peripheral tissues that can lead to infection, injury, and amputation."

The phenomenon of "white coat hypertension"

"White coat hypertension" is a term used to describe the phenomenon of elevated blood pressure readings in a clinic environment recorded on persons who have normal blood pressure outside the clinic. Visiting a physician's office may cause an increase in blood pressure among some individuals, possibly explained by a simple increase in stress due to the environment and the authority associated with a physician. Pickering et al, from The New York Hospital-Cornell University Medical Center (New York, NY), sought to determine the prevalence of white coat hypertension and to identify common characteristics among affected patients (*JAMA* 1988; 259:225–228). It is possible that some of these patients are receiving unnecessary treatment for hypertension.

Based on the results of the Hypertension Detection and Follow-up Program and similar trials, persons with diastolic blood pressures between 90 and 104 mm Hg are recommended for treatment. This study involved three groups of subjects: one consisted of 37 volunteers with blood pressures in the normal range and no history of hypertension or cardiovascular disease; the second group contained 292 patients with average clinic diastolic blood pressures of 90–104 mm Hg, as measured by a physician on two separate clinic visits, excluding the first visit; the third group of 42 patients had clinic diastolic blood pressure above 105 mm Hg. During this study, none of the patients was taking antihypertensive medication.

Previous studies have shown that multiple blood pressure measurements are more reliable than single recordings, and ambulatory readings correlate better with target organ damage and long-term prognosis than do clinic readings. The subjects in this study wore a noninvasive ambulatory blood pressure monitor for 24 hours. As the subjects followed their normal daily routine, the monitor recorded their blood pressure every 15 minutes during the day and at 30-minute intervals at night. A male physician took the clinic readings using a conventional mercury sphygmomanometer.

"Patients with elevated clinical blood pressures were classified as showing the white coat phenomenon if their awake blood pressures during ambulatory monitoring were below the 90th percentile of the normotensive distribution for *both* systolic and diastolic blood pressure." Using these criteria, 21% of group two and 5% of group three had ambulatory blood pressures within the normal range. Bivariate analysis revealed several features characteristic of the patients with white coat hypertension: they tended to be female, or younger age, of lesser weight, and to have been hypertensive for a shorter period of time than the other patients.

"[O]ne of the traditional arguments used to support the use of clinic pressures for making therapeutic decisions is that patients who show an exaggerated pressor response to the clinic situation may show a similar response to the more regularly occurring types of stress, such as might be experienced during a workday." The authors, however, see little validity in this theory, based on the results of this study and data of other investigators. They also argue against the obvious explanation that anxiety causes white coat hypertension. Rather, the authors propose that the phenomenon is a conditioned response. At first, a patient's blood pressure may be elevated due to anxiety. On subsequent measurements, "an increased level of sympathetic arousal, because the patient expects a high reading, may produce continually high readings. The authors conclude that their data suggest the existence of a low-risk group of patients for whom medical treatment for hypertension may not be necessary.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Gustave Adlerberg, MD, Old Bethpage. Died May 17, 1987; age 92. Dr Adlerberg was a 1920 graduate of Columbia University College of Physicians and Surgeons, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Arthur Joseph Antenucci, MD, New York. Died December 14, 1987; age 82. He was a 1930 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Antenucci was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Hyman A. Asher, MD, Brooklyn. Died December 8, 1987; age 80. Dr Asher was a 1930 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Benjamin Berman, MD, Hallandale, Florida. Died May 15, 1987; age 80. Dr Berman was a 1932 graduate of New York Medical College. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Martin V. Berrigan, MD, Clermont, Florida. Died November 16, 1987; age 64. He was a 1950 graduate of New York Medical College, New York. Dr Berrigan was a Fellow of the American College of Obstetricians and Gynecologists, a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the Medical Society of the County of Oswego and the Medical Society of the State of New York.

Inge A. Bogner, MD, New York. Died November 15, 1987; age 77. Dr Bogner was a 1936 graduate of Facolta di Medicina e Chirurgia dell'Universita di Firenze, Florence, Italy. She was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Nathan Brodie, MD, Staten Island. Died December 1, 1987; age 81. He was a 1930 graduate of New York Medical College, New York. Dr Brodie was a Diplomate of the American Board of Surgery. His memberships included the New York Academy of Gastroenterology, the American Fertility Society, the American Public Health Association, the American Society of Colon and Rectal Surgeons, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Arcangelo Mico Calobrisi, MD, New York. Died December 8, 1987; age 60. Dr Calobrisi was a 1951 graduate of Facolta di Medicina e Chirurgia dell' Universita di Roma, Rome, Italy. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

John M. Caracappa, MD, Elmont. Died November 19, 1987; age 73. He was a 1942 graduate of Medical College of Wisconsin, Milwaukee. Dr Caracappa was a Fellow of the American Academy of Family Practice and a member of the Academy of Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Joseph Solomon Chasnoff, MD, North Miami Beach, Florida. Died August 7, 1987; age 87. Dr Chasnoff was a 1924 graduate of Columbia University College of Physicians and Surgeons, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Joseph A. DeFilippi, MD, New York. Died November 21, 1987; age 44. He was a 1969 graduate of New York Medical College, New York. Dr DeFilippi was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology. His memberships included the Academy of Medicine, the New York State Urological Society, the American Urological Association, the American Fertility Society, the New York County Medical Society, and the Medical Society of the State of New York.

Frank Peter DeLuca, MD, Pelham Manor. Died December 10, 1987; age 80. Dr DeLuca was a 1933 graduate of Boston University School of Medicine, Boston. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

George Rehmi Denton, MD, Albany. Died December 19, 1987; age 73. He was a 1942 graduate of Albany Medical College of Union University, Albany. Dr Denton was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the American Geriatrics Society, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Ivan Fred Dunaief, MD, Setauket. Died November 14, 1987; age 50. Dr Dunaief was a 1963 graduate of Chicago Medical School, Chicago. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Jacob Feldman, MD, Brooklyn. Died August 27, 1987; age 83. Dr Feldman was a 1928 graduate of Boston University School of Medicine, Boston. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Isaac F. Gittleman, MD, Brooklyn. Died July 10, 1987; age 94. He was a 1930 graduate of the State University of New York Health Science Center at Brooklyn. Dr Gittleman was a Diplomate of the American Board of Pediatrics and a member of the New York Pediatric Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Alan M. Gutman, MD, Bronx. Died June 5, 1987; age 49. Dr Gutman was a 1962 graduate of New York University School of Medicine, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Raoul Albert Hebert, MD, Cohoes. Died November 20, 1987; age 94. Dr Hebert was a 1915 graduate of Albany Medical College of Union University, Albany. He was a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Morris Horowitz, MD, Fort Lauderdale, Florida. Died March 18, 1987; age 84. Dr Horowitz was a 1929 graduate of the University of Maryland School of Medicine, Baltimore. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Lewis A. Jarett, MD, Hudson. Died November 10, 1987; age 67. He was a 1951 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. Dr Jarett was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the Medical Society of the County of Columbia, and the Medical Society of the State of New York.

Milton E. Kahn, MD, Buffalo. Died December 13, 1987; age 85. He was a 1925 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr Kahn was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Florentine Lippetz Karp, MD, New York. Died December 17, 1987; age 102. Dr Karp was a 1911 graduate of Faculte de Medicine de l'Universite de Geneve, Geneva, Switzerland. Dr Karp was a member of the Society for Investigative Dermatology, the American Geriatrics Society, the American Medical Women's Association, the New York County Medical Society, and the Medical Society of the State of New York.

Martin Richard Katz, MD, New York. Died December 10, 1987; age 54. Dr Katz was a 1959 graduate of New York University School of Medicine, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Robert Landesman, MD, New York. Died December 6, 1987; age 71. He was a 1939 graduate of Cornell University Medical College, New York. Dr Landesman was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Academy of Medicine, the New York Obstetrical Society, the American Fertility Society, the Associate Editorial Board of the *New York State Journal of Medicine*, the New York County Medical Society, and the Medical Society of the State of New York.

Charles E. Manfredonia, MD, Pompano Beach, Florida. Died November 20, 1987; age 76. Dr Manfredonia was a 1936 graduate of Creighton University School of Medicine, Omaha. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Theodore P. Merrick, MD, Rochester. Died February 15, 1987; age 80. He was a 1934 graduate of Yale University School of Medicine, New Haven. Dr Merrick was a Diplomate of the American Board of Dermatology, Inc, and a member of the American Academy of Dermatology, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Max Miller, MD, Ontario, Canada. Died November 13, 1987; age 76. He was a 1936 graduate of Universitaet Wien, Medizinische Fakultaet, Wien, Austria. Dr Miller was a Fellow of the American College of Cardiology. His memberships included the New York State Society of Internal Medicine, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Charles James Paddock, MD, Amityville. Died December 12, 1987; age 86. Dr Paddock was a 1927 graduate of New York University School of Medicine, New York. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Charles A. Prudhon, MD, Boynton Beach, Florida. Died December 11, 1987; age 92. He was a 1920 graduate of the State University of New York Health Science Center at Syracuse. Dr Prudhon was a Diplomate of the American Board of Otolaryngology and a member of the Medical Society of the County of Jefferson and the Medical Society of the State of New York.

Alan Robert Rosenberg, MD, New York. Died May 25, 1987; age 62. He was a 1947 graduate of Chicago Medical School, Chicago. Dr Rosenberg was a member of the Academy of Medicine, the American Psychiatric Association, the American Board of Legal Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Donald Philip Ross, MD, New York. Died September 26, 1987; age 54. Dr Ross was a 1965 graduate of Universitaire Libre de Bruxelles Faculte de Medicine et de Pharmacie, Bruxelles, Belgium. He was a member of the New York State Dermatological Society, the American Academy of Dermatology, the Nassau County Medical Society, and the Medical Society of the State of New York.

John Hunt Rutledge II, MD, New York. Died December 5, 1987; age 37. He was a 1980 graduate of Duke University School of Medicine, Durham. Dr Rutledge was a Diplomate of the American Board of Internal Medicine, the American Society of Internal Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

George Schreiber, MD, New York. Died December 14, 1987; age 76. Dr Schreiber was a 1935 graduate of Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

John Henry Schultz, MD, Pittsford. Died November 23, 1987; age 71. He was a 1943 graduate of the University of Rochester School of Medicine and Dentistry, Rochester. Dr Schultz was a Diplomate of the American Board of

Obstetrics and Gynecology and a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Milton Irwin Weber, MD, Utica. Died November 16, 1987; age 62. Dr Weber was a 1956 graduate of Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. He was a member of the Academy of Medicine, the American Psychiatric Association, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Seymour Wimpfheimer, MD, New York. Died December 3, 1987; age 88. Dr Wimpfheimer was a 1922 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Academy of Medicine, the New York Obstetrical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Stanley Joseph Zambron, MD, Lackawanna. Died November 26, 1987; age 79. Dr Zambron was a 1934 graduate of State University of New York at Buffalo School of Medicine. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83: 925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

COURTESY DAVID S. ARSENAULT

Medical Society of the State of New York

ANNUAL CME ASSEMBLY

Newer Approaches to Habit Abuse & Dependency

April 22–24, 1988

and the

Annual Meeting of the House of Delegates

April 21–24, 1988

The New York Hilton
Avenue of the Americas at 53rd/54th Streets
New York City

HIGHLIGHTS . . .

- 25 Sessions
- Panel Discussions
- Technical Exhibits
- Two Receptions
- President's Reception & Dinner Dance

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REMARKS ______________________________

(please print)

NOTE: Reservations must be received no later than March 30, 1988 and will be held only until 6 p.m. on day of arrival unless guaranteed.

ARRIVAL DATE ____________________

(check-in time is 3 p.m.)

☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.

DEPARTURE DATE ____________________

(checkout time is 1 p.m.)

HAVE A CREDIT CARD? To take advantage of this credit card PAYMENT GUARANTEED reservation, please complete the information below.

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MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
April 21–24, 1988
Annual CME Assembly
April 22–24, 1988

Circle Preferred rate:
Singles: $135 $150 $160
Doubles/Twins: $160 $175 $185
Executive Tower: $205 Single/Double
Suites: (Parlor & 1) $365 $400
Suites: (Parlor & 2) $465 $500

Rates subject to $8\frac{1}{4}\%$ N.Y. State sales tax; $2.00 per room per night, and 5% N.Y.C. occupancy taxes. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

GENERAL INFORMATION

Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. Bring your family to New York City to enjoy the many sights.

All physicians, members of the allied professions, and their guests are invited to attend the Medical Society of the State of New York's Annual CME Assembly to be held April 22–24, 1988, at the New York Hilton. The two and one-half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. Over 125 outstanding speakers, including a number from out-of-state, will present papers at 25 sessions.

The theme of the CME Sessions will be "Newer Approaches to Habit Abuse and Dependency". A few of the subjects to be presented will be, Human Abuse Through the Ages: "From the Womb to the Tomb"; Managing Life — Style Abuses of the G.I. Tract; Wake Up! Sleep Apnea Can Kill You; AIDS and Self-Induced Skin Diseases. For details, see the Preliminary Program which appears in the January 1988 issue of the *New York State Journal of Medicine*. Our program will offer the clinician timely approaches in diagnosis and treatment of diseases in a wide range of medical specialties.

Here are comments from some of the attendees at the 1987 CME Assembly: "The best program I have attended in 25 years; "The speakers were stimulating and gave scholarly presentations", "The subjects were timely and practical". "The sessions were well organized and informative".

Professional representatives from more than 75 companies will be in the Rhinelander Gallery Exhibit Hall on Friday and Saturday to discuss their products and services. The Technical exhibits will include drug products, office management systems, medical and surgical suppliers, health insurance plans, pension plans and more.

SPECIAL EVENTS

Thursday, April 21

The Annual Dinner Dance, in honor of our President, Samuel M. Gelfand, M.D., will be held in the Trianon Ballroom of the New York Hilton. The reception will be held in the Mercury Ballroom starting at 7:00 p.m. All attendees are welcome. Tickets are $75.00 per person and should be ordered in advance from the Medical Society.

THE WOOLWORTH BUILDING
— *Courtesy David S. Arsenault*

Friday, April 22 and Saturday, April 23

A reception for physicians, their guests, the allied professions and exhibitors will be held both days in the Exhibit Hall between 5:00 p.m. and 6:00 p.m. A free drawing for a color TV set will take place at both receptions.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than March 30, 1988. After that date, rooms are on an availability basis only. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Exhibit Hall on Friday, April 22 and Saturday, April 23 from 8:00 a.m.–5:00 p.m., and on Sunday, April 24, registration will take place on the 2nd floor Promenade from 8:00 a.m.–12:30 p.m. There is a $25.00 registration fee for MSSNY members or members of any state medical society.

House of Delegates registration will take place on the 2nd and 3rd floor Promenades at the following hours: Wednesday, April 20 from 12 noon–6:00 p.m.; Thursday, April 21, Friday, April 22 from 8:30 a.m.–5:30 p.m., Saturday, April 23 from 8:30 a.m.–5:00 p.m., and Sunday, April 24 from 8:30 a.m.–12 noon. All MSSNY members are invited to attend the meetings.

CME MEETING HOURS

Friday, April 22 —	8:30am – 1pm 2pm – 5pm
Saturday, April 23	8:30am – 1pm 2pm – 5pm
Sunday, April 24	8:30am – 1pm

EXHIBIT HOURS

Friday, April 22 —	9am – 6pm
Saturday, April 23	9am – 6pm

1988 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City

Newer Approaches to Habit Abuse and Dependency

FRIDAY, APRIL 22

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Plenary Session: Human Abuses Through the Ages: "From the Womb to the Tomb," *Sutton South/Regent Parlor*
2:00 p.m. - 5:00 p.m.	Women's Medical Society/Occupational Health, *Gramercy B*
	Cardiovascular Diseases, *Sutton South*
	Physical Medicine & Rehabilitation/Family Practice/Orthepedics, *Gramercy A*
	Emergency Medicine/Critical Care, *Murray Hill B*
	Pathology/Pediatrics, *Bryant Suite*
	General Surgery/Radiology, *Regent Parlor*
	Neurosurgery, *Murray Hill A*
	Medical Liability Mutual Insurance Co., *Rendezvous Trianon*

SATURDAY, APRIL 23

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Internal Medicine (All Day), *Regent Parlor*
	Ophthalmology (All Day), *Sutton South*
	Hand Surgery/Neurology (All Day), *Sutton Parlor North*
	Psychiatry (All Day), *Gramercy B*
	Urology/Radiology, *Nassau A*
	Dermatology, *Murray Hill A*
2:00 p.m. - 3:00 p.m.	Medical Technology
2:00 p.m. - 5:00 p.m.	Internal Medicine, *Regent Parlor*
	Ophthalmology, *Sutton South*
	Hand Surgery/Neurology, *Sutton Parlor North*
	Psychiatry, *Gramercy B*
	Chest Diseases/Otolaryngology, *Murray Hill B*
3:00 p.m. - 5:00 p.m.	Computers in Medicine, *Nassau A*

SUNDAY, APRIL 24

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Allergy & Immunology, *Gramercy B*
	Obstetrics and Gynecology, *Murray Hill A*
	Gastroenterology/Colon and Rectal Surgery, *Nassau A*
	Orthopedics/Physical Medicine & Rehabilitation/Anesthesiology, *Regent Parlor*
	Sleep Disorders—Apnoea, *Nassau B*

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians.

◆

Technical Exhibits

United Nations Headquarters Overlooking the East River
Photo courtesy of David S. Arsenault

Advance Registration Form

1988 Annual CME Assembly, April 22–24
The New York Hilton, New York City

Complete and return this form to the Medical Society address as indicated below.

Name____
PLEASE PRINT LAST FIRST

Address____

City/State/Zip__________________________________

Medical Specialty_______________________________

REGISTRATION FEE *Please check categories*

☐ Active Member*	$25.00	☐ Nurse	☐ Lab Technician
☐ Non-Member Physician*	$50.00	☐ Physician Assist.	☐ PT/OT
☐ Allied Profession	$10.00	☐ Dentist	☐ Other________ *Specify*

NO FEE *Please check category*

- ☐ MSSNY Life Member
- ☐ Resident/Intern
- ☐ Medical Students
- ☐ Medical Assist.
- ☐ Speaker
- ☐ Spouse/Family
- ☐ News Media/Medical Writer

REMITTANCE ENCLOSED $__________

* of any state medical society

Please make check payable to:
Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services/MSSNY, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

(*continued from p 10A*)

College of Physicians, 4200 Pine St, Philadelphia, PA 19104.

Apr 23–27. **Miami Comprehensive Review Course in Anesthesiology.** Miami. Contact: Professional Seminars/University of Miami, PO Box 012318, Miami, FL 33101. Tel: (305) 547-6411.

MARYLAND

Apr 11–13. **Sickle Cell Disease—The State of the Art.** Hyatt Regency, Bethesda. *Apr 14–16.* **Arachidonic Acid Metabolism in the Nervous System: Physiological and Pathological Significance.** Hyatt Regency, Bethesda. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

MASSACHUSETTS

Apr 11–15. **Postgraduate Course in Gynecologic and Obstetric Pathology with Clinical Correlation.** 39 Cat 1 Credits. Boston. Contact: Department of Continuing Education, Harvard Medical School, 25 Shattuck St, Boston, MA 02115. Tel: (617) 732-1525.

MISSOURI

Apr 27–30. **Management of Diabetes: Now and in the Future.** Omni International, St Louis. Contact: American College of Physicians, 4200 Pine St, Philadelphia, PA 19104.

NEBRASKA

Apr 18–29. **Family Practice Review.** Omaha. Contact: Marge Adey or Brenda Ram, Center for Continuing Education, University of Nebraska Medical Center, 42nd and Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

AROUND THE WORLD

MEXICO

Apr 5–10. **International Health Economics and Management Institute—"Quality of Care."** Pierre Marques, Acapulco. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650. □

Medical Society of the
State of New York

Annual Dinner Dance

in honor of
Samuel M. Gelfand, M.D.
President

Thursday
April 21, 1988
Trianon Ballroom
The New York Hilton
53rd St. & Avenue of the Americas

Cocktail Hour
7 P.M.

Superlative
Dinner

Subscription $75.00 per person

Reservation Form

Make check payable to:
Medical Society of the State of New York
Attn: Meeting Services
420 Lakeville Road, P.O. Box 5404
Lake Success, New York 11042

Please make reservation(s)
for ______ at the Annual Dinner
Dance on April 21, 1988
Check enclosed for $________

PLEASE NOTE: Reservations and ticket sales will close at 12 Noon on Thursday April 21, one hour before the House Convenes.

Name____________________________________

Address__________________________________

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For patients who can't or won't tolerate liquid KCl.

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†Pooled mean serum potassium following oral administration of 30 mEq K-Tab compared to 24 mEq Slow-K in diuretic-treated hypertensives (n = 20) over 8 weeks.

C I B A

References: 1. Data on file, CIBA Pharmaceutical Company. 2. Skoutakis VA, Acchiardo SR, Wojciechowski NJ, et al: Liquid and solid potassium chloride: Bioavailability and safety. *Pharmacotherapy* 1980;4(6):392-397. 3. Skoutakis VA, Carter CA, Acchiardo SR. Therapeutic assessment of Slow-K and K-Tab potassium chloride formulations in hypertensive patients treated with thiazide diuretics. *Drug Intell Clin Pharm* 1987;21 436-440.

Slow-K®
potassium chloride USP
Slow-Release Tablets
8 mEq (600 mg)

BRIEF SUMMARY (FOR FULL PRESCRIBING INFORMATION SEE PACKAGE INSERT)

INDICATIONS AND USAGE

BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS.

1. For therapeutic use in patients with hypokalemia with or without metabolic alkalosis; in digitalis intoxication and in patients with hypokalemic familial periodic paralysis.
2. For prevention of potassium depletion when the dietary intake of potassium is inadequate in the following conditions: patients receiving digitalis and diuretics for congestive heart failure; hepatic cirrhosis with ascites; states of aldosterone excess with normal renal function; potassium-losing nephropathy; and certain diarrheal states.
3. The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases supplementation with potassium salts may be indicated.

CONTRAINDICATIONS

Potassium supplements are contraindicated in patients with hyperkalemia, since a further increase in serum potassium concentration in such patients can produce cardiac arrest. Hyperkalemia may complicate any of the following conditions: chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e.g., spironolactone, triamterene) (see OVERDOSAGE).

All solid dosage forms of potassium supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract. In these instances, potassium supplementation should be with a liquid preparation. Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to an enlarged left atrium

WARNINGS

Hyperkalemia (See OVERDOSAGE).

In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally. Potentially fatal hyperkalemia can develop rapidly and be asymptomatic.

The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment

Interaction With Potassium-Sparing Diuretics

Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e.g., spironolactone or triamterene), since the simultaneous administration of these agents can produce severe hyperkalemia.

Gastrointestinal Lesions

Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths. These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage, or perforation. Slow-K is a wax-matrix tablet formulated to provide a controlled rate of release of potassium chloride and thus to minimize the possibility of a high local concentration of potassium ion near the bowel wall. While the reported frequency of small-bowel lesions is much less with wax-matrix tablets (less than one per 100,000 patient-years) than with enteric-coated potassium chloride tablets (40-50 per 100,000 patient-years) cases associated with wax-matrix tablets have been reported both in foreign countries and in the United States. In addition, perhaps because the wax-matrix preparations are not enteric-coated and release potassium in the stomach, there have been reports of upper gastrointestinal bleeding associated with these products. The total number of gastrointestinal lesions remains approximately one per 100,000 patient-years. Slow-K should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs.

Metabolic Acidosis

Hypokalemia in patients with metabolic *acidosis* should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, or potassium acetate.

PRECAUTIONS

General:

The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis *per* se can produce hypokalemia in the absence of a deficit in total body potassium, while acute acidosis *per* se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium.

Information for Patients

Physicians should consider reminding the patient of the following:

To take each dose without crushing, chewing, or sucking the tablets.

To take this medicine only as directed. This is especially important if the patient is also taking both diuretics and digitalis preparations.

To check with the physician if there is trouble swallowing tablets or if the tablets seem to stick in the throat.

To check with the doctor at once if tarry stools or other evidence of gastrointestinal bleeding is noticed.

Laboratory Tests

Regular serum potassium determinations are recommended. In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis.

Drug Interactions

Potassium-sparing diuretics: see WARNINGS.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Long-term carcinogenicity studies in animals have not been performed.

Pregnancy Category C

Animal reproduction studies have not been conducted with Slow-K. It is also not known whether Slow-K can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Slow-K should be given to a pregnant woman only if clearly needed.

Nursing Mothers

The normal potassium ion content of human milk is about 13 mEq/L. It is not known if Slow-K has an effect on this content. Caution should be exercised when Slow-K is administered to a nursing woman.

Pediatric Use

Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

One of the most severe adverse effects is hyperkalemia (see CONTRAINDICATIONS, WARNINGS, and OVERDOSAGE). There also have been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see CONTRAINDICATIONS and WARNINGS); other factors known to be associated with such conditions were present in many of these patients.

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea. These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose.

Skin rash has been reported rarely

OVERDOSAGE

The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia. However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see CONTRAINDICATIONS and WARNINGS). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration (6.5-8.0 mEq/L) and characteristic electrocardiographic changes (peaking of T waves, loss of P wave, depression of S-T segment, and prolongation of the Q-T interval). Late manifestations include muscle paralysis and cardiovascular collapse from cardiac arrest (9-12 mEq/L).

Treatment measures for hyperkalemia include the following: (1) elimination of foods and medications containing potassium and of potassium-sparing diuretics; (2) intravenous administration of 300-500 ml/hr of 10% dextrose solution containing 10-20 units of insulin per 1,000 ml; (3) correction of acidosis, if present, with intravenous sodium bicarbonate; (4) use of exchange resins, hemodialysis, or peritoneal dialysis.

In treating hyperkalemia in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity

DOSAGE AND ADMINISTRATION

The usual dietary intake of potassium by the average adult is 40-80 mEq per day. Potassium depletion sufficient to cause hypokalemia usually requires the loss of 200 or more mEq of potassium from the total body store. Dosage must be adjusted to the individual needs of each patient but is typically in the range of 20 mEq per day for the prevention of hypokalemia to 40-100 mEq or more per day for the treatment of potassium depletion. Large numbers of tablets should be given in divided doses.

Note: Slow-K slow-release tablets must be swallowed whole and never crushed, chewed, or sucked

HOW SUPPLIED

Tablets—600 mg of potassium chloride (equivalent to 8 mEq) round, buff colored, sugar-coated (imprinted Slow-K)

Bottles of 100	NDC 0083-0165-30
Bottles of 1000	NDC 0083-0165-40
Consumer Pack—One Unit	
12 Bottles—100 tablets each	NDC 0083-0165-65
Accu-Pak® Unit Dose (Blister pack)	
Box of 100 (strips of 10)	NDC 0083-0165-32

Do not store above 86°F (30°C). Protect from moisture. Protect from light

Dispense in tight, light-resistant container (USP).

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C I B A

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200 mg sulfamethoxazole per 5 ml)

ROCHE®

Please see summary of product information on following page.

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BACTRIM ® (trimethoprim and sulfamethoxazole Roche)

Before prescribing, please consult complete product information, a summary of which follows:
CONTRAINDICATIONS: Hypersensitivity to trimethoprim or sulfonamides, documented megaloblastic anemia due to folate deficiency, pregnancy at term and during the nursing period, infants less than two months of age
WARNINGS: FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS.
BACTRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION Clinical signs, such as rash, sore throat, fever, pallor, purpura or jaundice, may be early indications of serious reactions In rare instances a skin rash may be followed by more severe reactions, such as Stevens-Johnson syndrome toxic epidermal necrolysis, hepatic necrosis or serious blood disorder Perform complete blood counts frequently
BACTRIM SHOULD NOT BE USED IN THE TREATMENT OF STREPTOCOCCAL PHARYNGITIS Clinical studies show that patients with group A ß-hemolytic streptococcal tonsillopharyngitis have a greater incidence of bacteriologic failure when treated with Bactrim than with penicillin
PRECAUTIONS: *General:* Give with caution to patients with impaired renal or hepatic function, possible folate deficiency (*e g* elderly chronic alcoholics, patients on anticonvulsants, with malabsorption syndrome, or in malnutrition states) and severe allergies or bronchial asthma In glucose-6-phosphate dehydrogenase deficient individuals, hemolysis may occur, frequently dose-related
Use in the Elderly May be increased risk of severe adverse reactions in elderly, particularly with complicating conditions, *e g* impaired kidney and/or liver function, concomitant use of other drugs Severe skin reactions, generalized bone marrow suppression (see WARNINGS and ADVERSE REACTIONS) or a specific decrease in platelets (with or without purpura) are most frequently reported severe adverse reactions in elderly In those concurrently receiving certain diuretics, primarily thiazides, increased incidence of thrombocytopenia with purpura reported Make appropriate dosage adjustments for patients with impaired kidney function (see DOSAGE AND ADMINISTRATION)
Use in the Treatment of Pneumocystis Carinii *Pneumonitis in Patients with Acquired Immunodeficiency Syndrome (AIDS).* Because of unique immune dysfunction, AIDS patients may not tolerate or respond to Bactrim in same manner as non-AIDS patients Incidence of side effects, particularly rash, fever, leukopenia with Bactrim in AIDS patients treated for *Pneumocystis carinii* pneumonitis reported to be greatly increased compared with incidence normally associated with Bactrim in non-AIDS patients
Information for Patients Instruct patients to maintain adequate fluid intake to prevent crystalluria and stone formation
Laboratory Tests. Perform complete blood counts frequently, if a significant reduction in the count of any formed blood element is noted, discontinue Bactrim. Perform urinalyses with careful microscopic examination and renal function tests during therapy, particularly for patients with impaired renal function.
Drug Interactions: In elderly patients concurrently receiving certain diuretics, primarily thiazides, an increased incidence of thrombocytopenia with purpura has been reported Bactrim may prolong the prothrombin time in patients who are receiving the anticoagulant warfarin Keep this in mind when Bactrim is given to patients already on anticoagulant therapy and reassess coagulation time Bactrim may inhibit the hepatic metabolism of phenytoin Given at a common clinical dosage, it increased the phenytoin half-life by 39% and decreased the phenytoin metabolic clearance rate by 27% When giving these drugs concurrently, be alert for possible excessive phenytoin effect Sulfonamides can displace methotrexate from plasma protein binding sites, thus increasing free methotrexate concentrations
Drug/Laboratory Test Interactions Bactrim, specifically the trimethoprim component, can interfere with a serum methotrexate assay as determined by the competitive binding protein technique (CBPA) when a bacterial dihydrofolate reductase is used as the binding protein No interference occurs if methotrexate is measured by a radioimmunoassay (RIA) The presence of trimethoprim and sulfamethoxazole may also interfere with the Jaffe alkaline picrate reaction assay for creatinine, resulting in overestimations of about 10% in the range of normal values
Carcinogenesis, Mutagenesis, Impairment of Fertility Carcinogenesis Long-term studies in animals to evaluate carcinogenic potential not conducted with Bactrim. *Mutagenesis.* Bacterial mutagenic studies not performed with sulfamethoxazole and trimethoprim in combination. Trimethoprim demonstrated to be nonmutagenic in the Ames assay No chromosomal damage observed in human leukocytes *in vitro* with sulfamethoxazole and trimethoprim alone or in combination; concentrations used exceeded blood levels of these compounds following therapy with Bactrim Observations of leukocytes obtained from patients treated with Bactrim revealed no chromosomal abnormalities *Impairment of Fertility* No adverse effects on fertility or general reproductive performance observed in rats given oral dosages as high as 70 mg/kg/day trimethoprim plus 350 mg/kg/day sulfamethoxazole
Pregnancy Teratogenic Effects Pregnancy Category C Trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefit justifies potential risk to fetus Nonteratogenic Effects See CONTRAINDICATIONS section
Nursing Mothers. See CONTRAINDICATIONS section
Pediatric Use Not recommended for infants under two months (see INDICATIONS and CONTRAINDICATIONS sections)
ADVERSE REACTIONS: Most common are gastrointestinal disturbances (nausea, vomiting, anorexia) and allergic skin reactions (such as rash and urticaria) **FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS (SEE WARNINGS SECTION)**
Hematologic Agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, neutropenia, hemolytic anemia, megaloblastic anemia, hypoprothrombinemia, methemoglobinemia, eosinophilia *Allergic Reactions* Stevens-Johnson syndrome, toxic epidermal necrolysis, anaphylaxis, allergic myocarditis, erythema multiforme, exfoliative dermatitis, angioedema, drug fever, chills, Henoch-Schoenlein purpura, serum sickness-like syndrome, generalized allergic reactions, generalized skin eruptions, photosensitivity, conjunctival and scleral injection, pruritus, urticaria and rash Periarteritis nodosa and systemic lupus erythematosus have been reported *Gastrointestinal.* Hepatitis (including cholestatic jaundice and hepatic necrosis), elevation of serum transaminase and bilirubin, pseudomembranous enterocolitis, pancreatitis, stomatitis, glossitis, nausea, emesis, abdominal pain, diarrhea, anorexia *Genitourinary* Renal failure, interstitial nephritis, BUN and serum creatinine elevation, toxic nephrosis with oliguria and anuria, crystalluria *Neurologic* Aseptic meningitis, convulsions, peripheral neuritis, ataxia, vertigo, tinnitus, headache. *Psychiatric:* Hallucinations, depression, apathy, nervousness *Endocrine:* Sulfonamides bear certain chemical similarities to some goitrogens, diuretics (acetazolamide and the thiazides) and oral hypoglycemic agents, cross-sensitivity may exist. Diuresis and hypoglycemia have occurred rarely in patients receiving sulfonamides. *Musculoskeletal* Arthralgia, myalgia *Miscellaneous:* Weakness, fatigue, insomnia
DOSAGE AND ADMINISTRATION: Not recommended for use in infants less than two months of age.
URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN *Usual adult dosage* for urinary tract infections is one DS tablet, two tablets or four teaspoonfuls (20 ml) *b.i.d* for 10 to 14 days Use identical daily dosage for 5 days for shigellosis *Recommended dosage for children* with urinary tract infections or acute otitis media is 8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses every 12 hours for 10 days Use identical daily dosage for 5 days for shigellosis *Renal Impaired* Creatinine clearance above 30 ml/min, give usual dosage, 15-30 ml/min, give one-half the usual regimen; below 15 ml/min, use not recommended.
ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS: Usual adult dosage is one DS tablet, two tablets or four teasp (20 ml) *b i d* for 14 days
PNEUMOCYSTIS CARINII PNEUMONITIS Recommended dosage is 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days See complete product information for suggested children's dosage table
HOW SUPPLIED: *DS (double strength) Tablets* (160 mg trimethoprim and 800 mg sulfamethoxazole)—bottles of 100, 250 and 500; Tel-E-Dose® packages of 100, Prescription Paks of 20 *Tablets* (80 mg trimethoprim and 400 mg sulfamethoxazole)—bottles of 100 and 500: Tel-E-Dose® packages of 100; Prescription Paks of 40 *Pediatric Suspension* (40 mg trimethoprim and 200 mg sulfamethoxazole per teasp)—bottles of 100 ml and 16 oz (1 pint) *Suspension* (40 mg trimethoprim and 200 mg sulfamethoxazole per teasp)—bottles of 16 oz (1 pint)
STORE TABLETS AT 15°-30°C (59°-86°F) IN A DRY PLACE PROTECTED FROM LIGHT STORE SUSPENSIONS AT 15°-30°C (59°-86°F) PROTECTED FROM LIGHT

P I. 0586

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YOHIMBINE HCI

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

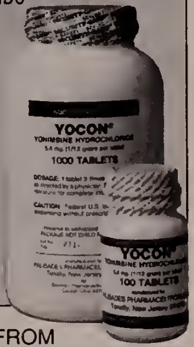
Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85



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Please see next page for brief summary of prescribing information.

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*The effect of food on the bioavailability of Theo-Dur 100 mg tablets has not been determined.

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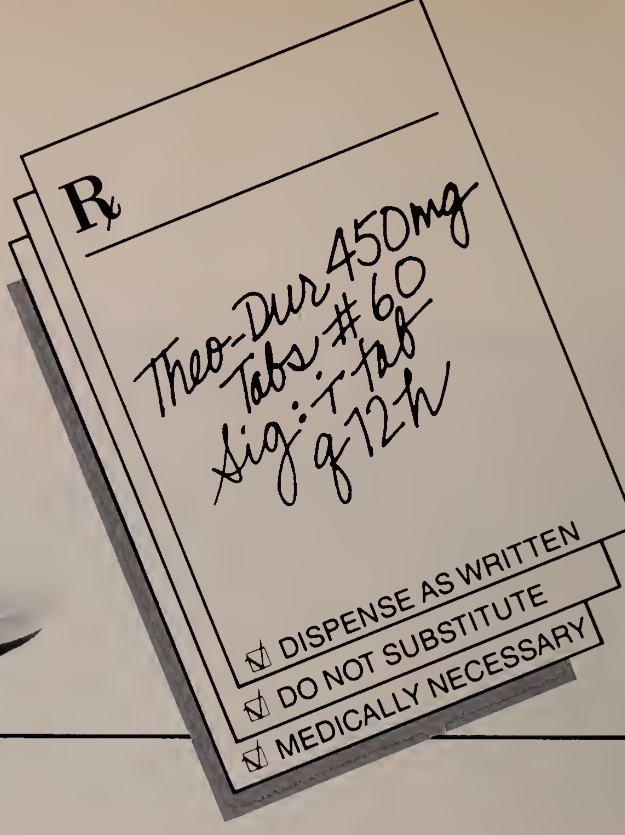

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INDICATIDNS: THEO-DUR is indicated for relief and/or prevention of symptoms of asthma and tor reversible bronchospasm associated with chronic bronchitis and emphysema.
CDNTRAINDICATIONS: THED-DUR is contraindicated in individuals who have shown hypersensitivity to theophylline or any of the tablet components
WARNINGS: Status asthmaticus should be considered a medical emergency and is defined as that degree of bronchospasm which is not rapidly responsive to usual doses of conventional bronchodilators. Dptimal therapy for such patients frequently requires both a*dditional medication*, parenterally administered, and c*lose monitoring*, preferably in an intensive care setting.

Although increasing the dose of theophylline may bring about relief, such treatment may be associated with toxicity. The likelihood of such toxicity developing increases significantly when the serum theophylline concentration exceeds 20 mcg/ml. Therefore, determination of serum theophylline levels is recommended to assure maximal benefit without excessive risk.

Serum levels above 20 mcg/ml are rarely found after appropriate administration of recommended doses. However, in individuals in whom theophylline plasma clearance is reduced *for any reason*, even conventional doses may result in increased serum levels and potential toxicity Reduced theophylline clearance has been documented in the following readily identifiable groups: 1) patients with impaired renal or liver function, 2) patients over 55 years of age, particularly males and those with chronic lung disease; 3) those with cardiac failure from any cause, 4) neonates, and 5) those patients taking certain drugs (macrolide antibiotics and cimetidine). Decreased clearance of theophylline may be associated with either influenza immunization or active infection with influenza

Reduction of dosage and laboratory monitoring is especially appropriate in the above individuals Less serious signs of theophylline toxicity, (i e nausea and restlessness) may occur frequently when initiating therapy. but are usually transient, when such signs are persistent during maintenance therapy, they are often associated with serum concentrations above 20 mcg/ml. Unfortunately. however, serious side effects such as ventricular arrhythmias, convulsions or even death may appear as the first sign of toxicity without any previous warning Stated differently: *serious toxicity is not reliably preceded by less severe side effects.*

Many patients who require theophylline may exhibit tachycardia due to their underlying disease process so that the cause/effect relationship to elevated serum theophylline concentrations may not be appreciated

Theophylline products may cause dysrhythmia and/or worsen preexisting arrhythmias and any significant change in rate and/or rhythm warrants monitoring and further investigation.

The occurrence of arrhythmias and sudden death (with histological evidence of necrosis of the myocardium) has been recorded in laboratory animals (minipigs, rodents and dogs) when theophylline and beta agonists were administered concomitantly, although not when either was administered alone The significance of these findings when applied to human usage is currently unknown.
PRECAUTIONS: THEO-DUR TA8LETS SHOULD NDT BE CHEWED DR CRUSHED

General: Theophylline half-life is shorter in smokers than in nonsmokers. Therefore, smokers may require larger or more frequent doses. Morphine and curare should be used with caution in patients with airway obstruction as they may suppress respiration and stimulate histamine release. Alternative drugs should be used when possible. Theophylline should not be administered concurrently with other xanthine medications. Use with caution in patients with severe cardiac disease, severe hypoxemia, hypertension, hyperthyroidism, acute myocardial injury, cor pulmonale, congestive heart failure, liver disease, in the elderly (especially males) and in neonates. In particular, great caution should be used in giving theophylline to patients with congestive heart failure. Frequently, such patients have markedly prolonged theophylline serum levels with theophylline persisting in serum for long periods following discontinuation of the drug. Individuals who are rapid metabolizers of theophylline, such as the young, smokers, and some non-smoking adults, may not be suitable candidates for once-daily dosing. These individuals will generally need to be dosed at 12 hour or sometimes 8 hour intervals. Such patients may exhibit symptoms of bronchospasm near the end of a dosing interval, or may have wider peak-to-trough differences than desired.

Use theophylline cautiously in patients with history of peptic ulcer. Theophylline may occasionally act as a local irritant to the G.I. tract although gastrointestinal symptoms are more commonly centrally mediated and associated with serum drug concentrations over 20 mcg/ml.
Information for Patients: The physician should reinforce the importance of taking only the prescribed dose and time interval between doses. THED-DUR tablets should not be chewed or crushed When dosing THEO-DUR on a once daily (q24h) basis, tablets should be taken whole and not split. As with any controlled-release theophylline product, the patient should alert the physician if symptoms occur repeatedly, especially near the end of the dosing interval.
Drug Interactions: **Drug-Drug**: Toxic synergism with ephedrine has been documented and may occur with some other sympathomimetic bronchodilators. In addition, the following drug interactions have been demonstrated:

Drug	**Effect**
Theophylline with lithium carbonate	Increased excretion of lithium carbonate
Theophylline with propranolol	Antagonism of propranolol effect
Theophylline with cimetidine	Increased theophylline blood levels
Theophylline with troleandomycin, erythromycin	Increased theophylline blood levels

Drug-Food: *THED-DUR 100 mg Sustained Action Tablets* have not been adequately studied to determine whether their bioavailability is altered when given with food Available data suggest that drug administration at the time of food ingestion may influence the absorption characteristics of theophylline controlled-release products resulting in serum values different from those found after administration in the fasting state.

A drug-food effect, if any, would likely have its greatest clinical significance when high theophylline serum levels are being maintained and/or when large single doses (greater than 13 mg/kg or 900 mg) of a controlled-release theophylline product are given.

THED-DUR (200, 300, and 450 mg) Sustained Action Tablets: The rate and extent of absorption of theophylline from THEO-DUR 200 mg, 300 mg, and 450 mg tablets when administered fasting or immediately after a moderately high fat content breakfast is similar.
Drug-Laboratory Test Interactions: When plasma levels of theophylline are measured by spectrophotometric methods, coffee, tea, cola beverages, chocolate, and acetaminophen contribute falsely high values.
Carcinogenesis, Mutagenesis, and Impairment of Fertility: Long-term animal studies have not been performed to evaluate the carcinogenic potential, mutagenic potential, or the effect on fertility of xanthine compounds.
Pregnancy: Category C—Animal reproduction studies have not been conducted with theophylline. It is not known whether theophylline can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Xanthines should be given to a pregnant woman only if clearly needed.
Nursing Mothers: It has been reported that theophylline distributes readily into breast milk and may cause adverse effects in the infant. Caution must be used if prescribing xanthine to a mother who is nursing, taking into account the risk-benefit of this therapy.
Pediatric Use: Safety and effectiveness of THEO-DUR administered:
1. Every 24 hours in children under 12 years of age, have not been established.
2. Every 12 hours in children under 6 years of age, have not been established.

ADVERSE REACTIDNS: The most consistent adverse reactions are usually due to overdose and are:
1. *Gastrointestinal:* nausea, vomiting, epigastric pain, hematemesis, diarrhea.
2. *Central nervous system:* headaches, irritability, restlessness, insomnia, reflex hyperexcitability, muscle twitching, clonic and tonic generalized convulsions.
3. *Cardiovascular:* palpitation, tachycardia, extrasystoles, flushing, hypotension, circulatory failure, ventricular arrhythmias.
4. *Respiratory:* tachypnea.
5. *Renal:* albuminuria, increased excretion of renal tubular and red blood cells, potentiation of diuresis.
6. *Other:* rash, hyperglycemia and inappropriate ADH syndrome.

OVERDOSAGE: **Management:** If potential oral overdose is established and seizure has not occurred.
A Induce vomiting
8. Administer a cathartic (this is particularly important if sustained-release preparations have been taken).
C. Administer activated charcoal.

If patient is having a seizure:
A Establish an airway.
8. Administer oxygen.
C. Treat the seizure with intravenous diazepam, 0.1 to 0.3 mg/kg up to 10 mg
D Monitor vital signs, maintain blood pressure and provide adequate hydration.

Post Seizure Coma:
A. Maintain airway and oxygenation.
B. If a result of oral medication, follow above recommendations to prevent absorption of the drug, but intubation and lavage will have to be performed instead of inducing emesis, and the cathartic and charcoal will need to be introduced via a large bore gastric lavage tube
C. Continue to provide full supportive care and adequate hydration while waiting for drug to be metabolized In general, the drug is metabolized sufficiently rapid so as not to warrant consideration of dialysis; however, if serum levels exceed 50 mcg/ml charcoal hemoperfusion may be indicated.

CAUTIDN: Federal law prohibits dispensing without prescription. For full prescribing information, see package insert. Revised 2/87

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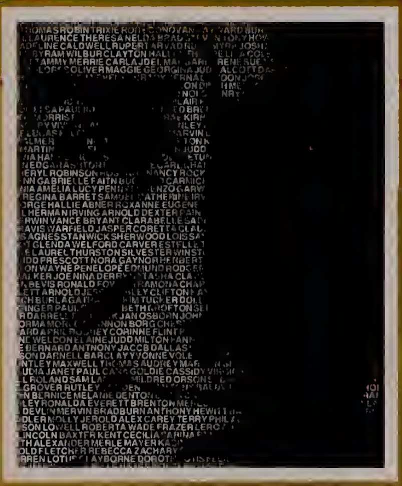

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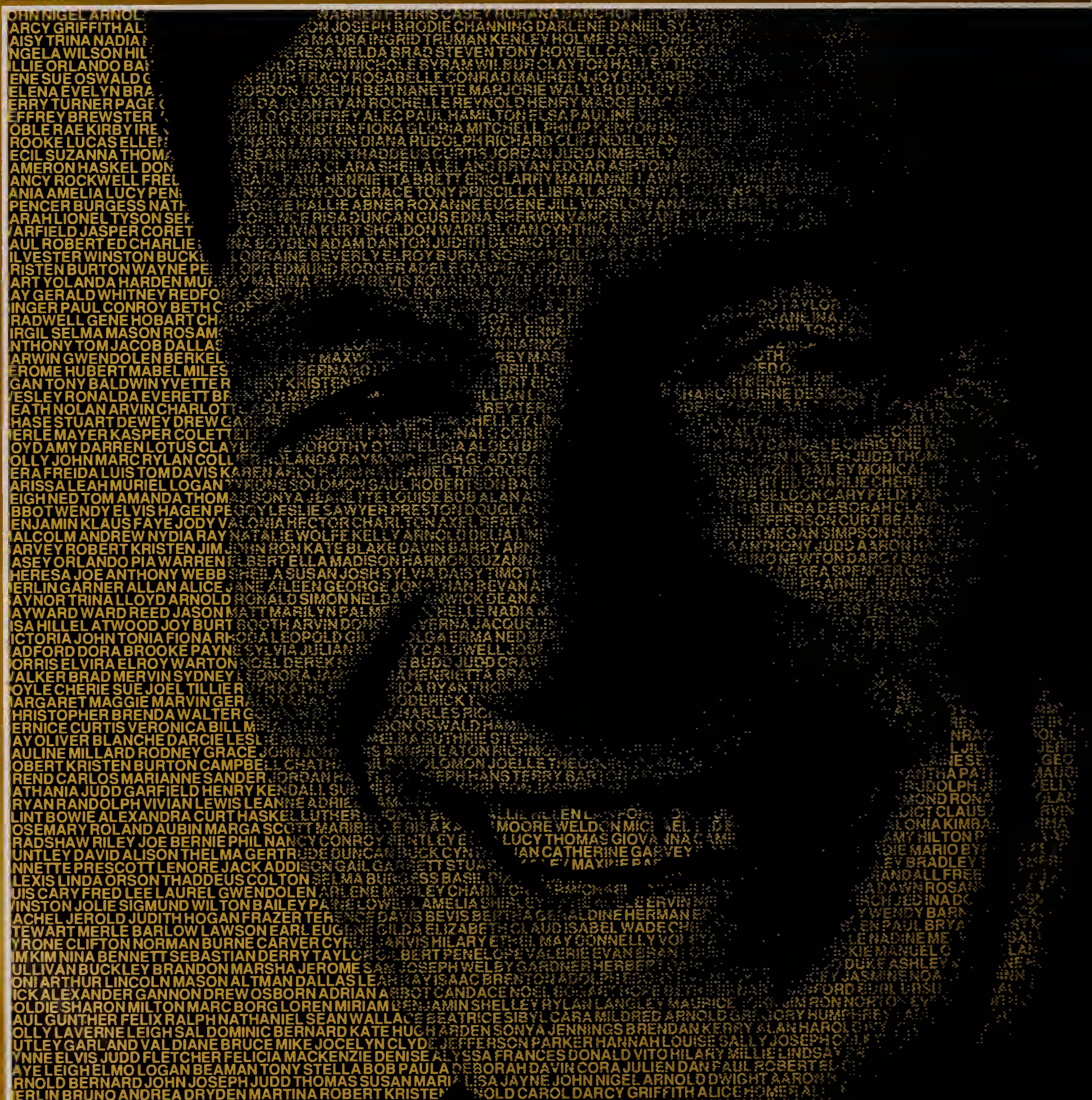

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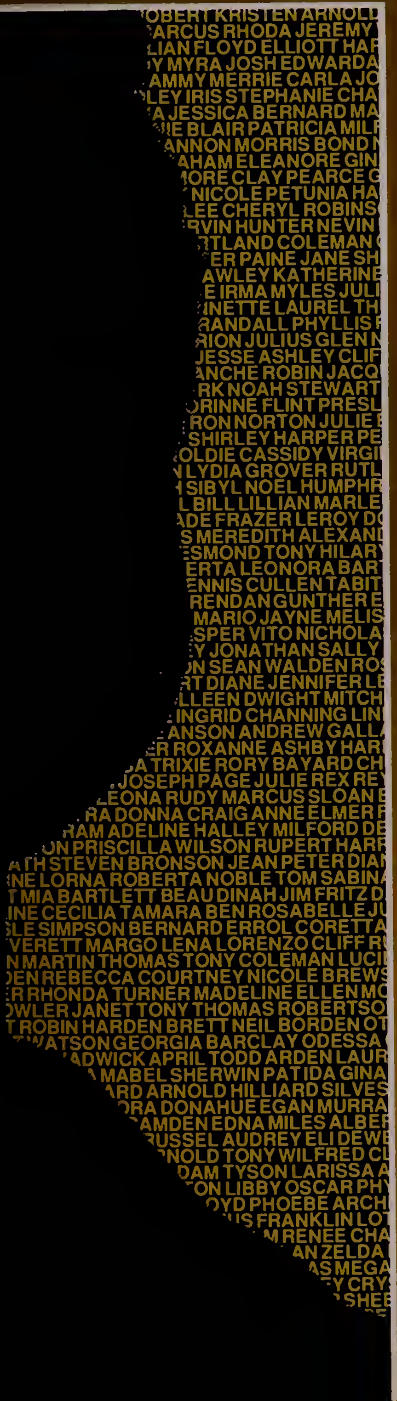

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Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree and bronchial asthma.

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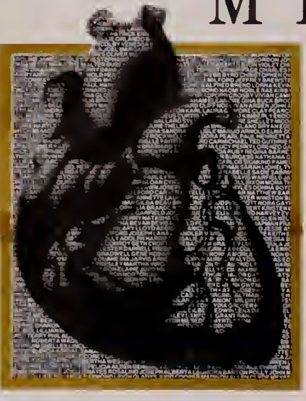

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BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride. INDERAL LA is available as 60 mg, 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure, and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first-degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected. Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL (propranolol HCl) is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Caution should be exercised when patients receiving a beta blocker are administered a calcium-channel-blocking drug, especially intravenous verapamil, for both agents may depress myocardial contractility or atrioventricular conduction. On rare occasions, the concomitant intravenous use of a beta blocker and verapamil has resulted in serious adverse reactions, especially in patients with severe cardiomyopathy, congestive heart failure, or recent myocardial infarction.

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol.

Ethanol slows the rate of absorption of propranolol.

Phenytoin, phenobarbitone, and *rifampin* accelerate propranolol clearance.

Chlorpromazine, when used concomitantly with propranolol, results in increased plasma levels of both drugs.

Antipyrine and *lidocaine* have reduced clearance when used concomitantly with propranolol.

Thyroxine may result in a lower than expected T_3 concentration when used concomitantly with propranolol.

Cimetidine decreases the hepatic metabolism of propranolol, delaying elimination and increasing blood levels.

Theophylline clearance is reduced when used concomitantly with propranolol.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS: INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type.

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Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL Tablets to INDERAL LA Capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg-for-mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary, especially to maintain effectiveness at the end of the 24-hour dosing interval.

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If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

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1. Data on file, Ayerst Laboratories.

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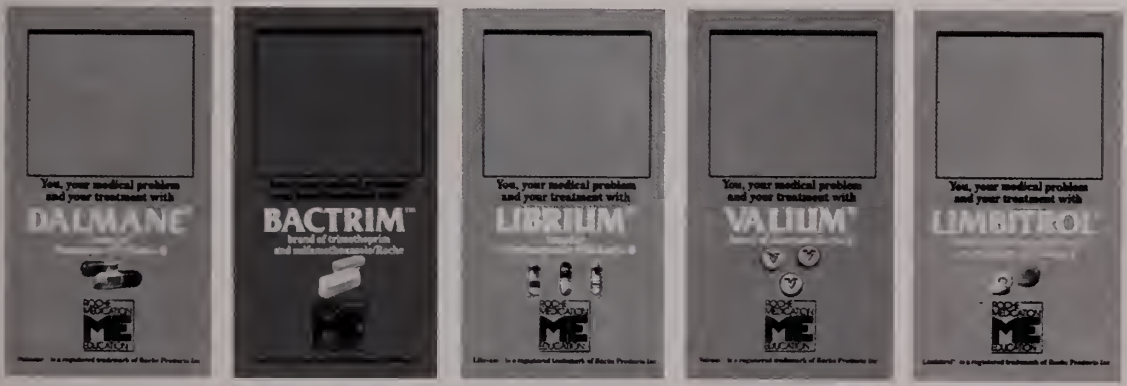
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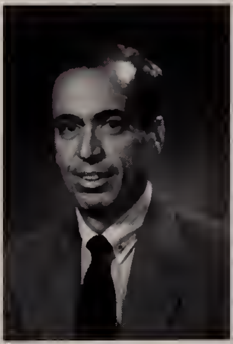
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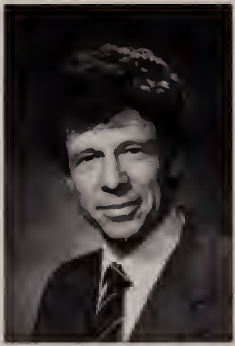
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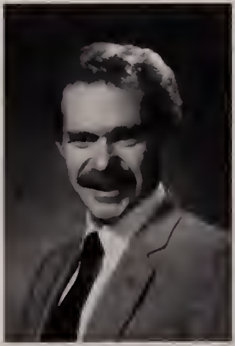
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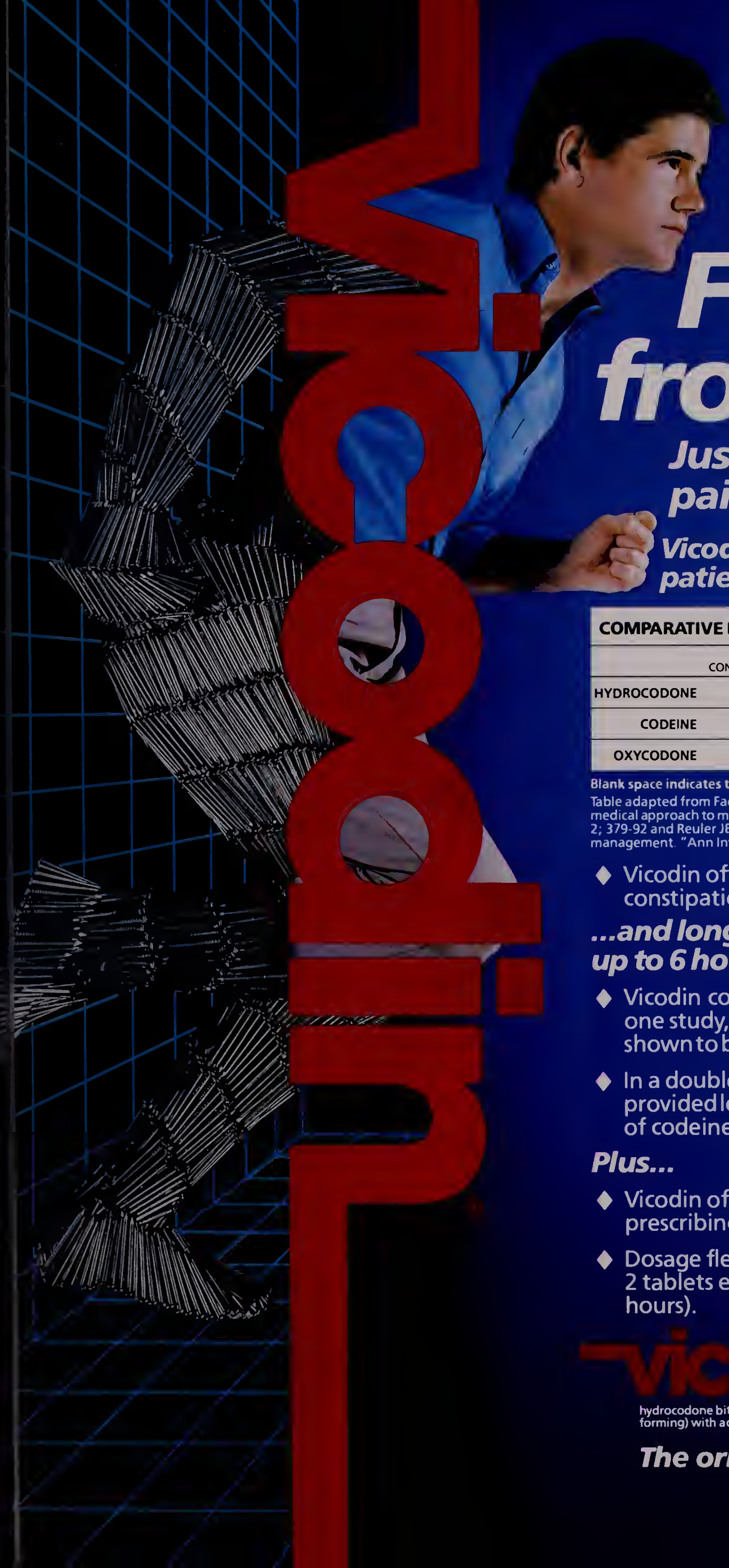

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Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975, 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

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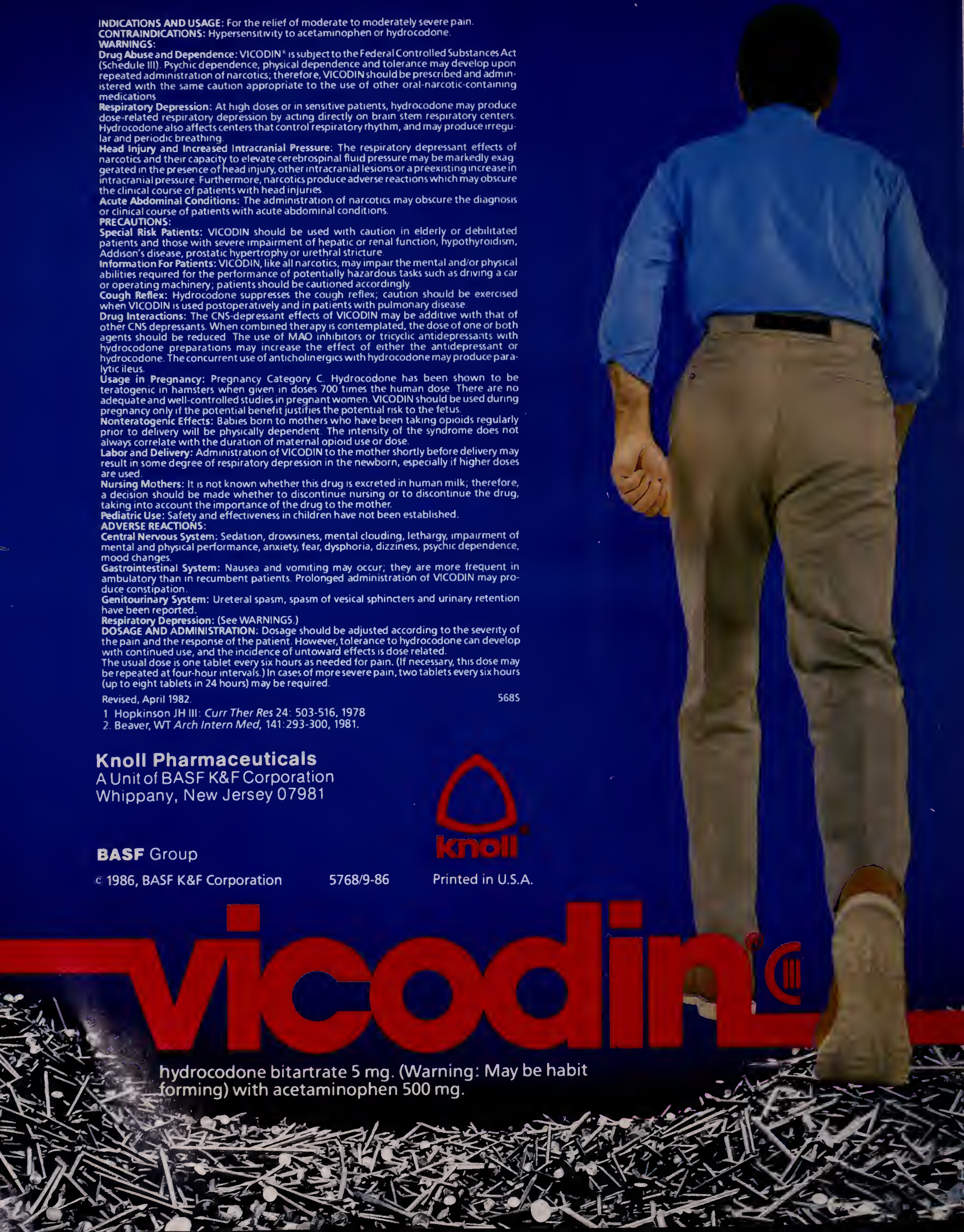

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Nursing Mothers: It is not known whether this drug is excreted in human milk; therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS:
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients. Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.
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Revised, April 1982. 5685

1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med*, 141:293-300, 1981.

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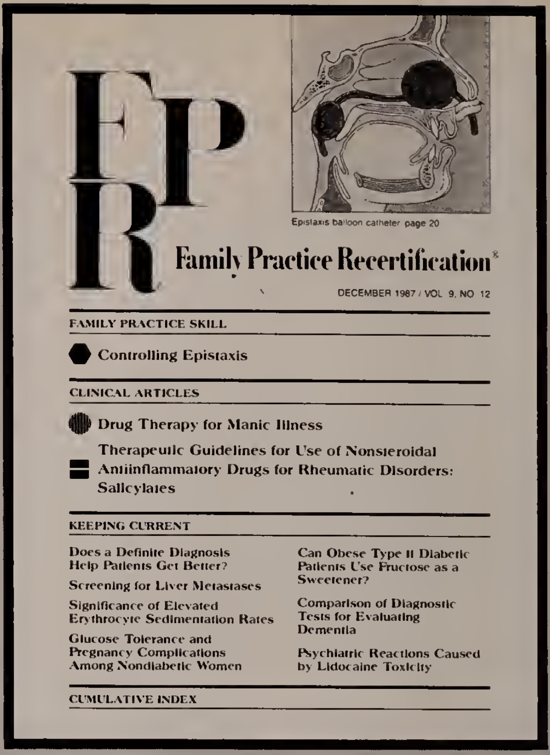
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AROUND THE STATE

MANHATTAN

May 2–6. **Surgical Neuroangiography.** 28 Cat 1 Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

May 5–6. **The L. W. Frohlich Awards Conference—Population Growth: Risks and Benefits.** Sheraton Centre. *May 18–20.* **Ethical Issues in Military Research: What Are the Issues? How Can They Be Handled?** Sheraton Centre. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd Street, New York, NY 10021.

May 7. **Extracapsular Cataract Surgery: Complications—What to Do.** Manhattan Eye, Ear & Throat Hospital. *May 14.* **Diseases of the Eyelids and Conjunctiva: Ophthalmologic and Dermatologic Perspectives.** Manhattan Eye, Ear and Throat Hospital. Contact: Martha Klapp, Manhattan Eye, Ear & Throat Hospital, 210 East 64th Street, New York, NY 10021. Tel: (212) 605-3762.

May 16–18. **A Workshop on the Role of Newer Drugs in the Treatment of Common Diseases.** New York. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

May 25. **The Nazi Doctors—Medical Killing and the Psychology of Genocide.** Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 130 Gold Pl, Malverne, NY 11565. Tel: (516) 887-4691.

SARATOGA SPRINGS

May 20–21. **Radiation Oncology.** The Inn at Saratoga. Contact: Diane M. Martin, Albany Medical College, Office of Continuing Medical Education, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

AROUND THE NATION

CALIFORNIA

May 5–7. **The National Conference on Health Care Leadership and Management.** 16 Cat 1 Credits. Sheraton Harbor Island, San Diego. Contact: Sherry Mason, American Academy of Medical Directors, 4830 W Kennedy Blvd, Suite 648, Tampa, FL 33609-2517. Tel: (813) 287-2000.

May 16–20. **Computing in Anesthesia and Intensive Care.** U.S. Grant Hotel, San Diego. Contact: Office of Continuing Medical Education, M-017, UC San Diego School of Medicine, La Jolla, CA 92093. Tel: (619) 534-3940.

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Help free your patients from recurrences.

Daily therapy

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Prevent or reduce recurrences

Although your patients have to live with herpes, they shouldn't have to suffer. Daily therapy with ZOVIRAX CAPSULES can help free them from the cycle of recurrent genital herpes. For many, one capsule three times a day can suppress recurrences completely while on therapy.

Generally well tolerated

Daily therapy with ZOVIRAX CAPSULES is generally well tolerated. The most frequent adverse reactions reported during clinical trials were headache, diarrhea, nausea/vomiting, vertigo, and arthralgia.

The physical and emotional difficulties posed by genital herpes are unique for each patient. The frequency and severity of recurrent episodes, as well as the emotional impact of the disease, should be considered when selecting daily therapy with ZOVIRAX CAPSULES.

Please see brief summary of prescribing information on next page.

Prevent recurrences month after month*

ZOVIRAX®
(acyclovir)
CAPSULES

Brief Summary

INDICATIONS AND USAGE: Zovirax Capsules are indicated for the treatment of initial episodes and the management of recurrent episodes of genital herpes in certain patients.

The severity of disease is variable depending upon the immune status of the patient, the frequency and duration of episodes, and the degree of cutaneous or systemic involvement. These factors should determine patient management, which may include symptomatic support and counseling only, or the institution of specific therapy. The physical, emotional and psycho-social difficulties posed by herpes infections as well as the degree of debilitation, particularly in immunocompromised patients, are unique for each patient, and the physician should determine therapeutic alternatives based on his or her understanding of the individual patient's needs. Thus Zovirax Capsules are not appropriate in treating all genital herpes infections. The following guidelines may be useful in weighing the benefit/risk considerations in specific disease categories:

First Episodes (primary and nonprimary infections — commonly known as initial genital herpes):

Double-blind, placebo-controlled studies have demonstrated that orally administered Zovirax significantly reduced the duration of acute infection (detection of virus in lesions by tissue culture) and lesion healing. The duration of pain and new lesion formation was decreased in some patient groups. The promptness of initiation of therapy and/or the patient's prior exposure to Herpes simplex virus may influence the degree of benefit from therapy. Patients with mild disease may derive less benefit than those with more severe episodes. In patients with extremely severe episodes, in which prostration, central nervous system involvement, urinary retention or inability to take oral medication require hospitalization and more aggressive management, therapy may be best initiated with intravenous Zovirax.

Recurrent Episodes:

Double-blind, placebo-controlled studies in patients with frequent recurrences (6 or more episodes per year) have shown that Zovirax Capsules given for 4 to 6 months prevented or reduced the frequency and/or severity of recurrences in greater than 95% of patients. Clinical recurrences were prevented in 40 to 75% of patients. Some patients experienced increased severity of the first episode following cessation of therapy; the severity of subsequent episodes and the effect on the natural history of the disease are still under study.

The safety and efficacy of orally administered acyclovir in the suppression of frequent episodes of genital herpes have been established only for up to 6 months. Chronic suppressive therapy is most appropriate when, in the judgement of the physician, the benefits of such a regimen outweigh known or potential adverse effects. In general, Zovirax Capsules should not be used for the suppression of recurrent disease in mildly affected patients. Unanswered questions concerning the human relevance of *in vitro* mutagenicity studies and reproductive toxicity studies in animals given very high doses of acyclovir for short periods (see Carcinogenesis, Mutagenesis, Impairment of Fertility) should be borne in mind when designing long-term management for individual patients. Discussion of these issues with patients will provide them the opportunity to weigh the potential for toxicity against the severity of their disease. Thus, this regimen should be considered only for appropriate patients and only for six months until the results of ongoing studies allow a more precise evaluation of the benefit/risk assessment of prolonged therapy.

Limited studies have shown that there are certain patients for whom intermittent short-term treatment of recurrent episodes is effective. This approach may be more appropriate than a suppressive regimen in patients with infrequent recurrences.

Immunocompromised patients with recurrent herpes infections can be treated with either intermittent or chronic suppressive therapy. Clinically significant resistance, although rare, is more likely to be seen with prolonged or repeated therapy in severely immunocompromised patients with active lesions.

CONTRAINDICATIONS: Zovirax Capsules are contraindicated for patients who develop hypersensitivity or intolerance to the components of the formulation.

WARNINGS: Zovirax Capsules are intended for oral ingestion only.

PRECAUTIONS: General: Zovirax has caused decreased spermatogenesis at high doses in some animals and mutagenesis in some acute studies at high concentrations of drug (see PRECAUTIONS — Carcinogenesis, Mutagenesis, Impairment of Fertility). The recommended dosage and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION).

Exposure of Herpes simplex isolates to acyclovir *in vitro* can lead to the emergence of less sensitive viruses. The possibility of the appearance of less sensitive viruses in man must be borne in mind when treating patients. The relationship between the *in vitro* sensitivity of Herpes simplex virus to acyclovir and clinical response to therapy has yet to be established.

Because of the possibility that less sensitive virus may be selected in patients who are receiving acyclovir, all patients should be advised to take particular care to avoid potential transmission of virus if active lesions are present while they are on therapy. In severely immunocompromised patients, the physician should be aware that prolonged or repeated courses of acyclovir may result in selection of resistant viruses which may not fully respond to continued acyclovir therapy.

Drug Interactions: Co-administration of probenecid with intravenous acyclovir has been shown to increase the mean half-life and the area under the concentration-time curve. Urinary excretion and renal clearance were correspondingly reduced.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg given by gavage. There was no statistically significant difference in the incidence of tumors between treated and control animals, nor did acyclovir shorten the latency of tumors. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive life-time bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system considered less sensitive.

In acute studies, there was an increase, not statistically significant, in the incidence of chromosomal damage at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats but not Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found after 5 days dosing in a dominant lethal study in mice. In 6 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. At 3 loci in a Chinese hamster ovary cell line, the results were inconclusive. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive responses for mutagenicity and chromosomal damage occurred, but only at concentrations at least 400 times the acyclovir plasma levels achieved in man.

Acyclovir has not been shown to impair fertility or reproduction in mice (450 mg/kg/day, p.o.) or in rats (25 mg/kg/day, s.c.). At 50 mg/kg/day s.c. in the rat, there was a statistically significant increase in post-implantation loss, but no concomitant decrease in litter size. In female rabbits treated subcutaneously with acyclovir subsequent to mating, there was a statistically significant decrease in implantation efficiency but no concomitant decrease in litter size at a dose of 50 mg/kg/day. No effect upon implantation efficiency was observed when the same dose was administered intravenously. In a rat peri- and postnatal study at 50 mg/kg/day s.c., there was a statistically significant decrease in the group mean numbers of corpora lutea, total implantation sites and live fetuses in the F_1 generation. Although not statistically significant, there was also a dose related decrease in group mean numbers of live fetuses and implantation sites at 12.5 mg/kg/day and 25 mg/kg/day, s.c. The intravenous administration of 100 mg/kg/day, a dose known to cause obstructive nephropathy in rabbits, caused a significant increase in fetal resorptions and a corresponding decrease in litter size. However, at a maximum tolerated intravenous dose of 50 mg/kg/day in rabbits, there were no drug-related reproductive effects.

Intraperitoneal doses of 320 or 80 mg/kg/day acyclovir given to rats for 1 and 6 months, respectively, caused testicular atrophy. Testicular atrophy was persistent through the 4-week postdose recovery phase after 320 mg/kg/day; some evidence of recovery of sperm production was evident 30 days postdose. Intravenous doses of 100 and 200 mg/kg/day acyclovir given to dogs for 31 days caused aspermatogenesis. Testicles were normal in dogs given 50 mg/kg/day, i.v. for one month.

Pregnancy: ***Teratogenic Effects:*** Pregnancy Category C. Acyclovir was not teratogenic in the mouse (450 mg/kg/day, p.o.), rat (50 mg/kg/day, s.c.) or rabbit (50 mg/kg/day, s.c. and i.v.). There are no adequate and well-controlled studies in pregnant women. Acyclovir should not be used during pregnancy unless the potential benefit justifies the potential risk to the fetus. Although acyclovir was not teratogenic in animal studies, the drug's potential for causing chromosome breaks at high concentration should be taken into consideration in making this determination.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zovirax is administered to a nursing woman. In nursing mothers, consideration should be given to not using acyclovir treatment or discontinuing breastfeeding.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS — Short-Term Administration: The most frequent adverse reactions reported during clinical trials were nausea and/or vomiting in 8 of 298 patient treatments (2.7%) and headache in 2 of 298 (0.6%). Less frequent adverse reactions, each of which occurred in 1 of 298 patient treatments (0.3%), included diarrhea, dizziness, anorexia, fatigue, edema, skin rash, leg pain, inguinal adenopathy, medication taste and sore throat.

Long-Term Administration: The most frequent adverse reactions reported in studies of daily therapy for 3 to 6 months were headache in 33 of 251 patients (13.1%), diarrhea in 22 of 251 (8.8%), nausea and/or vomiting in 20 of 251 (8.0%), vertigo in 9 of 251 (3.6%), and arthralgia in 9 of 251 (3.6%). Less frequent adverse reactions, each of which occurred in less than 3% of the 251 patients (see number of patients in parentheses), included skin rash (7), insomnia (4), fatigue (7), fever (4), palpitations (1), sore throat (2), superficial thrombophlebitis (1), muscle cramps (2), pars planitis (1), menstrual abnormality (4), acne (3), lymphadenopathy (2), irritability (1), accelerated hair loss (1), and depression (1).

DOSAGE AND ADMINISTRATION: Treatment of initial genital herpes: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 10 days (total 50 capsules).

Chronic suppressive therapy for recurrent disease: One 200 mg capsule 3 times daily for up to 6 months. Some patients may require more drug, up to one 200 mg capsule 5 times daily for up to 6 months.

Intermittent Therapy: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 5 days (total 25 capsules). Therapy should be initiated at the earliest sign or symptom (prodrome) of recurrence.

Patients With Acute or Chronic Renal Impairment: One 200 mg capsule every 12 hours is recommended for patients with creatinine clearance ≤10 ml/min/1.73/m².

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*In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.

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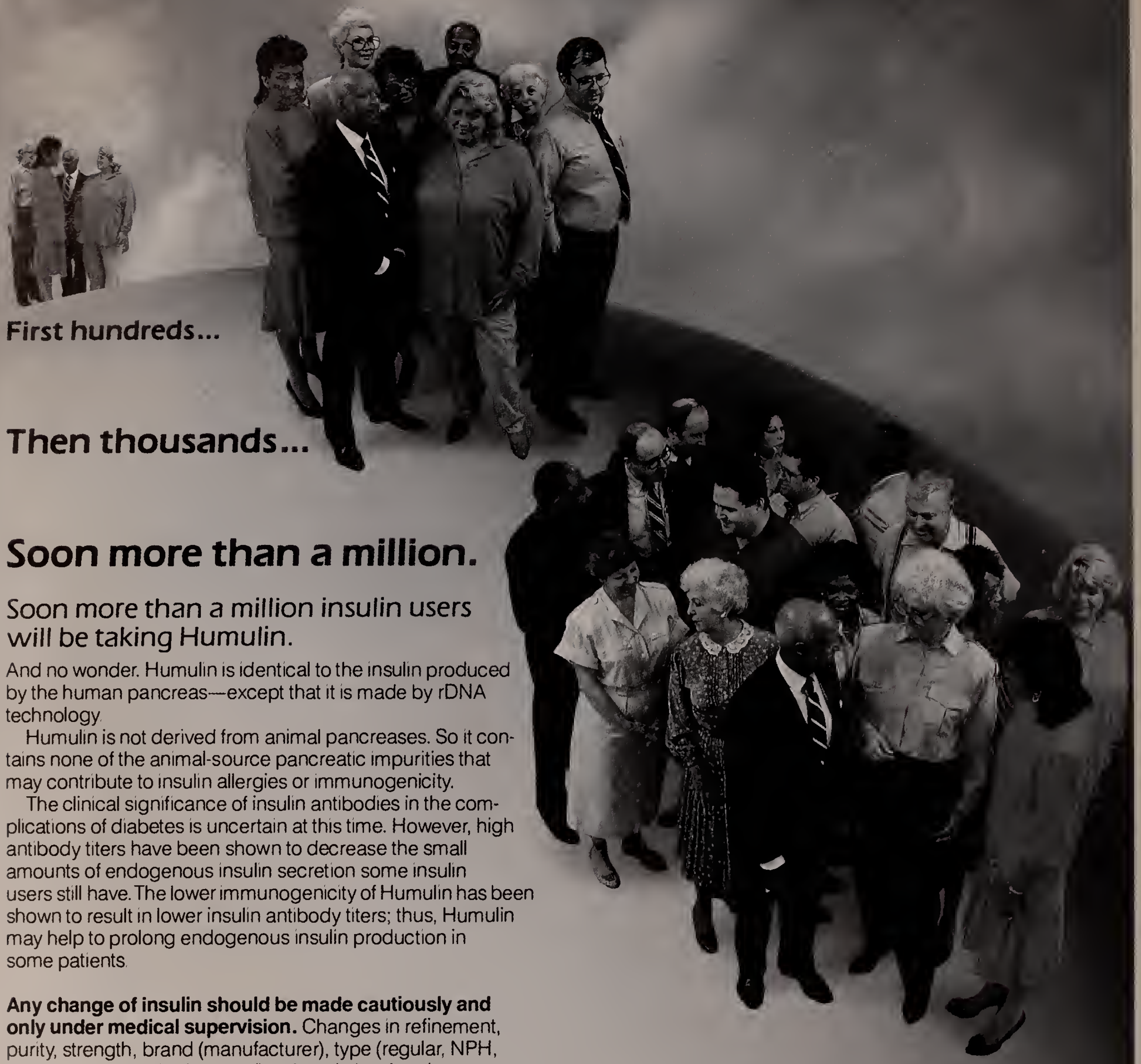

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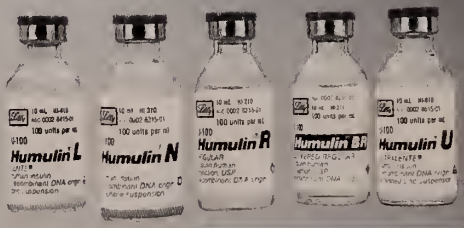

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NEW YORK STATE
JOURNAL OF MEDICINE

April 1988 • Volume 88, Number 4

COMMENTARIES

It isn't over, Doctor*

In the January 8, 1988, issue of the *Journal of the American Medical Association* (*JAMA*), an anonymous writer described an act of supposed "euthanasia" involving a very young woman dying a dreadful death due to malignancy.[1] The staff of *JAMA* published this article in the belief that it was based on a real event. The essay, which appeared in "A Piece of My Mind" (*JAMA's* feature column), has understandably produced a national outcry.[2] A brief synopsis, and our opinion concerning "It's Over, Debbie," follows.

A resident in gynecology was awakened by a phone call in the middle of the night. He (or she) detested these calls because they invariably led to hours of lost sleep and "not feeling good" the following day. The call was answered out of a sense of duty, and, half-asleep, the writer "trudged" to a nursing station with whose patients he was unfamiliar, " . . . bumping sleepily against walls and corners and not believing I was up again. . . ."

He "grabbed the chart from the nurses station on my way to the patient's room . . ." Enroute, the nurse gave him "hurried details" about the patient, a 20-year-old girl named Debbie who " . . . was dying of ovarian cancer." His response, he tells his readers, was "Hmmm . . . very sad."

He found a cachectic and dyspneic woman on intravenous drip and nasal oxygen, who had not slept or eaten for two days and was being given only supportive care. Alongside her stood a middle-aged woman, holding her hand. "Both looked up as I entered. The room seemed filled with the patient's desperate effort to survive." According to the writer, "It was a gallows scene, a cruel mockery of her youth and unfulfilled potential." Her only words to him allegedly were, "Let's get this over with."

The resident then writes, "I retreated to the nurses station. I could not give her health, but I could give her rest." He asked the nurse to draw up 20 mg of morphine sulfate into a syringe. ("Enough, I thought, to do the job.") He injected it intravenously, and " . . . watched to see if my calculations on its effects would be correct." They were, and he then " . . . waited for the inevitable next effect of depressing the respiratory drive." He then goes on to say, "With clocklike certainty, within four minutes the breathing rate slowed even more, then became irregular, then ceased." He goes on to end the essay by saying that the dark-haired woman in the room "seemed relieved."

We believe that the "mercy killing" recounted here does not represent euthanasia, but poor impulse control, and a lack of judgment so severe as to be criminal. The issue is not quality of life but quality of supervision. It is not "right to die" but "right to kill." It leads one to wonder how often residents alone in the middle of the night perform such acts, and how the medical educational establishment is prepared to deal with them.

The reader is left with the impression that the resident may be little more than a cold-blooded killer, ready to dispatch a patient who has interfered with his night's sleep. The cool and methodical way in which he disposes of a young woman, without consulting any of the significant others in her life or that of the institution supports this belief. Why did this young physician totally circumvent society's established mechanisms for dealing with such a complex and awesome situation?

One is struck by the dream-like, impressionistic quality of this recitative, in which vague allusion, metaphor, and double entendres are the language of killing. What did Debbie wish to "get over?" What "rest" was she actually requesting? What commodity was the resident supplying, and where was the three-day "I change my mind" clause in Debbie's contract? What, one wonders, were the powerful and uncomfortable feelings engendered in the resident which led him to seek relief for himself by injecting 20 mg of morphine sulfate into a woman he'd never laid eyes on before?

One can only guess at the unconscious fantasies and beliefs that prompted his action. Was he imbued with salvation fantasies, where, in some "cruel mockery" of the sleeping beauty, he does not awaken her to life but puts her deep into eternal sleep? Was the exhausted young physician so over-identified with his patient that sleep and death merged into one amorphous, blissful state? Was he merely grandiose, or was this the act of a true sociopath? What is clear is that something unspeakable happened on "3 North."

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* The opinions expressed in this editorial are those of the authors alone and do not constitute an expression of official opinion of the Medical Society of the State of New York.

The conduct recounted here is an affront to human decency, a violation of medical ethics, and a crime that should be investigated and dealt with to the full extent of the law. It should serve as a warning and object lesson to all of us as we continue our soul-searching on the painful issues of death and dying. To the writer of this article we say, "It isn't over, Doctor. It has just begun."

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1. Anonymous: It's over, Debbie. *JAMA* 1987; 259:272.
2. Mercy killing report is protested. *NY Times*, February 1, 1987, p A20.

An octogenarian looks at myocardial ischemia

In 1923, Wenkebach visited the University of Michigan Medical School and addressed the assembled faculty and students on rheumatic heart disease and atrial fibrillation, a rhythm of particular interest, since faculty members had contributed to its recognition in the dog and in man. For a neoyphyte it was a revelatory introduction to clinical medicine and an antiarrhythmic drug. Wenkebach told the later oft-repeated anecdote of the Indonesian tea planter's palpitation serendipitously relieved by an antimalarial agent, which led him to the conversion of atrial fibrillation by quinidine. In the same year, Frank Wilson taught physical diagnosis in the University Hospital medical wards, but I did not know then that he had any interest in electrocardiography. Three years later, H.E.B. Pardee,[1] who had described the cove plane T wave he attributed to coronary artery disease, taught me how to operate a string galvanometer and interpret three-lead electrocardiograms. A decade intervened before a fourth lead was added, and eventually the 12 Wilson leads which became the backbone of cardiology.[2]

Viewed in retrospect, there are two themes in the development of our concepts of coronary artery disease: the theme of obstruction of coronary flow by atheromatous stenoses, thrombi, or spasm; and coronary flow as a function of myocardial energy demand.

It was Herrick,[3] in 1912, who linked thrombus to myocardial infarction as a clinical entity, and confirmation of the diagnosis was then possible by electrocardiography. In the 1930s, the treatment of infarction was in open medical wards by bed rest, sedation, opiates, nitrates, digitalis, and the newly synthesized diuretics. It soon became apparent that thrombosis could not always be reliably implicated. Pathologists found infarcts without thrombotic obstruction and thrombi without infarcts, sometimes in the same heart.[4,5] Despite these reservations, the thrombus remained a major concern, and anticoagulation was added to the therapeutic regime. Controversy about its efficacy continued for more than a decade, before anticoagulation therapy was relegated to a peripheral use for thromboembolism, not to be revived until many years later. More fruitful inquiries were pursued by pathologists who demonstrated the anatomic communications of right and left coronary arteries and the experimental demonstration of infarct by ligation with the concominant appearance of collaterals.[6]

The coronary flow theme began with the suggestion by Sir Thomas Lewis in 1932 that angina was due to "muscular ischemia." [7] He demonstrated that a tourniquet to an arm shortened the exercise period necessary to produce forearm pain. When he spoke at the New York Academy of Medicine, Sir Thomas was introduced by E. Libman, who added threshold of pain sensitivity as a contributory factor. As elaborated by hemodynamic studies after the cardiac self-catheterization of Forssmann in 1928,[8] it became apparent that coronary reserve was autoregulated. It was well known that cardiac muscle extraction of oxygen at rest was almost maximal, and increased demand required a shift to anaerobic metabolism and increases in heart rate and blood pressure, which are mediated by nervous system effects on both the coronary and systemic circulation. The failure of balance of energy demand and supply explained angina, and five decades would elapse before electrocardiography confirmed that failure of balance could be silent.

Hemodynamic studies of flow also brought important changes in existing concepts of the determinants of congestive failure, namely preload, afterload, and muscle inotropy. A further dividend was the differentiation of epicardial arteries of conductance and resistance arteries with variable potential for infarction, and a particular vulnerability of the subendocardium to ischemia. The development of these themes of obstruction and flow led to Sones'[9] catheter for cineradiography. The use of open heart surgery for congenital anomalies and valve diseases combined with coronary radiography made possible the advent of bypass surgery for stenotic lesions and later of percutaneous transluminal coronary angioplasty.

These themes were illustrated in 1962 at a seminar at Wayne State University School of Medicine, chaired by R. J. Bing,[10] at which Sones described his catheter technique. Other presentations included reports by Bing on the measurement of coronary flow using rubidium 84 and by Ross[11] using xenon 133. This was followed by the widely accepted thermodilution method of Swan-Ganz[12] and many methods using isotopes. The theoretical aspects of coronary reserve were now overshadowed by preoccupation with bypass of obvious obstruction to flow. The results of surgery have been reported in individual and cooperative studies, and controversies comparable to the earlier disputes on anticoagulation have flourished.

As we approach the last decade of the century, the rediscovery of silent ischemia[13] has integrated the clinical subsets of stable, unstable, and variant angina into a single entity. Clinicians have long known that silent infarction, the obvious consequence of silent ischemia, is a frequent occurrence, estimated by some to represent as much as 25% of all infarcts. Silent ischemia with circadian rhythm detected by Holter electrocardiograms has been demonstrated in all clinical types of angina. The integration of clinical subsets is now confirmed by endoscopy of coronary arteries,[14] and the demonstration that disruption of the endothelium by necrotizing atherosclerotic plaques causes the release of constricting and platelet aggregating factors, which can lead to thrombotic obstruction and regional myocardial dysfunction. Myocardial ischemia, silent or anginal, is the result of the failure of coronary reserve to meet metabolic demand at the cellular level. This is autoregulated by vasodilation in the resistance vessels and collaterals, by complex autonomic nervous system responses accompanied by paradoxic variations in the conduit vasculature, and by normal endothelium-derived relaxing factor.

Although the types of therapy available have expanded exponentially in the last decades, the management of coronary artery disease remains difficult. The apparent improvement in mortality rates is due to the recognition and treatment of the known risk factors of hypertension, diabetes, and overweight, and the introduction of programs for alteration of behavior patterns relating to cigarette smoking, exercise, and diet for reducing low density lipoprotein levels. The asymptomatic patient as well as those with angina are at risk of acute infarction in a spectrum of clinical syndromes ranging from a silent event to a minimal, major, or catastrophic event. All patients at risk require screening by methods now available for symptomatic patients. The demonstration of silent ischemia by electrocardiography at rest or during exercise, abnormalities of perfusion shown using isotopes, or newer modalities now being developed may require a more intensive medical and possibly invasive prophylactic approach.

The medical treatment of acute myocardial infarction in a monitored environment has produced definite improvements in survival rates. This has been accomplished using antiarrhythmic agents, beta blockers, nitrates, anticoagulants, calcium entry blockers, antiplatelet aggregation agents, and better treatment of congestive failure. The aim is improvement in coronary flow and limitation of infarct size, which is the determinant of dysrhythmia and of regional and global myocardial dysfunction. A more vigorous approach is possible in the first few hours following acute myocardial infarction using intravenous streptokinase or tissue type plasminogen activator. Thrombolysis achieves improved regional wall motion in the infarct site and improved global function if the obstruction is subtotal or reperfusion is sustained, and if there are collaterals to the infarct region.[15]

The treatment of survivors of infarcts, whether they suffered angina or were asymptomatic, and of angina in those with stenotic arteries who have not had documented infarcts, is primarily medical and requires judicious use of the wide range of vasodilatory therapies, which improve coronary reserve and platelet antiaggregation.

Invasive intervention by bypass surgery is indicated for patients with intractable angina or restricted quality of life. It has no effect on the risk of infarction or survival rate except in left main artery obstruction in stable angina; it also offers no advantage in terms of risk of infarction or survival rate in unstable angina, except in cases of three-artery disease with impaired myocardial function.[16,17] The status of percutaneous transluminal coronary angioplasty, now widely practiced as a substitute for bypass surgery, or as a concomitant of thrombolysis, is as yet unresolved.[18,19]

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Immunoglobulin A glomerulopathy: Old remedies revisited

Patients with recurrent "synpharyngitic" hematuria displaying diffuse and granular deposits of immunoglobulin A (IgA) and complement (C3) in the glomerular mesangium were originally reported in France in 1968 by Berger and Hinglais.[1] This immunopathologic pattern must be unrelated to systemic lupus erythematosus, Henoch-Schönlein purpura, and alcoholic or obstructive liver problems to be considered diagnostic of primary IgA nephropathy (IgAN), or Berger disease.[2] The histology of IgAN spans the range from normality or minimal mesangial lesions to severe mesangiopathy consisting of expansion (90% of cases), cell proliferation (81%), necrosis (71%) and lysis; glomerular crescents may develop in about one half of the cases.[2-6] With regard to ultrastructural abnormalities, intra- and paramesangial electron dense deposits are common (81%); less often (43%) they compromise the peripheral subendothelium, tubular basement membranes, interstitial matrix, and small arterioles devoid of vasculitis.[2-8] Recent clinical reassessments of the natural course of Berger disease indicate that this glomerulopathy is not entirely benign, since 15–40% of patients may progress to end-stage renal disease (ESRD).[5,8,9] Male children and adults are preferentially affected. In France, the yearly prevalence and incidence reaches 2.5 and 10 per 100,000 for the two groups, respectively, with higher numbers observed in Asian countries. Of 800 children with IgAN reported between 1973 and 1983, 500 were Japanese.[8] In Australia, more than 20% of cases of ESRD, as diagnosed by biopsy, demonstrated IgAN.[4]

In the glomerulopathies of Henoch-Schönlein purpura, systemic lupus erythematosus, and alcoholic liver cirrhosis, deposits of IgA are consistently found.[2,5,7,8] Additionally, an assortment of entities bearing immunologically related pathogenetic mechanisms (including celiac and Crohn diseases, ulcerative colitis, dermatitis herpetiformes, pulmonary hemosiderosis, mucin secreting carcinomas, ankylosing spondylitis, Takayasu disease, and others) show granular mesangial deposits of IgA.[5,8] Clinical and experimental data gathered in the past ten years suggest the glomerular deposition of circulating polymeric IgA-containing immune complexes (IC).[3-5] Immunologic abnormalities—characterized by the presence of systemic IgA-containing IC, high serum levels of IgA, increased numbers of circulating lymphoid tissue IgA-bearing lymphocytes, and elevated T4/T8 cell ratios (as found in tonsils)—lend support to the immunopathogenesis of IgAN.[8-16] Further evidence is provided by the recurrence of IgAN in transplanted kidneys and by the good correlation between the immunofluorescent and electron microscopic studies of the mesangial deposits.[1-5,7,8,13] Immunoproteins eluted from the kidneys of patients with IgAN have demonstrated IgA_2-J chain complex reactive in vitro with the secretory component[2-4,17,18]; this finding, however, has been challenged by others who showed predominance of IgA_1 subclass in IgAN.[12] The data that emanated from these studies suggest an abnormal activation of the mucosal IgA system by unknown antigens.[2]

Alternatively, IC may have formed in situ by the reaction of a specific IgA antibody to antigen(s) lodged earlier in the mesangium.[3] This pathogenetic mechanism provides a more suitable explanation for the severe forms of IgAN. The mesangium, with its unusual histologic and functional properties,[3,19] may suffer relentless inflammation when subjected to heavy antigenic and or IC load. Additionally, whether IC form systemically or in situ, complement may be activated locally via the alternative pathway with subsequent mesangial deposition of C3 concurrently with IgA. That some of the immunologic abnormalities may be genetically predetermined has been suggested by tissue typing studies that correlate the development of IgAN with $HLA\text{-}DR_4$[21,22]; however, despite data demonstrating inheritance of IgAN in some families, the meaning of the $HLA\text{-}DR_4$-glomerulonephritis association, if any, remains controversial.[23]

Also unknown are the mechanisms whereby antigens may gain access to the systemic circulation. Studies in patients with alcoholic liver disease have shed some light on this dilemma. Both serum IgA (90% monomeric) and the polymeric variety (IgA_2-J chain and the SC) have been found in circulating IC and in kidney eluates of patients with liver disease and IgA deposits.[2,24] The serum-exocrine gland secretion concentration gradient of polymeric IgA_2 is maintained in the liver by transepithelial transport processes. IgA_2 synthesized by mucosal lymphoid (plasma) cells is dimerized intracellularly in the presence of J chain. The SC, a cell surface glycopeptide manufactured by secretory epithelia (in the respiratory and intestinal tracts, liver, and elsewhere) joins the IgA dimers by attaching itself to the J chain. Subsequently, the IgA_2-J chain-SC complex is transported to the secretory lumen, where it neutralizes antigens. Thus, mucosal or hepatic derangements may result in antigen release (intestinal bacteria or proteins incorporated in the enterohepatic circulation) and in regurgitation of IgA secretory complex back into the systemic circulation. Concomitantly, levels of circulating IC may increase due to reduced clearance efficiency.[2-4,24]

In summary, the data leave little doubt that IC are one of the pathogenetic mediators of IgAN. In addition, three major immune processes appear to be defective and presumably responsible for the formation and mesangial deposition of such complexes, namely mucosal exclusion of antigens, IC clearance, and regulation of IgA production.[2] Persistence of these abnormalities may lead to progressive glomerular inflammation which, whether acute or chronic, may result in ESRD.

Though the overall impact of IgAN in ESRD is seemingly not as important as that of other immune-mediated glomerulonephritides, such as membranoproliferative glomerulonephropathy (MPGN) and systemic lupus erythematosus, IgAN remains an important source of renal morbidity and mortality for which no efficacious therapies exist.[2-5,8,25,26] Numerous substances have been used in an attempt to control the progression of IgAN; these include immunosuppressive agents (cyclophosphamide, chlorambucil), anticoagulants, and antiplatelet aggregating drugs, diaminophenyl-sulphone-dapsone, urokinase, danazol, and phenytoin.[8,25] Tonsillectomy, long-term antibiotic treatment of infections, and addition of eicosapentanoic acid to the diet have been tried recently[8,25]; results were controversial in the former,[25] and the latter study is still in progress.

Prednisone, widely used in the field of immune-mediated diseases, deserves special consideration. Encouraging results, namely disappearance of hematuria, reduction of proteinuria, and preservation of renal function have been reported in IgAN and other progressive primary inflammatory glomerulopathies treated with steroids.[25,27] McEnery and associates[27] presented substantial evidence of the beneficial effects of prednisone in some patients with IgAN; children placed on short-term treatment improved considerably with recurrence of signs, however, on discontinuation of medication. Reinstitution of prednisone therapy once again induced remission of the urinary abnormalities. Recently, 14 patients with mildly proteinuric IgAN and normal kidney function were treated with prednisone for two to three years by Kobayashi and collaborators.[28] The patients were compared with 29 age- and sex-matched controls who received only nonsteroidal anti-inflammatory and/or antiplatelet-aggregating medications. These latter compounds were used in the prednisone group as well, after discontinuation of steroid therapy. At the end of the follow-up period (about seven years), creatinine clearance was maintained, proteinuria had decreased significantly, and fewer cases of arterial hypertension developed in patients receiving prednisone therapy as opposed to patients without this treatment. The mesangiopathy was attenuated considerably in prednisone-treated patients, whereas among those without the steroid, severe glomerulosclerosis developed that was well correlated with the deterioration of renal function.

The benefits of prolonged high-dose prednisone treatment given every other day by McEnery and collaborators[29] to patients with MPGN are impressive and ought to be evaluated further. These investigators demonstrated significantly increased survival and very low morbidity. One must consider, notwithstanding some differences, that immunopathogenetically, glomerulosclerosis and ESRD of MPGN (particularly Type 1) and IgAN are similar. Thus, it stands to reason that inflammation should be checked, reversed, and maintained in remission by the lengthy use of potent anti-inflammatory medications. Therefore, because of minimal steroidal toxicity, the protocol of McEnery et al,[29] whether used alone or reinforced with other known anti-inflammatory and antiplatelet-aggregation drugs such as dipyridamole and aspirin,[30] should be applied early to IgAN patients. This therapy ought to be maintained for at least five years or longer to prevent the establishment of irreversible glomerular lesions.

Recent developments in nutritional research suggest that diet supplementation with fish oil may be of benefit in patients with immune-mediated inflammation.[31-33] Eicosapentanoic acid, present in fish oil, may act as an important modulator of inflammation; reducing the synthesis of prostaglandins (PGE_2 and TXB_4) by this nontoxic mechanism offers a novel reinforcement to other forms of therapy.[31-33] Lastly, studies by Russell and collaborators[34] showed significant positive correlation between IgA_1 and IgG antibodies to casein in patients with IgAN. In addition, the authors found bovine milk protein antigen in glomeruli of 11 out of 19 patients with IgAN, whereas others had circulating IC containing this antigen. These clinical and experimental studies[35,36] suggest that a closer look must be taken at the pathogenetic role of alimentary tract components (food and bacteria) in IgAN. Children and young adults with IgAN should be tested to determine their reactivity to such antigens, particularly those found in milk. Positive results may be construed as a strong indication that the antigenic material should be removed from or substituted for in the diet to complement the other therapeutic approaches discussed earlier.

It is our distinct impression, however, that these encouraging therapeutic results in IgAN stem from treatment protocols judged by current standards to be unsuitable for drawing conclusions of statistical validity. A systematic, comprehensive, and prolonged prospective study of the salutary effects of prednisone, dipyridamole, and aspirin on IgAN and other similar glomerulopathies in a double blind and randomized fashion is long overdue.

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Deep venous thrombosis of the upper extremity

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ABSTRACT. The purpose of this study was to analyze immediate and long-term results in the treatment of 12 patients with upper extremity deep venous thrombosis (DVT). Six patients were given anticoagulant therapy, and six were not. There were no significant differences in age or length of hospital stay between the two groups. The causes of the DVT included effort thrombosis in four patients, indwelling catheters in four, and idiopathic spontaneous thrombosis in four. Pulmonary embolism did not occur in any of the patients. Only two patients had chronic disability, and both of them had effort thrombosis that was treated with anticoagulants. No long-term disability occurred in those patients who were not given anticoagulant therapy. These data suggest that in this small series the results with anticoagulants for effort thrombosis were poor. Patients with catheter-induced thrombosis or idiopathic spontaneous thrombosis did well without anticoagulant therapy. Anticoagulants may have made no difference in the occurrence of pulmonary emboli.

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Major venous obstruction in the upper extremity usually results from effort thrombosis or indwelling catheters, and sometimes appears spontaneously for no obvious reason. Approximately 1–2% of all cases of deep venous thrombosis (DVT) occur in the upper extremity,[1,2] and post-thrombotic sequelae such as chronic swelling, pain, and edema occur in 17–75% of these patients.[3-5] The recommended treatment of upper extremity DVT is anticoagulation in order to minimize propagation of thrombus and to reduce the risk of subsequent pulmonary embolism. However, treatment with anticoagulants has produced variable results. In young patients with effort thrombosis, more aggressive treatment, including thrombolytic therapy and/or venous thrombectomy, has been recommended in order to decrease the risk of long-term post-thrombotic sequelae.[6,7]

The aim of this study was to analyze long-term results in treating DVT of the upper extremity and to compare patients who did or did not receive anticoagulant therapy.

PATIENTS AND METHODS

The venograms of all patients with suspected deep venous thrombosis of the upper extremity were retrospectively reviewed for a five-year period extending from January 1, 1982, to January 1, 1987. The medical records of those patients with positive venograms were analyzed according to age, length of hospital stay, primary admitting diagnosis, symptoms, physical findings, anticoagulation status, and long-term results. Follow-up data were obtained with the use of questionnaires mailed directly to patients or completed by one of the authors during telephone conversations with patients. Patients were divided into two groups: those who received anticoagulants, and those who did not receive such therapy. The decision to treat with anticoagulants was based on each surgeon's evaluation of the individual patient risk for anticoagulation. Patients with indwelling-catheter-induced thrombosis in an intensive care setting were usually not given anticoagulants because of multisystemic problems. This selectivity may have introduced a bias in the different treatment groups. Those patients who received anticoagulant medication were given intravenous heparin to maintain their partial thromboplastin time at one and a half to two times control for one week, and were then maintained on oral crystalline warfarin sodium for three months.

The Student's t-test and Fisher's exact test were used to analyze differences between the two groups.

RESULTS

Thirty-seven patients underwent upper extremity venograms for suspected DVT. Twelve patients had positive venograms. Six of the 12 patients were given anticoagulants and six were not (Table I). The mean age of the entire group was 50 years. Among patients not given anticoagulants, it was 57 years, and among those given anticoagulant therapy it was 43 years. Although there was a 14-year mean difference in age between the two groups, this difference was not statistically significant. The mean hospital stay was 17 days for the patients given anticoagulants and 30 days for the other group. Patients with positive venograms who did not receive anticoagulant therapy had the longest hospital stay. No statistically significant difference existed between the groups. At initial presentation, all patients had some degree of swelling; five complained of pain in the involved extremity, four had cyanosis, and one had numbness. The cause of the subclavian vein thrombosis was effort thrombosis in four patients, indwelling subclavian catheters in four, and spontaneous idiopathic thrombosis in four. All of the four patients with effort thrombosis had been given anticoagulants, and two patients with metastatic cancer died during hospitalization. There were no bleeding complications in the group on anticoagulant therapy. Pulmonary embolism did not occur in any of the patients.

The average time from the onset of swelling to resolution was 12 days in all patients, with no difference between the two groups. At follow-up, two patients had chronic disability; both had effort thrombosis and both had received anticoagulant therapy. One of these patients had chronic swelling with limitation of motion, and the other had limitation of motion with paresthesias in the involved extremity. Neither patient showed evidence of a cervical rib as a

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TABLE I. Characteristics of 12 Patients with Upper Extremity Deep Venous Thrombosis

Patient	Age (yr)	Presentation	Diagnosis	Cause of DVT	Anticoagulants Given	Hospital Stay	Disability	Length of Follow-up
1	49	Swelling	Adenocarcinoma of the cervix	Indwelling catheter	No	Two months	None	Two years
2	65	Swelling	Lung cancer	Indwelling catheter	No	Two months	None	Two months
3	59	Swelling, cyanosis	—	Spontaneous	No	Three days	None	One year
4	83	Swelling, cyanosis	Pacemaker	Indwelling catheter	No	Outpatient	None	Two years
5	52	Swelling, cyanosis	Lung cancer	Spontaneous	No	Four weeks	None	Four weeks (died)
6	34	Swelling, cyanosis, pain	Hodgkin disease	Spontaneous	No	Six weeks	None	Three months
7	23	Pain, swelling	—	Effort	Yes	Seven days	Paresthesias, decreased range of motion	Two years
8	32	Pain, swelling	—	Effort	Yes	Five days	Chronic swelling, decreased range of motion	Six years
9	66	Pain, swelling	—	Effort	Yes	12 days	None	Three years
10	20	Pain, swelling	Systemic lupus erythematosus	Spontaneous	Yes	30 days	None	One year
11	32	Pain, swelling	—	Effort	Yes	Five days	—	Lost
12	84	Swelling	Bowel obstruction	Indwelling catheter	Yes	Six weeks	None	Three months

possible cause of thoracic outlet syndrome. One patient was lost to follow-up. There were no recurrences of DVT with a mean follow-up of 25 months (range, 3-72 months).

DISCUSSION

The treatment of deep venous thrombosis of the upper extremity remains a controversial subject. The data presented here suggest that in this small group of 12 patients not prospectively randomized, anticoagulation made no difference in long-term morbidity or subsequent pulmonary embolism. No patient in either group developed clinical evidence of pulmonary embolism or had a recurrence of symptoms in the post-thrombotic period. Of the two patients with chronic disability, both had effort thrombosis and both had received anticoagulant medication.

Tilney et al[5] reported 48 cases of upper extremity DVT with no pulmonary emboli, and Barnett et al[8] reported 300 cases with only one episode of a pulmonary embolism. The data reported here are in agreement with those of earlier reports. However, these results contradict those of Harley et al,[9] who found that pulmonary emboli developed in five of 14 patients with DVT of the upper extremity, and Donayre et al,[6] who reported that in 12% of 41 patients he studied, pulmonary emboli subsequently developed. Interestingly, in the study by Donayre et al, all of the patients in whom pulmonary emboli developed had received adequate amounts of heparin at the time the emboli occurred.[6] Similarly, Campbell et al[4] reported that two of 12 patients who had fatal pulmonary emboli were on heparin. Based on our data and those of others, it would seem that pulmonary embolism rarely occurs in these patients. In addition, when the rare pulmonary embolus occurs, it does so despite adequate anticoagulation.

The limitation of all these studies is that lung scans were not routinely done to document silent pulmonary infarctions, so that the true incidence of pulmonary emboli in this setting is unknown. More importantly, a large number of patients with upper extremity thrombosis would have to be identified to determine if anticoagulation made a significant difference in the occurrence of pulmonary emboli.

Anticoagulation is used in DVT to prevent the extension of clot and subsequent thrombosis of collateral vessels around the site of obstruction. It is postulated that by preventing further collateral vessel thrombosis, venous outflow will be better and long-term morbidity lower. In DVT of the upper extremity, the accuracy of this hypothesis is questionable. Donayre et al[6] reported that 50% of patients in their study had significant disability, especially if effort thrombosis was the underlying cause. They found that anticoagulation did not affect outcome vis a vis disability. Adams et al[7] also reported that 70% of patients with effort thrombosis had significant disability despite anticoagulation. Tilney and colleagues[5] likewise found residual disability in 74% of 48 cases despite anticoagulation in 52% of cases. Because of poor results with anticoagulation, both Donayre et al[6] and Adams et al[7] recommended aggressive treatment of effort thrombosis with thrombolysis and venous thrombectomy.

The data presented here confirm that effort thrombosis leads to long-term disability (50% of cases) despite anticoagulation. In addition, in this study, those patients with catheter-induced thrombosis and those with spontaneous idiopathic thrombosis did well without anticoagulation. The significant limitation of this study is that the treatment groups differed with regard to the cause of the thrombosis, which would introduce a bias when comparing the two treatment modalities.

We conclude that the decision to use anticoagulants to treat patients with upper extremity deep venous thrombosis should not be applied in all cases. In addition, the effect of anticoagulant treatment alone may not alter long-term disability without such adjunctive therapies as surgical decompression or systemic lysis. A prospective, randomized study of patients with upper extremity deep venous thrombosis is needed to help further resolve current therapeutic controversies.

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Membership survey of the New York State Academy of Family Physicians

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ABSTRACT. A questionnaire distributed to the 1,445 members of the New York State Academy of Family Physicians (NYSAFP), was designed to identify characteristics of the group as well as the professional needs of its members. The response rate to this anonymous survey was 39.8%. The results show that younger members were more involved in teaching and administration. Female respondents were more likely to be in salaried positions. Salaried physicians had less knowledge about the academy, and both this group and female respondents frequently thought that other organizations better represented their professional interests. The leading professional concern among the respondents was continuing education. Our findings provided the academy with suggestions regarding recruitment, education, developing better awareness of the NYSAFP, continuing education programs, the importance of involving more members—young and old—in activities of the organization, and the need for communication between the membership and leaders of the organization.

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Professional organizations have existed for as long as professions themselves. The rationale for the existence of these organizations was, from the beginning, maintenance of the prerogatives of the specific profession. Currently, professional organizations serve their memberships in an ever-increasing variety of ways. It is these membership services that keep the organizations viable.

Medical organizations function in a rapidly changing environment. Increased government involvement in the distribution of health care services as well as mounting interest by the business community in bringing down the costs of health care are pressuring physicians to change the ways in which they practice. Medically sophisticated consumer groups also participate through the government as well as through their own buying power in bringing about constructive changes in health care delivery systems. Medical organizations need to recognize these new interest groups and their influence on the health care system.

Young physicians expect their professional organizations to be more than just representative of the status quo. Many physicians now have different goals and ideals as well as different work environments from their predecessors. The differing needs of younger physicians must be recognized by organizations in order to satisfy their membership.

Medical organizations have the reputation of being slow to change. Leadership has often spent many years coming up through the ranks, and when they finally become leaders, they often conform to the precepts of the organization. If membership is to be maintained, however, there must be a modernization of thought and an awareness of new directions and objectives. Organizations need to find out what the membership is thinking and to reevaluate their goals continuously. In addition, ongoing communication must be maintained to allow for an exchange of thought and the development of trust between the membership and an organization's leadership.

The New York State Academy of Family Physicians (NYSAFP) recognized the need to obtain information from its membership in order to accomplish both short-

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TABLE I. A Comparison of the Demographic Characteristics of the Survey Sample (N = 575) with Those of the Full Membership of NYSAFP (N = 1,445)

	Survey Sample (%)	Membership (%)
Age (yr)		
Under 40	49.3	44.9
41–50	11.5	12.7
Over 50	38.9	42.3
Sex		
Male	89.0	87.0
Female	11.0	13.0

and long-term planning. It was thought that the most helpful information would include membership demographic data, members' perceptions of the organization, and what the membership perceived as their current needs.

To obtain this information, a questionnaire was prepared and administered to all active members. The objectives were to identify issues and needs, develop a view of the organization from the perspective of its membership, obtain suggestions for short- and long-term planning for the leadership, initiate a mechanism to track membership opinion, and identify members interested in becoming more active in specific areas in the NYSAFP.

METHODS

A questionnaire containing 29 questions was prepared for use in this study. A cover letter accompanied the questionnaire stating the objectives and informing the members that, if they wished, the questionnaire could be completed anonymously. Trial completion of the questionnaires by family physicians in a local group demonstrated that there was minimal ambiguity and that no more than 20 minutes was required to complete the four-page questionnaire.

The questionnaire was then sent to all 1,445 active members of the NYSAFP. Within two months, 575 questionnaires (39.8%) were returned with the requested information. On receipt of the completed questionnaires, the information was entered into a microcomputer.

RESULTS

Most of the questionnaires returned contained complete and appropriate responses. The response rate was comparable to that of other physician surveys completed since 1980. A Canadian Medical Society survey in 1984 elicited a response rate of 45.7%,[1] a Pennsylvania Medical Society survey in 1980 obtained a response rate of 50.0%,[2] and a Michigan Medical Society survey in 1980 received a 57.0% response.[3] The percentage responses noted in the following sections are percentages of the total responses to a specific question. The actual number of completed item responses ranged from 493 to 575.

An analysis was made to determine whether the 575 respondents were representative of the total organization. A comparison by age and sex of the practitioners in the sample and the organization is presented in Table I.

Comparisons by years of practice and type of practice are presented in Table II. A comparison between the male and female respondents showed that 50.8% of the females had been in practice for fewer than five years, but just 29.0% of the male respondents had been in practice for the same amount of time. Only 15.9% of the female respondents had been in practice more than 20 years, while 40.0% of the male respondents fell into this category.

Slightly more than 87% of the respondents said they were in private practice, 37.9% also participated in some type of teaching activity involving medical students and residents, and 24.0% noted that a significant amount of their work involved administration. Looking more closely at practice activity, Table II shows that younger physicians were more involved in teaching and administration than the older group.

Table III also shows methods of reimbursement by years of practice. It is clear that younger physicians are more involved in salaried and private prepaid work than are the physicians who have been in practice for a longer time period. Pay source grouped by years in practice shows that 26.8% of the respondents in the 0-5 years of practice category have salaried positions. This compares with the results of a 1984 American Medical Association study[4] which showed that 27% of doctors with less than six years in practice were salaried, compared to 10% of those with more experience. The Family Practice News Survey,[5] which had 1,175 randomly chosen family

TABLE II. Distribution of Respondents by Age and Practice Type (N = 575)*

	Total		Teaching		Administration		Private Practice	
Years in Practice	N	%	N	%	N	%	N	%
0–5	175	30.4	87	49.7	45	25.7	134	76.6
6–10	118	20.5	52	44.0	36	30.5	104	88.1
11–20	54	9.4	28	51.9	15	27.8	50	92.6
21–30	115	20.0	30	26.1	24	20.9	110	95.7
31–50	113	19.7	21	18.6	18	15.9	104	92.0
Total	575	100.0						

* Respondents could choose more than one practice type.

TABLE III. Practice Types by Years in Practice*

	Total		Fee for Service		Private Prepaid		Salaried		Other	
Years in Practice	N	%	N	%	N	%	N	%	N	%
0–5	175	30.4	114	65.1	4	2.3	47	26.8	14	8.0
6–10	118	20.5	90	76.3	13	11.0	28	23.7	2	1.7
11–20	54	9.4	46	85.2	0	0	9	16.7	4	7.4
21–30	115	20.0	108	93.9	0	0	5	4.3	5	4.3
31–50	113	19.7	104	92.0	2	1.8	12	10.6	6	5.3
Total	575	100.0								

* Respondents could choose more than one practice type.

TABLE IV. Practice Type by Sex of Practitioner[*]

Sex	Fee for Service N	Fee for Service %	Practice Prepaid N	Practice Prepaid %	Salaried N	Salaried %
Male[†]	424	75.6	12	2.1	73	16.9
Female[†]	38	52.1	7	9.6	28	38.4

[*] Respondents could choose more than one practice type.

[†] Difference between males and females is significant by t test ($p < 0.001$).

physicians responding, reported that 18% of the respondents were salaried. Eight and a half percent (8.5%) of the physician respondents reported spending more than 60% of their practice time on prepaid health care, and 17.4% reported spending greater than 30% of their practice time in prepaid health care.[5] These percentages are higher than in the present survey, probably because there is little prepaid health care in New York State.

A comparison between male and female physicians grouped according to the type of practice in which they are involved appears in Table IV. The larger percentage of female physicians in salaried positions and private prepaid practice is significantly different from that of their male counterparts ($p < 0.001$). This finding is only partially explained by the younger age of the female physicians. A comparison of similarly aged male and female physicians shows a statistically significant difference in practice type. Data from the Family Practice News Survey[5] showed that 37% of the female respondents were salaried and 24% of the females were associated with a closed panel health maintenance organization (HMO).

Some questions asked for the members' perceptions of the NYSAFP and whether or not it fulfilled their perceived professional needs. The questionnaire asked about the members' perceptions of their knowledge concerning the activities and the resources of the NYSAFP (Table V). Of the 575 respondents, 20.1% answered that they had a "very high" or "high" level of knowledge about the academy. Another 45.4% said that they had an "average" level of knowledge, and 34.8% said that they had a "low" or "very low" level of knowledge. It should be noted that the level of the knowledge of NYSAFP activities among the younger physicians appeared to be lower than that of the older group. The level of knowledge among different practice type groups varied slightly, but the differences were not significant.

When asked about their perceptions of their use of NYSAFP resources, 9.1% of the respondents answered "very often" or "often," 24.5% answered "sometimes," 45.5% answered "seldom," and 20.1% answered "never." Among individual categories such as years in practice, age, sex, and practice type, strong correlations were found between level of knowledge of NYSAFP activities and resources, and the perception of the level of resource utilization.

Tables VI and VII show whether the NYSAFP represents the interests of its members according to primary practice type and sex of the physicians, respectively. Salaried physicians and female physicians (clearly an overlapping group) showed more disagreement with the idea that organized family practice represented their interests. The questionnaire specifically asked if any other organizations represented the respondents' professional interests better, and 29.3% of respondents said "yes." In both of these categories of physicians, there were significantly different responses. More physicians in private prepaid practice and in salaried positions, and more female physicians, stated that other organizations represented their professional concerns better.

Table VIII shows the physicians' concerns or needs. The primary professional concern among physicians in all age and years in practice categories was continuing education, except in the age group 46–50 years, in which it numbered second. Younger physicians said they had a strong interest in practice management, how the profession affected the physician's family, and in teaching family practice. Physicians in different practice type groups also listed continuing education as the highest professional concern, with salaried physicians being highly concerned with teaching family practice. Interestingly, salaried physicians listed government regulation as a fairly low professional concern.

Other observations from the questionnaires revealed that activity in local or county family practice organizations correlated with both knowledge of the activities and resources of the NYSAFP and perceived use of these resources. In addition, a question about optimal local meeting times showed high preferences for midweek evening meetings. The most preferred frequency was quarterly (four times per year). In an open-ended final question, a number of members expressed an interest in participating in one or more of the standing committees or commissions of the organization. Some respondents stated that new committees were needed and listed suggestions.

DISCUSSION

Criticism is often leveled at professional organizations in general, and at medical organizations in particular, for not keeping up with changes in the characteristics and

TABLE V. Knowledge of the Resources of the NYSAFP by Years in Practice (N = 575)

Years in Practice	N	High Knowledge N	High Knowledge %	Average Knowledge N	Average Knowledge %	Low Knowledge N	Low Knowledge %
0–5	175	19	10.9	77	44.0	79	45.1
6–10	118	20	16.9	47	39.8	51	43.2
11–20	54	9	16.7	26	48.1	20	37.0
21–30	115	28	24.3	61	53.0	26	22.6
31–50	113	33	29.2	52	46.0	27	23.9

TABLE VI. Perception of Representation of Interests by the NYSAFP and Other Organizations by Practice Group

	Represents Interests						Other Organizations[*]			
	Agree		Unsure		Disagree		Yes		No	
Practice Type	N	%	N	%	N	%	N	%	N	%
Fee for Service (N = 462)	346	74.9	97	21.0	19	4.1	107	22.0	360	78.0
Prepaid Health Care (N = 19)	12	63.2	7	36.8	0	0	8	42.0	11	57.9
Salaried (N = 101)	72	71.3	23	22.8	6	5.9	44	43.6	57	56.4

[*] Difference between fee for service respondents and those in prepaid private practice and salaried positions was significant at $p < 0.01$ by chi square.

TABLE VII. Perception of Representation of Interests by Other Organizations by Sex*

Sex	Yes N	Yes %	No N	No %
Male (N = 512)	145	28.3%	367	71.7%
Female (N = 63)	27	42.9%	36	57.1%

* Difference between males and females significant at $p < 0.01$ by chi square.

needs of their membership. Many mechanisms may be used to initiate a dialogue between membership and leadership. A survey helps to gather useful information as well as establish a policy of continuous contact and feedback. "Nothing aids sound planning for the future more than accurate information about present circumstances."[2]

Respondents were representative of the NYSAFP membership. The distribution of respondents among the variables of age, years in practice, and sex was not different from that of the general membership. As was found in the 1980 Pennsylvania Medical Society survey,[2] appeals to increase membership may need to vary according to age and practice type. Each group requires a different approach.

In the present survey, 34.8% of the respondents thought that they had a "low" or "very low" level of knowledge about the resources and activities of the NYSAFP. Younger physicians made up the largest segment of this group, with salaried physicians demonstrating the least knowledge of the professional organization. This is similar to the results of the 1985 Canadian Medical Society survey,[1] in which younger physicians and female physicians were found to be less aware of the group's policies.

There was strong agreement that organized family practice represented the interests of the membership, but when asked if there are other organizations that represent professional interests better, 29.3% of all respondents said yes. Almost 50% of the women felt this way as compared to about 28% of the men.

Professional concerns of the membership in all practice types and age groups clearly highlighted continuing education. Although many educational programs exist, perhaps they are not well enough directed to the practicing family physician. Other professional concerns varied slightly among the respondents according to age and practice type. Younger physicians were more concerned with issues revolving around teaching family practice and how their profession affected their family life.

Any attempt to gain information about the perceptions and needs of the membership enhances the ability to do long-term planning. The NYSAFP is going through this process and is using the information gathered from this questionnaire to help determine priorities and communication needs. Important objectives include the need to use different approaches toward recruitment and education among different physician groups, educate younger physicians about the NYSAFP, review the continuing education programs available to family physicians and to fulfill this need in a timely and appropriate manner, directly involve more members in the activities of the organization (the greater the involvement, the more positive the image of the organization), satisfy the older members while moving on to issues concerning the younger physicians, and initiate and maintain communication between the membership and the leaders of the organization.

TABLE VIII. Summary of Professional Concerns as Noted by Respondents

Concern	% Respondents
Continuing education	62.3
Future direction of medicine	56.2
Status of family practice	51.4
Government regulation in medicine	40.1
Practice management	37.8
Effect of profession on family	36.1
Political concerns (ie, workman's compensation, insurance, etc)	32.7
Hospital privileges	26.5
Teaching family medicine	26.0
Public relations	13.2

The American Medical Association initiated a forum for salaried physicians at its annual meeting, recognizing that more than 40% of physicians receive some kind of salary,[4] and the Canadian Medical Society has made communication a high priority.[1] One result of the American Medical Association's meeting was the formation of a "Young Physician's Section." This signaled recognition of the need for medical organizations to be different things to different members. The American Medical Association is considering providing expert assistance on contracts, seminars on managerial and administrative skills, more meetings without registration fees, and additional efforts to open lines of communication.[4] Communication is an essential element for the success of medical organizations of all sizes.

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Computer assistance with information needs in clinical medicine

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Computer technology is capable of assisting physicians in dealing with the overwhelming quantity of medical information needed to make appropriate diagnostic and therapeutic decisions. Computer assistance can provide physicians with access to patient care data (medical records), medical knowledge, and expert consultation. Although a multitude of medical software programs are being aggressively marketed to physicians and hospitals, few have had adequate testing and validation. However, medical computing systems are being developed and evaluated at many academic centers. A review of clinical computer programs that address the information needs of the internist and that have published capabilities or effectiveness was undertaken. These programs are categorized according to the information needs addressed, and the potential utility of each program is discussed.

In a recent study, Covell et al[1] evaluated the information needs of a number of physicians who practice internal medicine in Los Angeles County. A striking 70% of informational questions raised by the physicians at the time of the patient visit went unanswered. The informational questions raised were categorized as medical opinion, 43% ("What is the best way to diagnose gout?"); medical fact, 40% ("What are the reported side effects of propranolol?"); additional information, 17% ("How can I get this patient a home health aide?"). There were no differences between these categories of questions asked by generalists compared to those asked by specialists. When these same physicians were asked to name information sources they would like to have in their offices, the following items were at the top of the list: newer textbooks, computers to answer diagnostic and treatment questions, better organization of existing medical literature, and computerized literature reviews. Such studies reveal the need for organized, accurate, and easily available information. Unfortunately, previous studies have shown that availability of information is ranked above accuracy in selecting a source when information is needed.[2]

The medical information needs of physicians can be divided into several categories: patient care data (the medical record with its laboratory data, radiology reports, etc); organized bibliographic information of the medical literature (Index Medicus); knowledge bases (textbooks of medicine); medical expert systems (artificial intelligence approaches that mimic expert consultation and decision making); time-oriented clinical databases (patient data from clinical research projects, eg, the Framingham Study); protocol management (reminders for periodic health examinations; algorithms, particularly for nonphysician clinicians). We have categorized medical software according to the information needs provided for by the software. In addition to describing the information capabilities of a number of medical software programs, key questions and considerations a physician can use as a guide in selecting software are delineated.

A summary of the most developed computer aids for clinical medicine and their capabilities appears in Table I. Some of these computer programs function in more than one domain. This listing of computer aids to clinical medicine is not exhaustive. Those listed have received the most critical attention to date.

PATIENT CARE DATA SYSTEMS

Computer-based medical records systems have been developed in many academic health centers. These include COSTAR,[3] Regenstrief Medical Information System,[4] PROMIS,[5] HELP,[6] and the Beth Israel Hospital program in Boston.[7] In addition to providing a computer record of the medical encounter and administrative functions, such as billing information, many of these medical information systems have additional features to help the clinician.

COSTAR (Computer-Stored Ambulatory Record) was originally developed by Barnett and colleagues[3] at the Laboratory of Computer Science at the Massachusetts General Hospital. COSTAR is a comprehensive ambulatory medical record system that has been commercially marketed. Clerical personnel transcribe data from a special encounter form into the computer system. In addition to providing some administrative functions and a record of the medical encounter, special reports can be generated, including case summaries, flow charts, and statistical summaries of visit patterns or other data on specified groups of patients). Many sites that have installed COSTAR have added subsystems that generate reminders to

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TABLE I. Computer-Based Medical Knowledge and Information Systems*

Information Need	Brief Description	Knowledge Base	Advantages	Disadvantages	Comment
Medical Expert Consultant					
INTERNIST-1/ Caduceus[22]	Access difficult; system for diagnosis in general internal medicine; 600 profiles in knowledge base	Expert consultant internal medicine diagnosis	Particularly good with difficult cases; medical education potential	Long interaction time; knowledge base incomplete; no explanations	Not yet available; knowledge base needs expansion
Knowledge Couplers[25]	General medicine knowledge base that can be coupled with patient findings to aid in diagnosis, treatment and monitoring	Internal medicine diagnosis and treatment	Can be used by nonphysician clinicians; can substitute as the medical record; medical education potential	Knowledge base currently small	No external validity documented; knowledge base needs expansion and maintenance
ONCOCIN[27]	Advice on chemotherapy protocols for cancer patients; a form of computer-based medical record	Cancer chemotherapy	Increases generalist capabilities in chemotherapy; high quality advice; includes patient care data; medical education potential	Specialized domain; difficulties with protocol delays	Not available for general use
AI/RHEUM[32]	Aids in the diagnosis of rheumatologic diseases	Criteria for diagnosis of 26 rheumatologic diseases	Increases generalist capabilities of diagnosis; medical education potential	Specialized domain; knowledge base incomplete	Not available for general use
MYCIN[31]	Aids in antibiotic selection for severe infection	Diagnosis and treatment of bacteremia and meningitis	Increases generalist capabilities in diagnosis and treatment; medical education potential	Specialized domain; knowledge base incomplete	Not available for general use
ABEL[30]	Aids in the diagnosis and management of acid-base and electrolyte disorders	Acid-base and electrolytes	Attempts to model the patient physiology	Specialized domain	
Knowledge Base (Electronic Textbook)					
QUICK[19]	Ready access to large knowledge base with differential capabilities	Internal medicine diagnosis (the INTERNIST-1 knowledge base)	Easy access, rapid interaction time; large knowledge base; implementation relatively easy	Knowledge base incomplete; no therapy advice; no explanations	Not available for general use; being tested outside development site; knowledge base needs expansion and maintenance

(*continued*)

Table I continued

Information Need	Brief Description	Knowledge Base	Advantages	Disadvantages	Comment
Patient Care Data Systems					
COSTAR[3]	Comprehensive outpatient medical record system with rule-based reminders for health maintenance	Patient data bank and rules re outpatient care	Substitutes for medical record; quality assurance capable; population analysis	Implementation system-wide; clerk enters data; limited knowledge base	Available; capable of many administrative functions
REGENSTRIEF[4]	Total hospital information system; rule-based physician reminders which have improved patient care; interactive	Patient data bank and rules re patient care	Substitutes for medical record; reminders are for health maintenance and specific care situations	Implementation system-wide; limited knowledge base	Available; can implement modules, eg, pharmacy, laboratory
PROMIS[5]	Problem oriented medical records system; interactive	Patient data bank	Substitutes for medical record; increases capabilities of nonphysician clinician	Implementation system-wide; knowledge base capabilities are incomplete	Not available for general use
HELP[6]	Hospital information system with primary care algorithms; interactive	Patient data bank and primary care algorithms	Substitutes for medical record; increases capabilities of nonphysician clinicians	Implementation system-wide; limited advice knowledge base	Available

* The programs listed represent some of the better documented ones which have diagnosis or treatment advice and/or patient information. Many other programs exist or are being developed. Programs listed (except PROMIS and Knowledge Couplers) have been subjected to some comparison with an external standard (case series, randomized clinical trial, comparison with human experts).

identify and schedule patients for health maintenance procedures, such as flu vaccine and routine cancer screening. In this manner, COSTAR has quality assurance capabilities. It is possible to have COSTAR provide rule-based advice on a number of clinical issues as well as being a computer-based medical record. For example, patients aged 65 or older require annual influenza vaccination. COSTAR is an extremely flexible system written in the MUMPS language. COSTAR requires a mainframe or "minicomputer," considerable storage capabilities, and data entry by a technician. Nevertheless, flexibility and 18 years of development and experience with COSTAR have led to its being installed in more than 100 sites.

The Regenstrief Medical Information System (RMIS), developed in Indiana,[4] is primarily an ambulatory care information system which stores information from patient encounters. Physicians must use special optically scanned data entry forms. RMIS is noted for its computer-generated reminders to physicians. In addition to health maintenance reminders, protocols are included with regard to patient care. For example, the computer can remind the physician to start antacid therapy on a patient who has just been started on high-dose aspirin therapy. McDonald has conducted several studies that show that such reminders are effective in improving the quality of care.[3,8] A recent clinical trial demonstrated improved quality of preventive care, including occult blood testing for gastrointestinal cancer, mammography, weight reduction, and immunizations.[9] The system has a growing medical knowledge data base which will be able to provide additional diagnostic and treatment advice. RMIS runs on a VAX (Digital Equipment Corporation) minicomputer system.

PROMIS is a medical records system that uses a problem-oriented approach to clinical recording and management.[5] It was developed in Vermont and has been used by physicians and nurse practitioners. The clinician enters the findings of patient encounters directly at the computer terminal through an interactive program. The program helps organize information and develops a list of patient problems and suggestions for possible management steps. Further development is needed prior to widespread distribution of this system. It is not currently functioning effectively in a medical setting.

The Beth Israel Hospital system is a hospital-wide clinical computing system that provides records of laboratory data, radiology and pathology reports, demographic data, drug profiles, and bibliographic search capabilities through MEDLINE.[7] It has been useful and has proven acceptable to clinicians at the hospital.

There are several other clinical information systems that have been developed and are in use. The Medical Record (TMR) at Duke University,[10] the Summary Time-Oriented Record (STOR) at University of California Medical Center at San Francisco,[11] and DUCHESS at the University of South Carolina and Duke University[12] are further examples.

Other considerations with automated medical record systems include the need for technical support from vendors or local personnel. Furthermore, considerable programming experience is required to modify programs to meet local needs. There are no well-defined methodologies for cost justification of such systems. Barnett reports minimal initial costs for COSTAR as low as $15,000 to $25,000 for a group of less than five physicians and $200,000 for a group of 15 to 40 physicians.[13]

BIBLIOGRAPHIC SEARCH

Many software programs provide capabilities to search the medical literature by subject or author. Usually, abstracts of articles can also be printed. MEDLARS[14] and PaperChase[15] are two examples of programs that access MEDLINE. A recent evaluation of 14 different software packages that access MEDLINE found that all are able to identify critical articles in the literature. However, more costly packages provide lower quality results.[16] Full-text medical literature retrieval by computer is becoming available.[17,18] Access to these systems requires a computer terminal or a microcomputer with a telephone modem for dial access.

Computer-assisted bibliographic search can identify important sources of medical information. However, clinicians must then obtain, read, understand, and apply the content to their practices.

KNOWLEDGE BASES (ELECTRONIC TEXTBOOKS)

Several knowledge databases have been developed for use as electronic medical textbooks. A medical knowledge database typically contains information about diseases and medical conditions. The classic medical knowledge database is a textbook of medicine. Medical knowledge can be entered into a computer database, and more sophisticated indexing and information sorting capabilities can be added. The Quick Index to Caduceus Knowledge (QUICK),[19] CONSIDER and RECONSIDER,[20] and the National Library of Medicine Hepatitis Knowledge Base[21] are examples.

QUICK is a software program under development by Miller and colleagues at the University of Pittsburgh.[19] QUICK provides easy access to the knowledge database of the INTERNIST-1/CADUCEUS computer-based expert consultant system (see below).[22] QUICK functions as an electronic textbook of medicine, a differential diagnosis generator, and a teaching tool. It has artificial intelligence capabilities in the form of heuristics that help to restrict the length of probable cause lists and provide estimates of likelihood of findings for given diseases and vice versa. Each finding or disease manifestation is given an "evoking strength" and a "frequency" score for the diseases listed in the differential diagnosis. The evoking strength is an indication of how specific the finding is for the disease. Hence, the highest evoking strength is assigned to pathognomic findings. The frequency score indicates how frequently patients with the disease will have this finding (similar to sensitivity of a test). Although the knowledge database used contains profiles of approximately 600 individual diseases, it is estimated that an additional 200 disease profiles should be added to the current database.

Miller and colleagues[19] have evaluated the use of the QUICK program at three Pittsburgh area hospitals, using a telephone modem hook-up to a VAX (Digital Equipment Corporation) minicomputer. Problems encountered by physician users of the system can be categorized as follows: lack of understanding of how to use the QUICK commands (33%); confusion of diagnoses with disease manifestation names (11%); spelling errors (13%); difficulty in phrasing medical terms so they are compatible with the QUICK program (33%); disease or manifestation not part of the knowledge base (9%). The overall success rate for interactions (meaningful information was obtained) with QUICK at all three hospitals was 65%.[19]

Although there were no differences in success rates between hospitals, users at one hospital (a community hospital) were much less enthusiastic about the QUICK program than those at the other two hospitals (a university teaching hospital and another community hospital). Most inquiries required from one to ten minutes of user time. Since the evaluation, Miller has developed a dictionary and phrasing program that helps to limit spelling and phrasing errors considerably. Miller has expanded the differential diagnosis capabilities of QUICK to the point where its successor, QMR, actually functions as a type of medical expert system. Further evaluation of the QMR program is underway at sites outside the Pittsburgh area.

QMR has multiple functions available: case analysis mode; display of a disease profile; display of the differential diagnosis of a single finding; associating a finding to a group of diagnoses; reviewing disease profiles classification hierarchy; listing diagnoses not currently included in the knowledge base. There are multiple subfunctions within these functions. For example, the case analysis mode includes a ranking of most attractive diagnoses, listing a single diagnosis or two diagnoses that explain all findings, the generation of a global hypothesis that has several concomitant or contributory diagnoses, and the ability to critique a diagnosis.

The dictionary and phrasing program allows "shorthand" keyboard entry of findings. For example, typing LY NO EN summons the list of numbered choices for different types of LYmph NOde ENlargement. QMR runs on a microcomputer.

RECONSIDER and portions of AMA/NET are computerized versions of existing medical textbooks without editing or artificial intelligence aspects. Recently, AMA/NET has been linked with DXplain.

DXplain is a more recently available decision-support system developed by Barnett and colleagues.[23] Barnett has wisely described the system as an "evolving computer-based diagnostic decision-support system."[23] DXplain is available for use, in its current stage of development, through AMA/NET and through the Massachusetts General Hospital Continuing Education Network. Among other functions, DXplain can accept a list of clinical manifestations and then suggest diagnostic hypotheses with explanations. The proposed diagnoses are grouped as "common diseases" and "rare diseases." Serious diseases (requiring prompt action) are also identified. The development of DXplain was strongly influenced by INTERNIST-1 and QMR.[23] The knowledge base was derived from Current Medical Information and Terminology, a computer-based information source published and supported by the American Medical Association.[24] The knowledge base can be rapidly updated or revised, since DXplain resides on a single, mainframe computer. A user can access DXplain through a computer terminal or by using a microcomputer with a telephone modem for dialing access. Though no formal evaluation of effectiveness has been conducted, anecdotal reports from more than 40 test sites are favorable.

Computer-based medical knowledge databases will have the greatest impact on patient care if physicians find them to be quick, easy, and informative, and if the computer can assist the physician in sorting and weighing the clinical findings. Theoretically, this form of computer assistance can improve physician knowledge and decision making in such a way that utilization of resources (diagnostic tests, consultation) is more selective.

MEDICAL EXPERT SYSTEMS

Computer-based medical expert consultation systems have been in development for nearly 30 years, yet none has obtained widespread acceptance. Although there is some variation in definitions, medical expert consultation computer systems generally attempt to mimic the information sorting process and capabilities of a human expert consultant. Specific findings in a medical diagnostic case are weighed as evidence for or against the presence of a particular diagnosis or disease. The computer-based expert consultant can sort the findings and determine the most likely diagnosis and identify any competing diagnosis. INTERNIST-1/CADUCEUS,[22] QMR, and Weed's Knowledge Couplers[25] can be considered forms of expert systems for the generalist. There are many systems for specialized areas: PUFF for pulmonary function testing,[26] ONCOCIN for cancer chemotherapy,[27] and CONSULT-I for acid-base and electrolytes.[28] Each of these has proven effective in the specialized area of use and continues to be improved and tested.

INTERNIST-1/CADUCEUS is an experimental expert consultation system with a large knowledge base that profiles approximately 75% of the diseases pertinent to internal medicine.[22] A physician can enter subjective and objective data with regard to a patient, and the artificial intelligence capabilities of the program take over and construct a differential diagnosis. Heuristic principles in the program, designed to model physician behavior, identify the most likely diagnosis(es) and eventually determine the most attractive diagnosis.

INTERNIST-1 capabilities were evaluated using its performance on a series of 19 clinicopathologic conferences (CPCs) taken from the case records of the Massachusetts General Hospital published in the *New England Journal of Medicine*.[22] (Note, CPCs typically involve extremely complex and difficult-to-solve diagnostic problems. Evaluating the performance of a computer-based system by using CPCs as test cases does not necessarily measure its performance in the clinical setting.) Within the 19 CPCs there were 43 possible correct diagnoses. INTERNIST-1 failed to make the correct diagnosis in 18 instances; the clinicians on the case failed in 15 and the human expert discussant failed in eight cases. INTERNIST-1 made the correct diagnosis in seven instances in which both the clinicians and the expert discussant failed. Overall, INTERNIST-1 performed extremely well in this evaluation.[22]

There are difficulties with the INTERNIST-1 program, many of which are common to other expert consultant programs.[29] Physicians interacting with the program must be familiar with the specialized INTERNIST-1 vocabulary of 4,000 manifestation names. It takes an average of one hour to enter and run a new patient case on the system. Although it is probably the most complete general internal medicine knowledge database organized into computer format, it remains incomplete. It currently is on a mainframe computer. Some of the developers of INTERNIST-1 are working on a successor to the program called CADUCEUS, which is not yet available.

QMR, as a program with many medical expert consultant features, was described above, since it is the next stage of development of QUICK.

Weed has developed Problem Knowledge Coupler (PKC) Software to couple findings entered into a computer program with a medical knowledge database.[25] The program requests specific subjective information (medical history and symptoms) for a given problem. The subjective information can be obtained and entered by a nonphysician. Specific examination findings and diagnostic test results are also entered at the request of the program. It is then possible to couple the findings with the medical knowledge database and obtain a list of possible causes that is ranked according to how many of the findings are explained by the cause. Additionally, for a particular cause, the user can obtain a list of those findings pertinent to the cause that are present and those that are not. Management options are suggested, and the patient can be given a personal copy of the record.

Weed's medical expert system (Knowledge Couplers) has several other features.[25] Analysis of the patient population can be performed. Structure and guidance are provided for pursuing each patient problem. Editor software is available to edit and add to the Knowledge Coupler software. Editor software is necessary since a limited number of knowledge couplers for patient problems are available. Weed's system has not had any clinical trials of performance in comparison with physician performance.[25] The medical knowledge database is also limited. However, other problem knowledge couplers are rapidly being developed. Knowledge Coupler software runs on a variety of microcomputers.

Many expert systems have been developed for specialized diagnosis and treatment areas. ABEL for acid base and electrolytes,[30] MYCIN for diagnosis and treatment of serious infectious diseases (bacteremia and meningitis),[31] ONCOCIN for cancer chemotherapy,[27] and AI/RHEUM for rheumatology problems[32] are just a few. ONCOCIN is particularly notable since it has the potential to increase the capability of general internists to provide chemotherapy to cancer patients and provides a computer-based medical record. A blinded evaluation by four lymphoma experts found ONCOCIN treatment selections no different from those of physicians.[27] The developers' intent is for use of ONCOCIN by oncologists. Though ONCOCIN is in operation, most of these programs are not yet available for general use.

Computer-based medical expert consultant systems that enhance the capabilities of the general internist appear to have the greatest potential. The value of such programs increases with the number of clinical situations for which the program can be used.

PROTOCOL MANAGEMENT

A number of computer-based protocol management systems are available. Many have been adapted from algorithms already in use on paper. More recently developed computer-based protocols are part of medical care patient information systems such as HELP at the University of Utah,[33] Regenstrief CARE,[4] COSTAR,[3] and PROMIS.[5] Such protocols are particularly useful when employed by nonphysician clinicians such as nurse practitioners and physicians' assistants.[33,34] Most of the algorithms provide branching logic approaches to specific patient problems or symptoms. Interactive protocols for common primary care problems such as upper respiratory infection, urinary tract infection, and chronic management of high blood pressure or diabetes are most frequent. The computer program poses questions such as "Does the patient have a sore throat?" "Are the tonsils inflamed?" The logic then generates a diagnosis, treatment recommendations, reminders with regard to drug allergies, etc. The capabilities of computer-based protocols are determined by the number of patient problems and diagnoses for which straightforward diagnostic and treatment approaches have been defined.

CONCLUSIONS

A selection of different types of computer aids to clinical medicine has been described. The programs selected are the more developed ones described in the literature and have the potential of enhancing the internist's capabilities. A potential user should take into account a number of general and user-specific considerations and key questions when selecting a program (Table II). Not all of these considerations will apply to every category of computer aid in clinical medicine.

Ease of Use. The ease of use of the computer assistance system is extremely important. The patient care data, medical knowledge database, and artificial intelligence functions should be readily accessible. Systems that require a working knowledge of computers and knowledge of a large software program vocabulary are less likely to be used. Ideally, the system should require a short training period.

Accuracy and Reliability. Accuracy and reliability are particularly important in electronic textbook and medical expert systems. The medical knowledge database in the program should be accurate and as complete as possible. A formal mechanism for periodic review and update of a medical knowledge database is critical. Any artificial intelligence functions should interact with the medical knowledge database to produce diagnoses or treatment options that are accurate by some external expert criteria. Intra- and inter-user reliability should be assessed.

TABLE II. Considerations and Key Questions in Assessing and Selecting a Computer Aid to Clinical Medicine

General Considerations and Key Questions

Ease of Use
- Is the user-program interface easy to learn (ie, user friendly)?
- How long does it take to train a clinician to use it?
- What is the processing time for each command?
- What is the average time to enter and run a case or ask a clinical question?

Accuracy and Reliability
- Is the medical knowledge database complete?
- Which areas of medicine (diseases) are missing?
- Is the program frequently updated? How?
- Has the accuracy been tested? How?
- Are references provided to help evaluate the knowledge base?
- Has intra- and inter-user reliability been tested? How?

Acceptability
- Will clinicians use it?
- Has it been tested external to the original development site?
- Has there been publication of its performance?
- Who developed the program?

Cost
- What type of hardware is required?
- What is the cost of the software?
- What is the cost of data entry and routine upkeep?
- What are training costs?
- What type of support staff is required?

Effectiveness
- Does the program improve quality of care?
- Does it change health care resource utilization? (hospital length of stay, test ordering, etc)
- Do physicians perform better with it than without it on specific test cases?
- Does the program provide a form of continuing education?

User-Specific Considerations and Key Questions

System Domain
- Is there a medical knowledge database?
- Is the database general or specialized?
- Is it a diagnostic or therapeutic program or both?
- Does it focus on outpatient or inpatient care?
- What are the age groups addressed? Adult? Pediatric? Geriatric?

Capabilities
- Does the system replace the traditional medical record?
- Can you print out patient data, differential diagnoses, or other parts of the interaction?
- Does the system generate reminders or guides for preventive care as well as patient care management?
- Are administrative functions such as scheduling or billing carried out?
- Does the system interface with the lab or pharmacy?
- Can a clinical database be developed for population analysis research?
- Can the system be used for quality assurance?
- Can the system be used to monitor utilization?
- Can the patient enter data?
- Can the program access Medline?

Acceptability. Acceptability is affected by factors that include ease of use, accuracy, reliability, and credibility of the program developer(s). Evaluations of acceptability should be carried out at an appropriate user site that is outside the medical environs in which the system was developed. Previous studies of acceptability (adoption and utilization) have produced inconsistent findings.[35,36]

Cost. It is important to know cost allocation for planning purposes. Hence, costs of hardware, software, training of users, and maintenance of the program are the minimum considerations.

Cost data with regard to implementation and maintenance of these computer systems are difficult to define accurately. Several investigators have been willing to provide verbal estimates for hardware, software, training, and maintenance. It seems clear that the larger systems, those requiring mainframe computing capabilities and labor-intense data entry and retrieval, are the more expensive systems. Huth[37] stated that the value of a computerized information system should be calculated via a utility-cost analysis. However, the utility (further defined as relevance, thoroughness, and efficiency) has not been adequately studied for most of the systems. Cost data are incomplete and quite variable depending on the site and its resources.

Effectiveness. Potential benefits should be weighed against the costs. Does the program improve or maintain a high quality of health care? Do physician practice patterns improve? Are costs reduced by decreasing unnecessary test-ordering, appropriate use of human consultants, or substituting nonphysician clinicians for physicians? Is the program a form of on-site continuing medical education?

User-Specific Considerations. The needs of the user must be matched with the capabilities of the computer aid. Most difficult is identifying the specific objectives or category of information problems the user expects to meet or solve with computer assistance. The objectives are often realistically limited by time, facilities, and financial resources.

Pilot testing a computer program can be very helpful. Acceptability can be rapidly assessed in this manner. Testing a microcomputer-based, medical knowledge database or medical expert is relatively easy. Testing a computer-based medical records system or a time-oriented clinical database usually requires a visit to the developer's site, since the clinical database is not easily portable. Items listed in Table III are offered as possible evaluation measures during pilot testing.

Cautions. The state of the art for computer-assisted medical diagnosis and treatment is such that a number of problems remain. Among these are the need for greater user acceptability and accessibility, improvement in the medical knowledge databases, better explanation by the programs of how the conclusions are reached, and broader applications.[29] Currently, software programs are being developed and marketed for medical diagnostic use without validation. There is no validation procedure or official approval mechanism for software devices for use in patient care—there is no FDA jurisdiction to approve such software. There may be medicolegal considerations.[38] Clearly, a physician is culpable for malpractice based on computer advice. However, one could ask if the physician is liable for failing to take correct computer advice. The overall medicolegal realities and other results of computer assistance with medical information needs are not yet fully known.

TABLE III. Evaluative Measures for Pilot Testing Computer Aids to Clinical Medicine

- Acceptance
  - Actual use
  - User satisfaction and assessment of the system
- Quality of care
  - Review of cases managed with computer assistance
  - Comparison with those without computer assistance
- Effectiveness
  - Changes in hospital admissions, length of stay
  - Changes in test-ordering rates
  - Changes in use of human consultants

Most of the computer-based systems decribed here are still under development, including those that are available for use. All of them can function as a computer aid to physicians. Ideally, an excellent computer-based medical information and diagnostic system should always be developing and evolving with input of new medical knowledge and input from the users of the system.

In summary, computers can provide for many of the information needs of practicing physicians. Many software programs are being developed to aid physicians in general and specific fields. Most programs still have one or more of the following problems: inadequate testing external to the development site; the need for support personnel capable of programming; and inadequate resources needed to complete and maintain high quality, current medical knowledge databases. Nevertheless, medical computing is here, and physicians can benefit from its capabilities.

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The Island of Tindholmur, Faroe Islands (Pascal James Imperato, MD). Taken with a Minolta Maxxum 7000.

“The return of the oppressed”: Reflections on repeating the house staff experience after 20 years

NAOMI R. BLUESTONE, MD, MPH

> A cheerful interest in the activities of the day, strong motivation, and purpose in life will overcome fatigue and enable an individual to work efficiently with small amounts of sleep.
>
> *Harrison's Principles of Internal Medicine*, 3d edition (New York, McGraw-Hill Book Co, 1958)
>
> (*credo and talisman carried in the notebook of the author during long nights and difficult years*)

Although not many physicians are sufficiently motivated to repeat all of medical school as Ludwig Eichna did a decade ago,[1] many practitioners have lived through two specialty training periods separated by a number of years. Although second residencies are usually undertaken in response to changing professional interests, they offer an opportunity to study responses to the experience from the crow's nest of greater age and maturity. The recurrent focus of this essay will be one of the most crucial challenges in both first residency and second: physical endurance and its relationship to performance and morale.

House staff overwork, fatigue, and exhaustion is a topic that cannot fail to arouse mental frothing in all who have undergone the experience, whether once or, as in my own case, twice. It has been the subject of countless unresolved debates: Does it build character? Is it necessary to produce a disciplined physician? Contrary to the expressed belief of Dr Solomon[2] that “An important reason for the propagation of our unconscionable residency system is that physicians trained a generation ago did not experience the problems of overwork and chronic exhaustion that we now experience daily,” physicians since the beginnings of time have complained that the doctor's lot, like that of the policeman, is not a happy one.

Dr Lempert correctly points out Maimonides' description of his battle with the need to sleep.[3] The memoirs of my grandfather,[4] to which I have alluded previously,[5] spell out quite clearly the miasma of weariness that dogged his footsteps, both before and after his graduation from the New York University School of Medicine in 1890. Dr Arthur E. Hertzler, a self-confessed “kitchen surgeon,” describes the same phenomenon in his 1938 autobiography, *The Horse and Buggy Doctor*.[6] Antique and near-contemporary reports such as these speak to us with an immediacy that evokes our most sympathetic sleep hunger.

It is truly remarkable, in fact, how little the system has changed, given the universality of the phenomenon of exhaustion, and the decreasing tolerance of young physicians to being exploited. It is clearly a measure of our shame and dereliction that effective impetus for change had to come, not from the standard bearers of the Olympian Aesculapius, but from Zion.[7]

Like many other physicians of my generation, I have always thought of my internship year as the best year of my life. I wondered as I returned to the indentured life of a house officer if I would change my mind about that. After all, the physical hardships as well as the satisfactions were extreme. How well I remembered the day I fell asleep in broad daylight when my car broke down by the median of a midtown expressway after an on-call weekend. And the time I tried in vain to wash my hands, only to realize at some point that I was massaging a tube of toothpaste instead of a bar of soap! And the 60 hours of nonstop deliveries when I fell asleep writing up the last, and cut my forehead on the edge of the nursing cabinet. How could anyone so chronically exhausted, terrified, and agonized still be able to rise above the ordeal to proclaim the validity of the experience? Rich memories are not built exclusively on foundations of repression and denial. If indeed there is more to the life of a house officer than walking masochism, we would be well served to uncover and preserve whatever it might be.

Since completing the second residency, I have been contacted by other older physicians contemplating such a move. They have asked if it was difficult for me to go from associate professor at a major medical school to scutwork in the suburbs. (The answer was no, although at times it was amusing how much clout I had taken for granted until it was no longer in my possession. Another returnee well known at the hospital mourned that the hardest part was having to give up her key to the doctors' parking lot!) Others wanted to know how difficult the night calls were in middle age. (Actually, my psychic defenses, political savvy, understanding of the power structure, and ability to negotiate a work schedule were better developed than when I was young.) There were concerns about the loss of

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income over a three-year period, but few seemed aware of the vast experiential differences between trainees of yesterday and today. These actually provided the most stressful stimuli of all for me.

First, let us acknowledge the difficulties of attempting an exercise based on comparing the "there and then" with the "here and now." No two residencies are comparable for the same reason that no two siblings have the same mother. Furthermore, the several apprenticeships that served as the field sites for my reflections were as different as the person who undertook them. I have tried to be objective in my subjectivity, and to generalize only where I felt myself reacting as one of many others. My conclusions can never be proven.

In 1961, youthful and idealistic, I served for half a year as a junior intern on the wards of my medical school hospital, working every other night under the supervision of residents (as there were no interns). It was a particularly difficult time as I was barely recovered from a severe bout with "mono" the preceding year. The following year I served a rotating internship in a busy, academically affiliated general hospital in the same city (Philadelphia). This internship was selected because it offered an every-third-night rotation, a provision of paramount importance to me after the agony of "every other night." I then went on to a master's degree, a residency, and a career in public health.

In 1982, my ears well cauliflowered in the medical care arena, I began three years of residency in psychiatry, which is now very much in the mainstream of biological medicine. This was served at a state psychiatric center and a local university-affiliated hospital, both chosen primarily for their proximity to my suburban home. (The instinct to ensure sleep was immutable after years of susceptibility to extreme fatigue.) The return to full-time hospital work with patients for the first time in several decades fulfilled my curiosity to see how much this world would still be recognizable and familiar to me. Unlike other physicians returning to residency who remained in the mainstream of patient care and accommodated to change gradually, I was truly returning "de novo."

The baptism of exhaustion came almost immediately. It seemed to me that I was doing many more on-calls than had been implied in application interviews. A female resident, curse her and her unborn child, developed female troubles early in pregnancy and elected to drop out and spend her year supine. We in her class were expected to pick up her night calls, as no provision had been made for this contingency. I had the grace to feel ashamed as several decades of liberal, humanistic personal development came crashing down around me, along with whatever feminist dogma I thought I believed in. All I knew, as I dragged my weary carcass along, was that this woman may have been very dedicated to her procreation, but I certainly was not. Call it one more unwanted pregnancy. The resentment, anger, and sense of helplessness against oppression came back in full force. Yes, it was very familiar. The House Officer as Victim. Whither Social Justice?

I handled the issue far differently the second time around. When I became ill and could not appear for work, I was told, as in the old days, that the night's coverage was my responsibility and that if I could not work, I would have to find someone to take my place. The message reminded me of the old "If you don't work Sunday, don't come in Monday" era in our nation's history. I knew that it would be impossible for me to "pay back" nights while still not well, and that this solution was stop-gap and ultimately foolish. I also did not wish my already overburdened classmates to vent their rage upon me for doing to them what the woman with the nicely developing belly had done to us already. Protected by a realization that never would have occurred to me the first time around, that the hospital needed me as much as I needed them (for without me wouldn't they be in a pretty pickle?), I made it clear that 36-hour calls so frequently spaced would be a disaster for both parties. It was arranged that my on-calls would henceforward be confined to weekends. I would be forced to work no more than 24 hours at a stretch, and would not have to work as often (extra points being racked up for weekend duty). I was more than willing to work for a year without a full weekend off to uphold my fair share of coverage and consider myself lucky. I detail it because it was an episode that could have happened to any resident in this country.

Until the agreement was effected, I went through a period of feeling that no attending physician could possibly remember the horror of being exhausted and helpless, a prisoner in a Chinese cookie hospital. I had felt that way many years before and repressed it nicely. I vowed that when this experience was over, I would not forget again. But even as I made the promise, I knew it could never be kept. It was like demanding remembrance of an episode of starvation, incarceration, or great physical pain. The veil of repression and denial would drop over me again as soon as I would return to my normal, privileged existence. This essay may be considered in part an attempt to keep that promise to myself. Nurse, may I have the sodium amytal, please?

I believe my anger welled up because by now I could see so clearly that the issue was not service to patients, dedication to medicine, or fulfillment of educational imperatives. It was a whole rogue's gallery of unfair labor practices. It was exploitation of the low man on the totem pole, bureaucratic buck-passing, rigid policy adherence, inflexibility of responsiveness to individual human need, favoritism and cronyism, and a lot of other dirty dealings having little to do with medicine. The residency, whose managers considered themselves to be reasonably enlightened educators, was being run in a manner that could never have passed the scrutiny of a human subjects committee. (I know; I used to serve on one.) Even worse, it was hard to blame them. They were no longer running the show.

Forced to comply with the dictates of procedural manuals and the coercive, pseudo-collegial phone calls pressing both collaterally and from above, they had become helpless beings, scurrying about serving many masters, and still trying to teach in between. The complicated network of interfering agents, whose presence was less noxious and with whom we barely concerned ourselves the first time around, was now painfully familiar even to the lowliest resident.

Even our complaints were no longer met with age-old and time-honored rejoinders such as: "This is vital to your education." "You had better get used to it; this is the life of a doctor." "This is the only way to really understand a disease, to see it played out night and day." We were now frankly told, "The administration wants it that way." "This is a requirement of the (fill in the blank)." "If you don't do it, we will lose our (fill in the blank)." Or, worse, "Yes, you are right, and yes, it makes no sense, and yes, we have tried to change it, and no, there's nothing we can do about it, so DO IT and don't give us a hard time" (note of irritation creeping into the voice). And so we went to work feeling as if we were fulfilling the agenda of some foreign agency, which had little to do with our own needs, or those of the patients.

Sometimes it is necessary to strip down ruthlessly a complex corpus of problems to its bony matrix. When I do so here, the same skeleton emerges. The young people of today have been deprived of the heart of medicine: the great spirit which characterized the training periods predating the explosive technological and fiscal revelations of recent years. Those who say, either in judgment or support, "We did it and you can do it too," must recognize that the "it" is not the same. It is difficult for me to acknowledge this without feeling a great sadness, and an overwhelming sense of inadequacy about sharing my feelings of loss with strangers.

Some of my colleagues have criticized today's young people as being narcissistic, overly concerned with good pay and short hours, unwilling to sacrifice personal lives for medicine, no longer idealistic, undisciplined, and lacking in dedication to the good of the patient above all else. Without commenting for the moment on that assumption, I would point out that they share a simultaneous reluctance to recognize that these young people are no dopes. They have eyes and they can hear. They can read between the lines and they know when they are being conned. They are more practical, more accustomed to disillusionment. They were raised in a different generation, and have confronted more in their young lives than many of us have in middle age.

There is absolutely no doubt that young physicians of today work inhuman hours, as have all physicians still alive and practicing. Who with a beating heart could dispute that reform is in order? But having established that for the record, I think we are obligated to note also that the fatigue of today's residents is intensified by depression and directly related to their feelings of being pawns in an oppressive system that offers only conditional rewards. Belief in the good will of colleagues and the value of the task is vital to sweetening fatigue. An overworked resident whose morale is high and whose natural stream of cheerfulness can come bubbling up will tolerate exhaustion far better than one who is expending emotional energy on resentment, anger, and resignation to what is experienced as an intolerable situation.

Perhaps useful insights will emerge if I try now to account for the reasons why it seemed easier for us to maintain our morale in the "old days." Clearly the training experience then was not just an exercise in medical masochism.

First of all, we thought we owned the hospital, and in a real sense, we did. Right or wrong, the elitist attitudes and perks which are now stripped away softened our difficult lives. Doctors in those days were truly respected; we did not have to endure the ignominy of being pushed around by entitled patients who considered us their somatic lackeys. True, we had our share of pampered ladies and irascible alcoholics, but it was all part of the game. Our worst insult was "Are you an intern or a doctor?", an ignorant remark heard less frequently today. If patients were losing their awe of doctors in general, at least they considered their own doctor above reproach.

Hospitals were cozier, too. They were older, less functional, more aesthetic, and dirtier, with all the warmth and discomfort of a claw-footed Victorian tub. The attendings down at Fifth and Reed truly loved that old medical dump and retired there as if to their private club. So did we. It may seem bizarre to today's hard-pressed residents, but we used to hang out on our nights off, helping teammates, popping in on old Mrs So-and-so, and using the hospital as our community center. (I remember, for example, spending some nights off helping the orthopedic resident hack up a diabetic leg down in the morgue. He needed the anatomic experience and I needed the fun.)

There are also an incredible number of service personnel underfoot nowadays, too. They splinter and fragment much of the patient care package that used to be a cohesive whole for the house officer. Today's hospital belongs to everyone but the house staff, an inhibiting and chilling reality which does not encourage "hanging out."

Perhaps because we felt as if we owned the joint, we were more free to laugh. I anticipate being criticized for this, but it seems to me that today's house staff don't know how to play a decent practical joke, or escalate it to the heights of lunatic perfection. I have recounted in a past series of articles some of the fun we used to have as medicine's irrepressible little darlings, and will not repeat myself here. But now I recall a vignette I have not thought of in years: One of the residents was about to rotate to a different division of the hospital, and I realized that we would miss him. We had grown accustomed to his lanky, laid-back form sprawled across the old green sofa in the resident's lounge, a cigarette eternally dangling from his lips. I recall staying up most of the night fashioning a miniature couch for him to take along to his new assignment. It was made of tongue depressors, two by fours, green ink filched from the hospital chief's office, cotton padding from the ER, and Elmer's glue-all. I even burned a cigarette hole in it to correspond to that of the original. It was a masterpiece of pop art. When I saw him at a meeting years later, he told me he still had it. Somehow, it is hard to think of today's harrassed house staff routinely wasting their time on such tomfoolery. I don't even think they have fun with the nurses anymore . . . maybe because there aren't too many left. (Another essay some day.)

In those days we were required to rotate through a number of services before specializing in one. Accordingly, we had a chance to see urology in action (a fate worse than death!), deliver babies, sew cuts on pre-adolescents, and try all of medicine's subdivisions. We could enjoy everything knowing that it would soon be over if it was not really for us. Today we see too many people who felt forced to choose prematurely, usually as early as the beginning of their senior year. Many lived to regret it before the training was half over. I think the pressure of "Is this what I want to do for the rest of my life?" is a depressing albatross to carry on rounds.

Although our paperwork was no worse then than it is now, at least in those days everyone had to do it the hard way. No one had computers, modern dictating equipment, or the other paraphernalia that would have minimized the time required for record keeping. Now it seems that hospitals prefer paying interns to write longhand. Clerical staff are more expensive to feed. It is ludicrous for such highly trained people to waste their time at the nurses' station scrawling their illegible hieroglyphics, but they are still compelled to do so. They'd be better off napping. The irony is that no one reads most of it anyway! I cite this as but one example of the scutwork that is dumped on house staff today with much less objective reason than in the past. Samuel Johnson defined the lexicographer as "a harmless drudge," but that's because he didn't know any house staff of 1988.

One area that contributed tremendously to a now-lost sense of autonomy was our freedom from the restraints imposed upon us today by the medical ethicists. I say this with consternation, as much of my professional life has been spent trying to make people aware of the breaches in ethical values that have always permeated our medical care system. Recognizing that this state of affairs was unduly paternalistic and did patients an injustice, I nevertheless feel that it helped us to bear our burden of weariness. It is not pleasant to feel your wrists bound in the performance of duty, a phenomenon experienced by many professionals in today's society. We were spared that. I shudder to think of how we manipulated the grieving for autopsy releases, disparaged the concept of "informed consent" (even though psychiatrists know how much of a myth this actually is), and told fibs about prognoses that should have made our noses grow ten feet. We had no need to rationalize our behavior as today's residents do, for we did not believe it to be wrong. And in consideration of the excesses of the ethical and civil rights zealots, I'm *still* not sure some of it was so wrong. By bearing the burden of responsibility we now fob off on patients, we remained professionals rather than brokers or agents.

I would like to say something at this point about sexism, then and now. My view may appear paradoxical, and it pertains only to the issue of being a woman in medicine. I must admit, if I am to be as honest as I can, that in many ways it was easier to be a woman in medicine 20 years ago. I know that flies in the face of our well-documented history of prejudice, harrassment, closed opportunities, unfair expectations, and all the other abominations with which women have had to contend. In what regard was it easier? First of all, many of us did not appreciate how badly we were being treated, particularly those of us who had been fortunate enough to attend the (then) Woman's Medical College of Pennsylvania. I was too naive to know what was going on and thought some people just didn't like *me* (a common misconception by women in those days). Only retrospectively did I realize that many of the problems of my internship were due to the handful of men who would have preferred that I remove myself to the great female powder room in the sky. On the up side, it was rather fun to be one of the few female doctors in the bunch.

It is fashionable now to refer disdainfully to women who enjoy being unusual and who are comfortable in the company of men as "Queen Bees" who put down female competition. This is unfair. We were pioneers, and it was truly exciting. We did not present ourselves in such numbers as to be considered a serious threat, nor had legislation caught up with the rampant chauvinists, and forced them to go into hiding with their prejudices. Today's young women sometimes bring to their residencies an understandable anger and confusion about their situation, as well as feminist expectations that they fulfill the dream and "have it all." What a burden to bear! Sometimes I think we were better off ignorant and hopeful, willing to work and enjoy the rewards without bitterness about what we did not have. At least we didn't have to deal with men frustrated by years of having to share their sandbox. If that's a feminist, 20th century Uncle Tomism, so be it.

The issue of "having it all" also feeds the discontent of today's male house staff. In the old days, it was just expected that people would try to postpone marriage, home, family, and normal human pursuits until the long training period was over. We were not pressured to "have it all," which translates into loans that could bankroll a developing nation, two young children who think Daddy must be divorced like everyone else's daddy since he never comes home, and alimony based on the spouse's contribution to the joint effort of "having it all." It may have been stark and monodimensional, but it had its saving graces. There is no way I could have been married during my internship and still emerge considering it "the best year of my life."

I believe further that the nature of the enemy was less pernicious in those days. Our scourges were patients and a few nasty nurses and attendings. Our skirmishes with administration, third-party payers, and legislators were buffered by a layer of medical superiors who tried to keep us vestal pure for medicine. This could not continue, of course, given our fiscal innocence and total lack of any business acumen. But the end result has been a move from revolt against administrative fiats concerning medical practice to revolt against commercial intrusion into our professionalism, and, as mentioned earlier, it grows harder and harder for academic physicians to shield students from this kind of assault. They are sensitive to the antagonistic jockeying, the adversarial positioning that is always playing itself out. Academic warlords in particular often threaten to break loose from the fragile tenets of civilized restraint. The rippling circle of these pressures washes at the shore where today's house staff are trying to scrub their whites.

Now let me move on to another area where our exhaustion was made more bearable than it is today. Knowingly I indict my own generation by suggesting that there are fewer doctors around who will give of themselves and their time off to impart to house staff a sense of community and entry into a profession.

We had attendings and teachers we will always remember. Yes, there were sadists, and I have written about them, too. But prominent in memory are deeds of concern, kindness, and cooperative participation that were not matched the second time around. There were attendings who did not "delegate" but sat up at night with me and the patient, tending to the two of us as if we were a sacred triad: the patient, the student, the teacher, at one in a common purpose. A goodly number of obstetricians at my college hospital routinely stayed at the bedsides of women through their entire labor. Many hours were spent in the middle of the night, drinking coffee, feet up on the table, discussing cases, exchanging diagnostic and therapeutic tricks, developing our own verbal apocrypha. If we were thrown on our own, it was to help us learn or because there was no choice, not because someone was too lazy or too busy making a buck to join us. Idealizing through the retrospectoscope? I don't think so. There was more personal concern for us and our place in the profession 20 years ago. This collegial warmth seemed withered and attenuated the second time around, even granted that there was no exposure to the surgical specialties where one would expect this to be more common.

As I remember that old internship in Philadelphia, names come back to me that I have not heard in years. Lillian Balter. Jose Auday. Maurice Fishbach. Someone named Silk. They were real presences in our lives. And always at night, when we were all utterly exhausted, and, I realize now, they were no longer young. I am only three and a half years fresh from the second residency, and my memories of night call are all solitary ones. Alone in the ER. Alone writing up cases on the wards. Occasionally having a passing chat with an attending in the hospital in the ungodly hours. But I honestly cannot remember ever sitting down with anybody in the middle of the night to discuss a case thoroughly. No one was there.

I admit, to my sorrow, that few if any of the residents now on call can sing *my* praises as a nocturnal guide to the mysteries of psychiatry. I wasn't there for them, either. Maybe it was because they were all too intimidated to say, "Hey, come in, I could use you." All I can say in my own defense is that I believe I would have come had I been asked, and had I thought about what I am writing today. If my experience is anything but an aberrance, there is something amiss in our apprenticeship system today. We are too busy for our students; we neglect them as badly as we neglect our children.

The new technology has been blamed for making the intern even more exhausted than we were. I am not sure we should buy this argument wholesale. It is true that there has been an explosion of equipment to accumulate great bodies of microdata about patients by invading even those parts of the body which used to be good for nothing but superficial fascia. It is also true that the house officer is the foot soldier expected to pick up this equipment, lug it on his back, check its nuts and bolts, and take the blame when it lies down in a corner belly up. But the exhaustion of the intern hauling the heavy artillery is due not to its real time demands, but to its intrusiveness on the intimacy with the patient.

I think it is much easier to order a PET, CAT, or DOG scan on a patient than to assist with a pneumoencephalogram, or any of the other hideously invasive maneuvers we attempted with more primitive instruments. But we did not have to contend with those large, gray monsters that tick and belch out their mechanized intelligences while the patients become psychotic from the environment and rip out all their lines. (No, we didn't have haloperidol in those days either, nor did we need so much of it. If some benevolent foundation would provide every ICU bed in the United States with a mandatory wall clock, calendar, and in-flight movies, we might not need the stuff at all.)

As I made my consultation/liaison rounds, it seemed to me that the residents on medicine were overwhelmed trying to get through to patients cowering behind all the ticks and bleeps. The amount of distancing they required was extraordinary, and none of it did any good. How exhausting to have to pretend a relationship, to substitute stock phrases for real inquiry, to fight the no-win temptation to stick around and relate. We were less prone to the sham that passes for the house staff/patient relationship today. As bad as it was, it was easier to get close to the bedside, deal with the anxious or depressive feelings we were experiencing, and get out without too much guilt.

Speaking of "getting out," sometimes patients need to do that too, and I'm not speaking of discharge or transfer. We were not put in the position of having to decide if we should permit a patient to die, because the choice was frequently taken out of our hands. We did not have the technology for putting patients on machine-assisted storage for such long periods of time. Therefore, we didn't have accumulations of bodies placed on suffering hold until we could come to terms with our mandates as physicians. I will never forget the day my roommate, a Philippino woman physician, was summoned to the office of the administrator and summarily dismissed. Given only 24 hours to remove her silks from our closet, she expressed genuine bafflement. "They called me to come see a patient last night but I refused. What was the point? He was an old man and he was going to die anyway!" Such an attitude was as inconceivable then as it is today, but it was more representative of our reality. Today's residents have been forced to give up the classical approach to death . . . that it is a mortal enemy we continually fight on behalf of the patient, but when we are licked, we do what we can to ease suffering. They are subjected to an obsessive-compulsive, stereotyped, and repetitive salvation ethic that reflects inability to let go. Like the nurses, who also must live in this sci-fi horror story night in and night out, they develop almost a Sonderkommando mentality; after a while, it reduces to "It's them or us." Doing this to people who went into medicine with at least some altruistic motive is cruel and inhuman punishment. Losing a patient one has loved after doing one's best is infinitely less stressful than having to keep facing that same patient when he mutely begs to die. (It is often mutely because there is often a trach, and the inability to talk usually intensifies the final process.) No wonder my young colleagues are exhausted! Sacking out is not just a physical repair but an emotional anodyne. Frankly, we are lucky that more of our house staff are not into heavier manipulation of their neurochemicals than they are right now.

The bottom line of all this is that the problems of the educational system, always existent and resistant to change, are now so heavy that the entire structure is corroded and heaving, like a neglected bridge ready to give way at last in the height of rush hour. The most recent attempt to shore it up comes from the State of New York, which is presently engaged in a serious effort to regulate the working hours of this strenuous ordeal we call graduate medical training.

If I have not already made my point clearly, I will say it one last time. We would be mistaking the footprint for the shoe to consider resident fatigue as the real issue behind the current brouhaha. I believe it to be a smokescreen for something even more malignant. Fatigue is bearable, endurable, and ego-syntonic if it comes within a context of feeling good about oneself and dedication to one's work. It is glucose; it is fuel for students, revolutionaries, and committed artists; it is a "high" when one can feel that "it was all worth it." When tiredness is a result of unfair impositions by banal but impenetrable forces unconcerned with the well-being of the house staff, it is a moral wrong which imperils the society of medicine and the health of the country. It will not be cured by sleep.

Yes, fatigue and exhaustion are directly related to lack of shut-eye and long hours without adequate breaks. But they are also related to demoralization, frustration, rage, depression, and powerlessness. They are related to feelings best expressed by Dostoyevski, whose gloomy literature proclaimed that the worst thing one could do to a human being was to render his life work meaningless. The feeling that one is a cog in a huge machine, that one must do one's time, that one must not make waves, destroys the spirit as surely as exhaustion saps the body. This may explain why literature attempting to evaluate decremental functioning secondary to sleeplessness is so equivocal.

Today's house officers may earn a living wage, participate nominally in committees serving their interests, unionize and organize. Soon they may even be working to an alarm clock. But I truly wonder if they will remember their internships as "the best year of their lives."

We all appreciate the impetus behind the state's attempt to address the issue of house staff overwork. We reserve judgment as the recommendations of Dr Bell and his committee[8] filter their way through the legislative process. We are aware that the Accreditation Council for Graduate Medical Education has initiated a task force to study resident hours. We are grateful that the beast has left its cage and the trainers encircle it with hearts intent on intelligent mastery.

May we ask those whose recommendations are being sought to consider not only the rigidly temporal issues that appear to be their mandate, but the spatial ones that define the young physician's professional environment as well? I call to mind an ancient Hebrew expression to which my parents often referred, "tikkun olam," which means "mending or repairing the world." Anything we can do to repair the world of our young generation will revitalize us, strengthen them, and benefit the patients to whom we are all ultimately responsible.

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Obstruction of the common hepatic duct by ectopic pancreas

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The majority of tumors that cause complete obstruction of the common hepatic duct are malignant and generally are associated with a poor prognosis. Complete obstruction of the common hepatic duct by a benign tumor is rarely encountered but is readily amenable to surgical correction. Though extremely rare, heterotopic pancreatic tissue has been reported as a cause of distal biliary tract obstruction.[1-5] The case reported here, of a patient whose history and preoperative diagnostic studies suggested a malignant tumor, represents an even rarer type of bile duct obstruction by heterotopic pancreas.

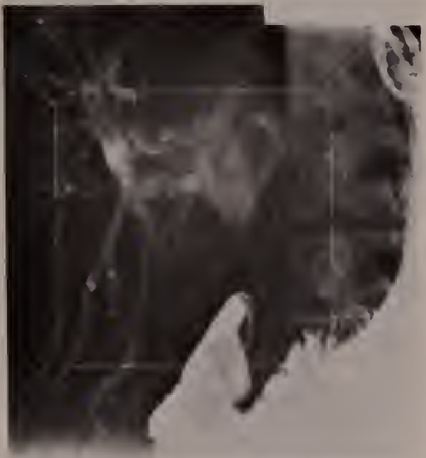

FIGURE 1. Transhepatic percutaneous cholangiogram showing complete obstruction of the proximal common hepatic duct (barium is present in the stomach).

CASE REPORT

A 43-year-old woman was admitted to the hospital with a history of progressive pruritus, painless jaundice, and a 12-pound weight loss. She was icteric; no abdominal mass was palpable; and her stool was clay colored. Pertinent laboratory studies showed progressive increases in total serum bilirubin to 15.4 mg/dL and alkaline phosphatase to 328 U/L (upper limit of normal, 115 U/L). The serum glutamic-oxaloacetic transaminase and serum glutamic-pyruvic transaminase were only minimally elevated. Upper gastrointestinal radiographs disclosed no abnormality. Percutaneous transhepatic cholangiography demonstrated complete obstruction of the common hepatic duct just distal to the confluence of the right and left hepatic ducts (Fig 1). These findings were consistent with a malignancy.

At operation the gallbladder was collapsed and the common bile duct was normal in size. The pancreas was normal and no mass was present in the porta hepatis. A 3-mm-long stricture of the common hepatic duct lay just distal to the confluence of the right and left hepatic ducts. The tissues surrounding the extrahepatic biliary tree were normal. The confluence of the right and left hepatic ducts was transected and the bile duct was resected in continuity with the gallbladder down to the retroduodenal portion of the common duct. Reconstruction of the biliary tree was accomplished using a Roux-en-Y limb of jejunum.

Gross examination of the operative specimen showed complete occlusion of the proximal common hepatic duct. Microscopic examination of the wall of this portion of the duct revealed replacement by dense fibrous tissue which harbored foci of ectopic pancreatic tissue associated with acute and chronic focal inflammation (Fig 2). Further sectioning of serial tissue blocks disclosed no carcinoma or additional sites of ectopic pancreas.

The patient continues to do well five years later, and her liver function studies are normal.

DISCUSSION

The term "pancreatic heterotopia" was adopted by Barbosa et al[1] to describe the presence of histologically identifiable pancreatic tissue in an abnormal location without parenchymal, stromal, or vascular connection with the main pancreas. Numerous reports describe a variety of locations of heterotopic pancreas including the stomach, duodenum, ileum, Meckel's diverticulum, mesentery, omentum, spleen, biliary tract, umbilical region, liver, colon, esophagus, lung, fallopian tube, and lymph node.[1,6-11] The ectopic tissue appears most commonly in a submucosal location[12] of the stomach (24-38%), duodenum (9-36%), or jejunum (0.5-27%). The incidence of this anomaly discovered during surgical exploration of the upper part of the abdominal cavity has been estimated to be as frequent

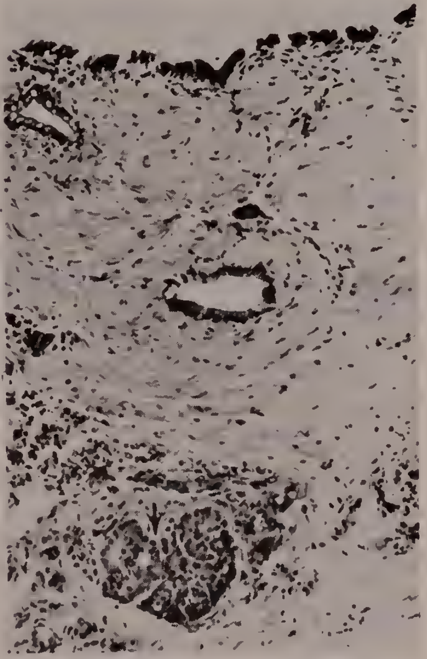

FIGURE 2. Common hepatic duct at the site of stricture showing a focus of ectopic pancreas (arrow) with surrounding acute and chronic inflammation, dense mural fibrosis, and thinning of epithelium. The gland in the center of the section is a residual epithelial crypt, which is a common finding (hematoxylin-eosin stain; original magnification × 320).

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as one in 500.[1] Somewhat higher figures have been cited in necropsy studies.

A number of theories have been proposed to explain the appearance of pancreatic tissue in ectopic sites. Most implicate faulty embryological development but none adequately account for the wide-ranging dispersion of heterotopic pancreas.

The embryonic pancreas is composed of two anlagen: a dorsal bud derived from the intestine, and a ventral bud which arises from the distal choledochal tract. The anlagen coalesce into one organ through the process of circumrotation. Horgan[13] postulated that branching buds of the primitive pancreas adhere to surrounding stomach or intestine and detach as grafts during separation and migration of the organs. The theory suggests, on the basis of proximity, that pancreatic tissue incorporated into the biliary tract originates in the ventral anlage.

Pancreatic tissue is rarely found in the biliary tree. We have only been able to find eight reported cases in the literature of heterotopic pancreas in the bile duct.[1-5] In these cases the ectopic tissue was located in the distal common bile duct or ampulla of Vater and caused partial obstruction. The clinical presentation most often mimics a malignant obstruction, as in the patient described here. This case, however, is unique in two respects. The ectopic pancreatic tissue was situated in the proximal common hepatic duct and was associated with complete obstruction. Carcinoma, a localized form of sclerosing cholangitis, inflammatory stricture, and a rare amine precursor uptake and decarboxylation cell tumor are among other conditions causing a stricture at the hilum in the absence of previous biliary surgery.[14]

Heterotopic pancreas may become clinically apparent at any age but is more frequent in the fifth and sixth decades of life. Symptoms depend on its size and location in conjunction with intrinsic changes or secondary changes in surrounding tissues. The aberrant tissue is subject to the range of pathology that affects the normal pancreas. The dense fibrosis leading to the complete biliary obstruction in our patient was most likely a result of the acute and chronic pancreatitis observed in the specimen. Malignant changes were not observed in the present case, but there are reports of adenocarcinoma arising in gastric heterotopic pancreas.[15-16] Islet cell tumors may also originate in heterotopic pancreatic tissue.[17-18]

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Chronic asymptomatic dissecting aneurysm of the aorta

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An enlarging mediastinal mass in an asymptomatic patient can be a diagnostic dilemma. Common mediastinal lesions include lymphoma, retrosternal thyroid, thymic tumors, teratoid tumors, and pericardial cyst in the anterior compartment. Lesions in the middle compartment include lymph node enlargement, esophageal lesions, aneurysm of the aortic arch, hiatal hernia, and bronchogenic cyst. Lesions in the posterior compartment include neurogenic tumors, aneurysm of descending aorta, lymph node enlargement, and paraspinal manifestations of spinal lesions.

The following case illustrates how the selective use of modern imaging modalities can rapidly resolve the dilemma with relatively noninvasive techniques. Lesions not clearly identified are often amenable to percutaneous computed tomography (CT)-guided thin needle biopsy.

Case Report

A 55-year-old man with known hypertension presented to the emergency room with epistaxis. Due to poorly controlled hypertension, with blood pressure measuring 200/110 mm Hg, he was admitted for further evaluation and treatment.

His medical history included heavy cigarette smoking, myocardial infarction, and

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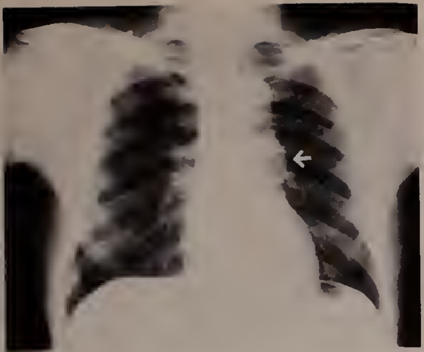

FIGURE 1. Admitting chest film showing slight fullness in left hilum (arrow).

pulmonary embolus. The physical examination was remarkable for a blood pressure of 200/110 mm Hg. The heart rate was 80/min; respiratory rate, 16/min; and the lungs were clear. Examination of the cardiovascular and neurologic systems was normal. A chest film taken on admission showed clear lung fields and slight fullness in the left hilum (Fig 1). A lateral view was recommended at the time, but no additional follow-up was obtained. The patient was discharged after one week and was normotensive at that time.

Three years later, the patient was admitted for an elective inguinal herniorrhaphy. He had been asymptomatic during this time interval, but it is not known how well his blood pressure was controlled. His admission chest films taken at this time showed a smooth, large, left-sided mediastinal mass (Fig 2). The patient underwent a contrast-enhanced CT scan of the chest, and the diagnosis of chronic asymptomatic dissecting aneurysm of the aorta was made (Fig 3).

DISCUSSION

Dissecting aneurysms of the aorta are generally catastrophic events with a high mortality in the first few hours or days. When the patient survives more than six weeks the aneurysm is considered to be chronic.[1] Another group of dissecting aneurysms that have received more attention recently are those that present little or no symptoms and seem to have a more favorable course. These are referred to as "chronic," "painless," "healed," or "unsuspected."[2] This may be a separate group from those aneurysms that originally present as acute dissections and become chronic. It is possible that these aneurysms begin as subclinical events with small bleeds within the aortic wall and progress with little or no symptoms until incidentally discovered. Many of these manifest themselves because of worsening hypertension, chronic congestive failure, or aortic regurgitation, or as an enlarging mass on a chest radiograph.[3]

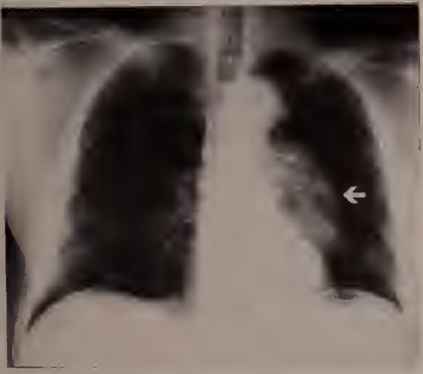

FIGURE 2. Chest film from second admission showing large, smooth, left-sided mediastinal mass (arrow).

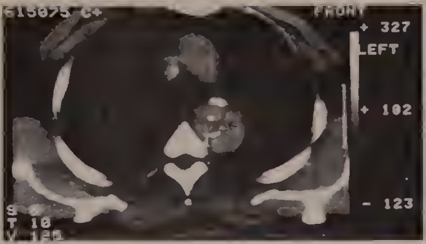

FIGURE 3. CT scan of descending thoracic aorta showing partially calcified intimal flap bisecting aorta into true and false lumens (arrow). Square cursor centered on true lumen for density reading.

The diagnosis in this case was provided by a contrast-enhanced CT scan of the chest. As shown in Figure 3, an enlarged descending thoracic aorta is clearly bisected by a partially calcified intimal flap defining a true and false lumen, both of which contain contrast. This is a type III aortic dissection according to the DeBakey classification, or a type B according to Shumway.[3]

Although arteriography has been the sine qua non for diagnosing aneurysms, the less invasive CT scan is now the initial procedure of choice.[4] According to Moncado et al,[4] the CT characteristics of dissection include the following: increased diameter of the ascending or descending aorta; medial displacement of intimal atherosclerotic plaque; increased or decreased density of partially congealed hematoma in false lumen on precontrast scans; demonstration of two channels; identification of the dissected flap; compression deformity of the true lumen; delayed flow through false channel; pleural or pericardial leakage. Several of these criteria are satisfied in the case presented here.

With the advent of magnetic resonance imaging (MRI), the ability to demonstrate vascular abnormalities without the injection of contrast material or the use of ionizing radiation has become a reality. Unlike CT, which measures only tissue density, an MRI signal is more tissue specific, measuring the physical characteristics of magnetized protons returning to their resting state. Glazer et al[5] and Dinsmore and colleagues[6] demonstrated the intimal flap in aortic dissections as a medium-signal structure separating the true and false lumina. Blood flowing at a normal rate of speed in a vessel escapes detection and is represented on an image as a signal void surrounded by signal-producing vessel wall. When blood flow is disturbed, such as when blood flows at a slower rate of speed in the false lumen of a dissecting aneurysm, it begins to emit a detectable signal which can be recorded as an image. In addition, Dinsmore et al[6] identified the actual intimal tear as a direct extension of the flow void from the true lumen into the false channel. Identification of this tear has not been possible with computed tomography.

Another advantage of MRI in the diagnosis of aortic dissection is the ability to image in transverse, coronal, and sagittal planes. This allows easier determination of the relationship of the dissection to arch vessels.[5]

Differentiating thrombosed false lumen in an aortic dissection from an aortic aneurysm with mural thrombus is difficult with MRI. One reason is that peripheral calcification in an aneurysm and displaced intimal calcification in an aortic dissection, which are helpful clues on CT examinations, cause little or no signal on MRI.[5] Other current disadvantages of MRI are that patient motion severely degrades the images, and critically ill patients with life-support systems cannot undergo MRI. Claustrophobia in the MRI gantry has also been reported.

Despite these limitations, MRI is a promising method for diagnosing aortic dissections, and it is hoped that vascular abnormalities may be better defined with little or no risk to the patient.

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Left pulmonary artery agenesis

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HARVEY EISENBERG, MD

Congenital abnormalities of the respiratory system are uncommon clinical and radiographic presentations in adulthood. This report describes a woman with pulmonary artery agenesis accompanied by pulmonary hypoplasia.

CASE REPORT

A 39-year-old woman sought treatment for progressive dyspnea on exertion and rest, chronic left-sided chest discomfort, and a history of episodes of cough productive of blood-tinged sputum. There was no hemoptysis on the current admission. She was born in Puerto Rico and had moved to the United States at age three. She had had multiple protracted respiratory illnesses during childhood and had been treated for presumed pulmonary tuberculosis. As a child and teenager, she experienced dyspnea on exertion, which remained stable until age 36, when progressive worsening of exercise tolerance developed.

Physical examination revealed normal vital signs with a respiratory rate of 18/min. There was no jugular venous distention. Cardiac examination showed a localized 1/6 systolic ejection murmur at the left sternal border. The right lung was clear, while the left lung demonstrated dry rales over the lower two thirds. There was no cyanosis, clubbing, or edema.

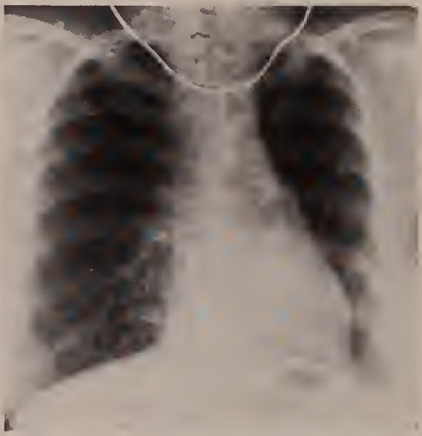

FIGURE 1. Posteroanterior radiograph of the chest demonstrating a diminutive left hilum and hypoplastic lung.

Arterial blood gas levels were as follows: pH, 7.44; $PaCO_2$, 33 mm Hg; PaO_2, 85 mm Hg; and 94% saturation breathing room air. Pulmonary function tests showed mild restriction (forced volume capacity, 72% predicted; total lung capacity, 82% predicted) with a slightly reduced diffusing capacity (71% predicted). Echocardiographic examination showed normal-sized cardiac chambers, valves, and function. A chest roentgenogram (Fig 1) revealed a diminutive left hilum without arborization of the pulmonary artery and a decreased left lung volume. Computed tomography of the chest with contrast (Fig 2) demonstrated that the main and right pulmonary arteries were of normal caliber, while the left pulmonary artery tapered abruptly after crossing over the bronchus. Additionally, irregular vascular markings and vessels, presumably of bronchial origin, were seen arising from the left posterolateral chest wall. A perfusion scan (Fig 3) confirmed the marked reduction in blood flow to the left lung. The pulmonary parenchyma revealed interstitial changes predominantly in the left lung.

DISCUSSION

Congenital left pulmonary artery agenesis associated with pulmonary hypoplasia, a left aortic arch, and normal cardiac anatomy is an uncommon clinical presentation. A diverse spectrum of maternal and fetal abnormalities have been associated with pulmonary hypoplasia.[1,2] Left pulmonary artery absence is more frequent and is typically associated with a congenital cardiac or great vessel abnormality. The absence of a right pulmonary artery is usually not accompanied by congenital abnormalities.[1,3] The congenital cardiac or great vessel abnormalities have included patency of the ductus arteriosus, tetralogy of Fallot, atrial septal defect, coarctation of the aorta, right aortic arch associated with an absent left pulmonary artery, and Eisenmenger syndrome.[4,5] Radiographic changes found with this clinical condition include absence or marked diminution of the normal hilar markings on the affected side, an affected lung which may appear smaller than normal, and elevation of the diaphragm on the abnormal side.[6] The pulmonary parenchyma of the affected lung is usually supplied by enlarged arteries of the systemic circula-

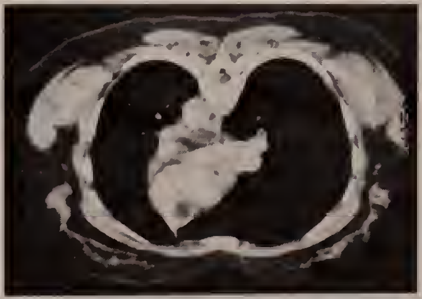

FIGURE 2. Thoracic computed tomographic scan demonstrating absence of the left pulmonary artery.

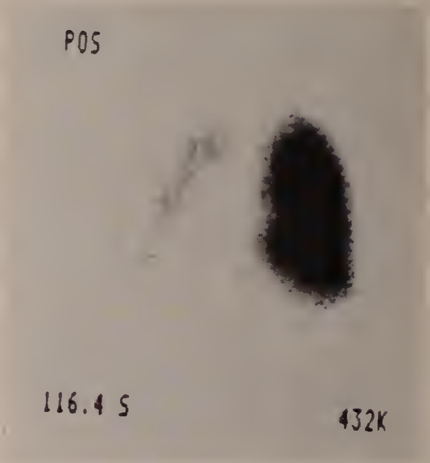

FIGURE 3. Posterior perfusion scan of the lungs showing marked absence of blood flow to the left lung.

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tion which are either bronchial or aberrant in origin.[4,7] Hemoptysis can be a major complication of an absent pulmonary artery and is felt to be secondary to an excessive collateral circulation.[4]

Physiologic measurements in these patients have revealed a significantly reduced oxygen uptake in the affected lung, a nearly normal ventilatory volume, normal or slightly reduced vital capacity, and normal pulmonary artery pressure at rest associated with minimal pulmonary hypertension during exercise.[4,6] An exercise study performed with this patient demonstrated an elevated heart rate and severely reduced oxygen uptake for her level of activity, hyperventilation, and normal arterial saturation.

The diagnostic approach in this case was unfortunately limited to a noninvasive evaluation. A recent review by Curranino et al[5] demonstrated that cardiac catheterization and pulmonary angiography were the techniques most frequently used to document the underlying cardiopulmonary and vascular anatomy. Noninvasive evaluation of pulmonary hypoplasia has been limited to ultrasonography performed during the prenatal period.[8] The patient in this case declined further invasive procedures.

Surgical resection has been reserved for selective cases that involve severe massive hemoptysis and recurrent pulmonary infection.[1] This patient is currently enrolled in a pulmonary rehabilitation program.

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FROM THE LIBRARY

DISSECTING ANEURYSM OF THE AORTA

Diagnosis and Operative Relief of Acute Arterial Obstruction Due to This Cause

. . . Dissecting aneurysm of the aorta is a hemorrhagic extravasation which begins most often in the ascending portion of the arch of the aorta and spreads through the media, usually adjacent to the adventitia. In its course the extravasation may cause compression of any of the branches of the aorta, including the coronary arteries. In the majority of cases severe hypertension with systolic pressure of over 200 has been pre-existent. Dissecting aneurysm is not the common terminus for severe hypertension, suggesting that the aorta in these cases must be more fragile than usual. Obliterating sclerosis of the vasa vasorum with consequent degeneration of the media has been frequently described. The findings are those of malignant hypertension, with the vasa vasorum the site of the most advanced arteriolar lesions. Syphilis is rarely a cause of this condition because the layers of the luetic aneurysm fuse, and rupture is usually not accompanied by splitting. Likewise accidental traumas may cause rupture but rarely dissection. Status lymphaticus and coarctation of the aorta have been described as causing rupture with a certain amount of dissection.

According to Kellogg and Heald, sixty-five per cent of cases of dissecting aneurysm die immediately from complete rupture of the aneurysm. Rupture is most frequently into the pericardium, pleural cavities or mediastinum. Another fifteen per cent die in a few days (the patient they reported died of gangrene of the lower extremities). The remainder have a good chance of recovery but may rerupture.

Healing occurs by (a) absorption of blood and eventual fusion together of the layers of the media or (b) by establishment of a new arterial channel along the course of the dissection, lined by endothelium and communicating above and below with the lumen of the aorta or one of its branches. The operative procedure used in the case which is being reported would facilitate healing by either method as well as relieve the immediate obstruction.

The diagnosis of dissecting aneurysm should be *suspected* when an individual, usually (75%) male, usually with history or evidence of severe hypertension, suddenly develops pain at the level of the precordium without evidence of acute myocardial damage by E K G, fall in blood pressure or marked rise in pulse. Often the pain in these cases is more severe over the thoracic spine or in the epigastrium than under the sternum. The diagnosis should be made when following the above, signs of acute arterial blockage or internal hemorrhage develop. Valvular heart disease, auricular fibrillation or other source of embolism can ordinarily be ruled out. Infection, cachexia or mechanical pressure causing thrombosis are also absent in dissecting aneurysm.

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JAMES W. BULMER, MD
RICHARD DERBY, MD
(*NY State J Med* 1935; 35:1200–1202)

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Limiting the working hours of interns and residents

TO THE EDITOR: I read with amusement the commentary by the Associated Medical Schools of New York that was published in the November 1987 issue of the *Journal*.[1] It strikes me as an attempt to deny that a traditional, but pointless and dangerous system of physician coverage is in place in many of our hospitals.

I will assume that the authors are simply misinformed, but at the very institution that they list as their address, a number of services are routinely staffed on an "every-other-night" basis; indeed, some are on "staggered every other" (in a two-week period, a resident will be on call Monday, Wednesday, Saturday–Sunday, Tuesday, Thursday–Friday). While it is true that a resident on call for the Saturday AM to Monday PM shift is "not necessarily awake for that period of time," I can vividly remember from my time there that when on call on some of the services, the only sleep generally available was that obtained when one collapsed for an hour or two in a doctors' or nurses' station. Even assuming that a service had an every-third-night schedule, when the resident had not slept the night before, he or she was usually working below standard the following day. While it is hard to disagree with the concept of continuity of care, I challenge the authors of the commentary to provide any evidence that the "quality of care" provided by an exhausted resident is in any way superior (or even equivalent) to that demonstrated by a thoroughly briefed and well-rested replacement. In my opinion, we should limit the length of the resident's day to a maximum of 24 hours, followed by a minimum of eight hours off. Discussions with a number of residents at varying levels and from several different specialties revealed that there is usually no major difficulty in completing a 24-hour on-call period, even without sleep, as long as relief is available at the end of the shift. While this would result in the availability of fewer residents during the day (in general, about one third fewer), it would be infinitely easier to implement than the proposed 16-hour limit.

And what of the concern over the recommendation that 24-hour, in-person attending supervision of all residents be provided? It seems logical that physicians still in training not be allowed a level of practice in a teaching hospital that would be considered illegal if they were in a different building. All accrediting specialty boards require the careful supervision of residents during their training, and require that independent performance of an activity only be permitted after competence is demonstrated. All too often, supervision of junior residents is totally in the hands of senior residents, and the seniors are automatically granted independence on the basis of rank, with little or no attending participation in their day-to-day actions. This problem is magnified at night when few if any attending physicians are normally in the hospital, and more of the patients admitted are seriously ill. Faculty attendings who would rather spend their time in research or private practice are easily convinced that the system is working well, since to decide otherwise might mean an increase in their own required involvement. Part of the solution is obvious: at least during the day, active, attending-level supervision of the care of *all* patients should be the rule, and faculty members should be required to provide it as a prerequisite for their own privileges. To require faculty attendings to spend nights at a teaching hospital on a routine basis seems radical. However, to require that they at least review by phone the case of any seriously ill patient admitted, and follow it up by a personal visit in the morning, might prove to be an acceptable alternative in most instances. I submit that simply hiring a licensed "house doctor" to cover at night might satisfy the need on paper, but would not be best for residency training or patient care.

I share in the concern that issues such as these have become the business of the state legislature. However, when the people who should address the problems continue to deny their existence, they invite outside intervention by those less knowledgeable. The obvious way to keep government out of the day-to-day supervision of residency training is to demonstrate that the problems are recognized and that a good-faith effort is being made to resolve them. The commentary by the Associated Medical Schools of New York demonstrates just the opposite. The time to get their heads out of the sand is long overdue.

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1. Associated Medical Schools of New York: A view of the proposed New York State regulations governing the professional activities of residents. *NY State J Med* 1987; 87:587-589.

New York State regulations governing the work of residents

TO THE EDITOR: The commentary, "A view of the proposed New York State regulations governing the professional activities of residents,"[1] expresses the grave concern of the Associated Medical Schools of New York over the potentially harmful impact of a series of proposed New York State regulations governing the work of residents in New York hospitals. One can empathize with the loss of control which has obviously been felt by many who are burdened with the responsibility of carrying out medical training programs. Unfortunately, the recommendations of the Ad Hoc Committee on Emergency Services represent a response to tragic deficiencies that have existed for many years. These deficiencies have not gone without notice. From time to time, physicians in training have registered complaints with the directors of training programs and through the Committee of Interns and Residents in New York City without adequate response. How well I remember a specific instance in which an extremely bright and capable surgical resident, after having been on duty for approximately 36 hours at a hospital in the Bronx, New York, apparently fell asleep at the wheel of his car. He crashed into the rear end of a truck and tragically suffered brain damage as a result. We did witness some changes in the house staff on-call schedules at our hospital at that time, but little change occurred at other institutions.

In addition to complaints about the abuse to physicians in training, many have eloquently expressed their concern about the judgment errors that result when physicians in training are required to work excessive hours without adequate relief. Despite these expressions of concern over the years, inadequate solutions have been implemented by those with the authority to do so. In his editorial, "Supervising interns and residents and limiting their working hours," in the August 1987 issue of the *Journal*,[2] Pascal James Imperato, MD, provides an insightful overview of the issues as well as a refreshing hint that reasonable solutions might be at hand.

Perhaps a more appropriate response of the Associated Medical Schools of New York to the recommendations of the Ad Hoc Committee on Emergency Services might have been to acknowledge their own failure to respond adequately to the many cries of concern about excessive on-call hours, and to express their gratitude to the ad hoc committee for taking an active role in attempting to address the overall problem. To increase the likelihood of achieving optimal solutions to the many facets of the problem, they could have offered to work together, without charging anyone with intrusion into their territory.

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1. Associated Medical Schools of New York: A view of the proposed New York State regulations governing the professional activities of residents. *NY State J Med* 1987; 87:587-589.
2. Imperato PJ: Supervising interns and residents and limiting their working hours. *NY State J Med* 1987; 87:425-427.

Fatigue in medical training

TO THE EDITOR: The juxtaposition, in the same issue of the *Journal*, of an article covering Maimonides' description of his fatiguing clinical practice[1] with Dr Emanuel E. Garcia's complaints of "the misery" of medical training was fortuitous.[2] It suggests that being "bone-weary" may not be unique to the training period. Perhaps learning to remain enthusiastic and compassionate even when personally exhausted is part of medical education.

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1. Rosner F: The medical writings of Moses Maimonides. *NY State J Med* 1987; 87:656-661.
2. Garcia EE: Sleep deprivation in physician training. *NY State J Med* 1987; 87:637-638.

Overworked residents

TO THE EDITOR: I read with great interest the commentary in the December 1987 issue of the *Journal*, "Sleep deprivation in physician training," by Dr Emanuel Garcia.[1] An important reason for the propagation of our unconscionable residency system is that physicians trained a generation ago did not experience the problems of overwork and chronic exhaustion that we now experience daily. They believed their residencies to be an essential part of their education; they just cannot fathom how dramatically the system has changed in a single generation.

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1. Garcia EE: Sleep deprivation in physician training. *NY State J Med* 1987; 87:637-638.

Sleep deprivation in internship and residency training

TO THE EDITOR: The commentary by Dr Emanuel Garcia[1] is extremely troubling on several counts. It appears to have been written as an outgrowth of the Bell commission report and the publicity attendant on that report. Indeed, Dr Garcia mentions the newspaper coverage of the issue specifically. The commission itself arose from the notoriety accompanying a single case and, to an unfortunate degree, reflected the state's interest in appearing to be dealing with what were alleged to be clearly demonstrated deficiencies. In this public setting, the validity of the conclusions drawn is, at least, open to review.

I do not suggest that sleep deprivation is desirable, either as an absolute or as a part of medical training. Indeed, my opinion is that it is often counterproductive. More important, though, is the question of whether the significant changes which are being implemented by the state, largely based on such opinions and on what is "obvious," are justified at this time. If the study of medicine teaches us anything it is that what is obvious, what "everyone knows," is not necessarily true; that dispassionate scientific study is a far better tool for decision making than is a reflex response to popular opinion. The study by Reznick and Folse[2] on sleep deprivation among surgical residents, while noting reports which indicate significant concerns, at the very least raises questions about the validity of Dr Garcia's primary contention. There is certainly reason to believe that whatever effects there are may not be uniform, and, therefore, should not be addressed in any single sweeping "reform" which is not uniformly appropriate. In view of the major impact of the anticipated changes in the method of delivery of health care, should they not be better grounded scientifically?

Equally troubling, though, is the indictment of "the system" for inflicting "a common form of torture employed in oppressive political regimes" as well as turning "compassionate people into cynical automata." Such characterizations are themselves, to use Dr Garcia's terminology, "hogwash." It is easy to blame an undefined "system," a "they," for all the problems one believes to exist. It is certainly easier than demonstrating the validity of the accusations. Such claims are the coin of populists and politicians, and, unfortunately, we have come to accept them, in the political sphere, with a shrug rather than a response. For such arguments to be used by scientists, however, raises serious questions about the scientific method and the reasoning of those who employ them. Certainly physicians and other scientists are entitled to opinions, but they should be treated as such and not used as the basis for wide-ranging changes in well-established processes. Those changes must result from careful and impartial scientific study.

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1. Garcia EE: Sleep deprivation in physician training. *NY State J Med* 1987; 87:637-638.
2. Reznick RK, Folse JR: Effect of sleep deprivation on the performance of surgical residents. *Am J Surg* 1987; 154:520-525.

In reply. I wish to thank Dr Rosen for his interest in my commentary.[1] Any discussion of the important issue of sleep deprivation in physician training should prove valuable. However, I would like to correct certain assumptions and misconceptions, and to address some of the issues in Dr Rosen's letter.

My commentary was written in June 1987, shortly after I had completed my internship. It was solely inspired by my experiences and observations, and was completely independent of the Bell commission report, of which I was ignorant at the time. Except for a few minor alterations, it appeared in the *Journal* as it was originally composed.

Although Dr Rosen claims not to suggest that sleep deprivation is desirable, he nevertheless avers that the report by Reznick and Folse[2] "at the very least raises questions about the validity of Dr Garcia's primary contention." In fact, the study by Reznick and Folse is, as the authors themselves freely admit, a very limited one. It tested three areas of performance by surgical residents—factual recall, ability to concentrate, and manual dexterity—and found them not to be significantly affected by moderate sleep loss (defined as less than three hours of sleep per 24-hour period). Despite these findings, the authors raise concerns about the role of sleep deprivation in the psychosocial sphere, and advocate the elimination of periods of severe sleep deprivation. They did not investigate the consequences of chronic, periodic sleep loss, such as is routinely encountered in residency training. On the other hand, a number of authors have found that the sleep deprivation incurred in residency results in cognitive and motor impairment, as well as psychopathology.[3-6] I believe that these studies, and the testimony of fellow house staff that I have accurately represented the state of affairs, support my claims.

I find it difficult to understand Dr Rosen's contention that my method and reasoning should be in question because I have noted the use of sleep deprivation as a means of torture (the Inquisition, by the way, employed it), and because I have described the changes in attitude that are, at least in part, the result of this torture. Is it unscientific to report that residents can become cynical, robot-like, and less compassionate when sleep-deprived, when they (myself included, of course) admit to such changes and when their behavior betrays the transformation? Is it unjustified to cast the blame for this inhumane treatment of house staff on a system that leaves no alternative, and that has failed to heed the many calls for reform in the past? As McCue's sensitive article has shown,[7] the "system" has not been functioning quite as well as Dr Rosen would like us to think.

As I stated in my commentary, I believe that the effects of sleep deprivation are quite profound. I wholeheartedly welcome further investigation, but I do not believe that the much-needed reforms should be stymied. Given the evidence to date, I join with Asken and Raham[8] who, in their review of the literature on resident performance and sleep loss, write: "The burden of proof that sleep-deprived night call is an educational experience and an exemplar of competent and humane care rests on its proponents."

It is a great pity that the honor of making the long-overdue changes in the direction of humanity and better medical care will go to the public, and not to the organized medical profession itself.

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ERRATA

There was a manuscript error in the article by Randall Bloomfield, MD, in the January 1988 issue of the *Journal*. The first line of the third paragraph on page 6 should read as follows: "The relationship between doctors and their hospitals has been affected by the changing market conditions of an aging population, an interest in wellness, and a decreasing number of hospitals" [not ". . . an increasing number of hospitals" as it originally read].

The name of Jack A. DeHovitz, MD, was inadvertently omitted from the list, published in the February 1988 issue, of referees who reviewed manuscripts for the *Journal* in 1987. Dr DeHovitz has been one of our most active peer reviewers, and we regret the omission.

LEADS FROM EPIDEMIOLOGY NOTES

Reprinted from the January 1988 issue of Epidemiology Notes (*Vol. 3, No. 1*), *published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

Syphilis

During the first three months of 1987, 8,274 cases of early syphilis were reported in the United States. This represents a 23% increase over the same period for 1986. The largest percent changes took place in California (41%), Florida (92%) and New York (71%). Increases occurred mainly in the heterosexual population, primarily among blacks and females.

This information has prompted two major concerns: that increases of heterosexual syphilis will increase the most severe form of the infection—congenital syphilis; and those infected with syphilis may be at increased risk for human immunodeficiency virus (HIV) infection.

Clinical aspects

The causative organism of syphilis is a spirochete, *Treponema pallidum*, which is passed person to person by sexual intercourse or from mother to child in utero. The disease is characterized by acute primary and secondary stages, a latent period of variable length and ensuing late syphilis characterized by damage to the central nervous system and/or cardiovascular system.

The hallmark of primary syphilis is a solitary, painless, indurated lesion (chancre). Any genital lesion, whether typical or not, must be considered as possible syphilis until proven otherwise, and a high index of suspicion must be maintained for single typical lesions on other parts of the body.

In females, this lesion is often located internally. Thus, the primary stage of the disease will go undetected unless the woman is informed of possible infection by her partner, or syphilis is identified by routine testing.

Whether treated or not, the chancre will disappear. Six weeks to six months after exposure, symptoms of secondary syphilis develop; these vary but typically include flu-like symptoms, generalized lymphadenopathy, skin rash, mucous patches and hair loss. Rash on the palms of the hands and soles of the feet is particularly diagnostic of syphilis in this stage. After these symptoms resolve, the disease becomes latent. Relapses generally occur within the first year, but may take up to four years to appear. Tertiary syphilis occurs in about 25% of patients who are untreated.

Diagnosis (see Table 1)

The diagnosis of primary syphilis is conclusive if *T pallidum* is seen by darkfield examination. Failure to find the organism, however, does not rule out syphilis. A nontreponemal reagin test (VDRL or RPR) is typically performed first and a positive result is generally confirmed by use of the more sensitive fluorescent treponemal antibody-absorption (FTA-ABS) test.

Reagin tests are not usually positive until four to five weeks after infection. If initial tests are negative and syphilis is suspected, the test should be repeated at one week, one month and three months. A four-fold increase in titer for paired sera is a presumptive diagnosis of syphilis.

Reagin tests should be performed for any patient suspected of having secondary syphilis. Almost all patients having secondary syphilis will have a positive nontreponemal test; those with a VDRL of less than 1:16 should have a repeat test and a treponemal test to confirm infection.

A presumptive diagnosis of early latent syphilis is made when reagin and treponemal tests are positive.

Treatment

Appropriate treatment for syphilis is related to the stage of the disease. Schedules for treatment can be found in *Sexually Transmitted Disease Treatment Guidelines 1986*, available from: New York State Department of Health, Bureau of Communicable Disease Control, STD Control Program, Corning Tower, Room 649, Empire State Plaza, Albany, NY 12237.

Penicillin is the antibiotic of choice for syphilis. For patients allergic to penicillin, either tetracycline or erythromycin is generally used; these drugs, however, have not been as extensively evaluated as penicillin and compliance with use of oral medicines is a problem with some patients. Tetracycline cannot be used in the pregnant patient and erythromycin treatment during pregnancy has been associated with reports of treatment failure. Therefore, in certain patients, it is essential to confirm penicillin sensitivity

TABLE I. Diagnostic Criteria

Primary syphilis	
Definite:	Specimen from lesion showing presence of *T pallidum*.
Presumptive:	Presence of a typical lesion and: a) reactive reagin test; b) the titer of the present reagin test increased 4-fold over the last test; c) the lesion appeared within 10–90 days of exposure to a partner with syphilis.
Suggestive:	Presence of a lesion resembling a chancre at the site of sexual exposure regardless of the result of a reagin test.
Secondary syphilis	
Definite:	Positive nontreponemal serologic test. Darkfield demonstration of *T pallidum* confirms the diagnosis.
Presumptive:	Presence of signs of secondary syphilis and either a newly reactive serologic test or 4-fold increase in titer.
Suggestive:	Signs of secondary syphilis within a year of exposure to a sexual partner with syphilis despite a nonreactive or "rough" reagin test. A suspect prozone can be confirmed by use of quantitative VDRL or RPR test.

before alternative treatment is prescribed. All individuals who have been exposed to syphilis within the preceding three months should receive treatment even if they have no signs or symptoms of the disease.

Congenital infection

Newborn infection results from passage of the organism across the placenta of the infected mother to the fetus. Several studies have reported that infection can be acquired as early as eight to nine weeks. The likelihood of the fetus's acquiring syphilis depends upon the stage of the mother's infection—the longer the mother has had the disease, the lower the chance of fetal infection. Also, the earlier in pregnancy that syphilis is detected, the greater the chance that the infant will be born free of this disease.

Congenital syphilis is a preventable infection. Adequate prenatal care should assure detection of almost all infected women. New York State requires that every woman being seen by a physician at the first visit during pregnancy have blood drawn and tested for syphilis. This is the most important method presently available to prevent congenital syphilis. Since some women become infected late in pregnancy, routine third trimester testing should be done in high risk women.

Adolescent, nonwhite and unmarried mothers are at increased risk for transmission of congenital syphilis. In the general population, 95% of mothers have at least one prenatal visit, whereas only 52% of mothers of syphilitic babies have prenatal care. Therefore, although prenatal testing is the most important approach to congenital control, other measures are clearly necessary. These include renewed emphasis by the STD Control Program upon specific activities to detect syphilis in pregnant women. Efforts are being made to establish outreach programs with facilities likely to have needy pregnant women as clients, eg, Social Service facilities, WIC clinics and women's detention facilities. It is equally important that health care providers pay special attention to follow-up of pregnant women who have reactive serology to ensure they receive treatment and to ascertain that sexual contacts have been treated. All named sexual partners must be reached to prevent reinfection of the pregnant woman late in pregnancy. All women seen for any genital infection should be queried regarding their last menstrual period. If a woman has not had a period in six weeks, she should immediately be tested for pregnancy.

HIV and syphilis

An increasing number of reports of treatment failures of primary and secondary syphilis which resulted in development of neurosyphilis has raised questions about the ability of penicillin to eradicate this infection particularly in immunocompromised individuals. Homosexuals who engage in sex with numerous partners are at risk for both syphilis and HIV infection. Disease control in this population is a current challenge. At present, the most important approach is early detection of syphilis. All HIV positive people should be tested for syphilis and all those with syphilis should be tested for HIV. Since HIV infection is acquired sexually, anyone at risk for a sexually transmitted disease is at risk for HIV/AIDS.

Summary

Several possible explanations can be offered for this rapid upturn in syphilis:

1. The geographic areas of major increases of syphilis correspond to areas of highest rates of penicillinase-producing *N gonorrhoeae* (PPNG). For the past several years, these areas have increasingly used spectinomycin rather than penicillin for treating gonorrhea. However, unlike penicillin, spectinomycin is not effective for treating incubating syphilis. This hypothesis should be self-limiting since most areas have now begun using ceftriaxone, a drug that has better success in curing coexistent incubating syphilis.
2. There is an increasing concern about sex-for-drugs relationships among heterosexuals based on crack addiction. This hypothesis holds that multiple partner (often anonymous) exposures have led to major increases in syphilis transmission.
3. The current focus on HIV infection and AIDS by health care professionals may have diverted attention and resources away from other serious sexually transmitted diseases.

Syphilis is still a public health problem with serious consequences. Education and awareness in the medical community remain the most effective countermeasures. For more information on syphilis, contact the STD Control Program at (518) 474-3598.

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Changing cancer incidence rates in New York State, 1976–1984

Over the past decade, cancer incidence rates have remained fairly constant for many of the major sites: colon, rectum, breast, pancreas, uterus, ovary, bladder, kidney, brain, lymphoma and leukemia. This report focuses on three sites which have experienced changing incidence over the decade: cancer of the uterine cervix (invasive and in situ), cancer of the lung and malignant melanoma of the skin. Age-adjusted rates (adjusted to the 1970 US population) are presented for New York State, 1976 through 1984.

Cervical cancer

The incidence rate for invasive cancer of the uterine cervix has declined in New York State over the decade, from about 12 cases per 100,000 in 1976 to about 10 cases in 1984. This decline is considerably less than was seen in the upstate New York data over the previous two decades. Most of the decrease is attributed to two factors. First, Pap smears are detecting cervical abnormalities earlier in the disease process. Early treatment of in situ cancers has prevented the development of invasive cervical cancers which have a poorer prognosis. Second, the increased number of hysterectomies performed in the older age groups had effectively removed these women from further risk of developing cancer of the cervix.

At the same time, the incidence rate of carcinoma in situ of the uterine cervix has declined over 25% from approximately 26 cases per 100,000 in 1976 to 19 per 100,000 in 1984. A 35% reduction occurred in women aged 25–34. The incidence rate, however, remains almost 2½ times higher in black than in white women in New York State.

The explanation for this decline in the rate of in situ cervical cancer is not clear. It may have resulted from past intensive efforts of screening where prevalent cases were detected; however, there is some evidence that pathologists are now classifying some cases as dysplasia that were previously called premalignant in situ.

Certain population subgroups are not being screened adequately to detect this disease early. They include blacks, immigrants and women from lower socioeconomic groups. Physicians and other health care providers should encourage women to have a Pap smear every three years after two negative smears one year apart. Women over 20 years of age and sexually active women under 20 should be screened.

Lung cancer

Cancer of the lung has been increasing rapidly in New York State and is the leading type of cancer in men and the second leading type (after breast cancer) in women. Cigarette smoking has been identified as the major cause of lung cancer. The lung cancer incidence rate has continued to rise more sharply in females than in males in recent years, reflecting the continuing and growing popularity of cigarette smoking among women over the past several decades. There is some evidence nationally that lung cancer incidence and mortality rates in men are leveling off and may be declining. Data for New York State as presented in Table I indicate the incidence rate in females has increased about 36% from 1976 to 1984, while the rate in males shows some evidence of a leveling off and possible decline. Provisional data for 1985 indicate a continuing trend.

The elimination or reduction of tobacco exposure should continue to be a major goal of public health programs as well as private practitioners. Educational programs designed and implemented at the local level should focus on discouraging children and young adults from starting to smoke. Campaigns should also include smoking cessation programs for adults who already smoke. Women and blacks should be targeted specifically in antismoking campaigns.

TABLE I. Age-Adjusted Cancer Incidence Rates per 100,000,[1] Selected Sites by Sex, New York State, 1976–1984

	Uterine Cervix		Lung[4]		Malignant Melanoma of Skin[5]	
Year	Invasive[2]	In situ[3]	Male	Female	Male	Female
1976	12.33	25.75	81.07	23.86	5.56	4.30
1977	11.19	26.09	80.38	24.82	6.37	4.86
1978	11.12	22.43	83.08	24.82	5.94	4.68
1979	11.37	22.20	82.72	27.48	6.53	4.91
1980	10.91	20.49	84.66	28.20	7.42	5.50
1981	9.51	19.96	82.51	29.60	7.03	5.28
1982	9.77	20.53	84.79	31.94	7.18	5.67
1983	10.06	20.13	85.70	33.34	8.32	5.91
1984	10.26	18.75	83.39	32.39	7.55	5.50

Source: New York State Cancer Registry.

[1] Age-adjusted to 1970 US census.

[2] International Classification of Diseases, 9th rev, 1975, 180.

[3] ICD, 1975, 233.1.

[4] ICD, 1975, 162.

[5] ICD, 1975, 172.

Skin cancer

Melanoma is a cancer of melanocytes, the skin cells that produce the dark pigment, melanin. The incidence rate is highest among whites. Melanoma is related to exposure to ultraviolet radiation and there is some evidence that heavy, blistering overdoses are responsible. Family predisposition and hormonal factors may also play a part. The malignant melanoma incidence rate has increased steadily in both men and women throughout the decade in New York State. In 1976, there were about 5.5 cases per 100,000 in men and 4.3 in women. By 1984, there were 7.6 cases in men and 5.5 in women.

The data on malignant melanoma indicate that high risk individuals (those with blond hair, light skin color and those who sunburn easily) should avoid overexposure to sunlight and use sunscreens and protective sunglasses. Early diagnosis of this cancer improves survival rates substantially.

For further information about cancer incidence in New York State, contact Patricia Wolfgang, associate director of the Cancer Registry, at (518) 474-2255, or write: Cancer Registry, New York State Department of Health, Corning Tower, Room 536, Empire State Plaza, Albany, NY 12237.

Reference

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Immunizable diseases in children with HIV infection

Two unimmunized children with acquired immunodeficiency syndrome (AIDS) died recently of measles pneumonia. They were the first deaths attributable to measles in children in the United States since 1983. The likelihood of serious complications of measles or varicella in children with HIV infection is not known. Like children with leukemia or other immunodeficiency diseases, symptomatic

HIV-infected children with significant exposure to measles or varicella should receive passive immunization with immune globulin.

These two reported deaths from measles suggest that HIV-infected children may be at increased risk of severe measles disease. Health care providers are encouraged to evaluate children with measles pneumonia or encephalitis and their families for possible risk factors for HIV infection. Voluntary serologic testing and counseling are recommended for those children with complications of measles who have risk factors for HIV infection.

Parents at risk for HIV infection include intravenous drug users; bisexual men; hemophiliacs; those who were born in countries where heterosexual transmission is thought to play a major role; transfusion recipients; and sexual partners of those at risk.

Immunodeficient individuals have a higher risk of developing vaccine-associated poliomyelitis than normal individuals (either as vaccine recipients or contacts). Measles pneumonia has been reported in leukemic children given a less-attenuated measles vaccine than is currently used in the United States. The risks and benefits of immunization of HIV-infected children with live virus vaccines is not known with certainty and requires the prompt reporting and evaluation of vaccine-associated adverse events. The Immunization Practices Advisory Committee (ACIP) recommendations for the immunization of HIV-infected children were published in the *Morbidity and Mortality Weekly Report* (Vol. 35/No. 38, 9/26/86, pp 595–598) and endorsed in a State of New York Department of Health Memorandum (Public Health Series 86-129).

In order to maintain appropriate immunization policies for HIV-infected children, the New York State Department of Health is requesting immediate reporting of serious complications of vaccine-preventable diseases and vaccine-associated adverse events. The following conditions should be immediately reported to a regional immunization program representative or the New York State Immunization Program central office (518-473-4437):

- measles pneumonia;
- measles encephalitis;
- pneumonia or encephalopathy following MMR (measles-mumps-rubella trivalent vaccine);
- neurologic illness (particularly paralytic disease) following receipt of OPV or contact with an OPV recipient.

CLINICAL ELECTROCARDIOGRAPHY. A PRIMARY CARE APPROACH

By Ken Grauer, MD, and R. Whitney Curry, Jr, MD. 544 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $24.95 (paperback)

The authors wrote this book about electrocardiography to "accommodate both the beginner and the experienced electrocardiographer." Ken Grauer, MD, is a certified family practitioner. R. Whitney Curry, Jr, MD, is certified in both internal medicine and family practice. Both are associate professors in the Department of Community Health and Family Medicine, College of Medicine, University of Florida. Dr Grauer is the assistant director and Dr Curry is the director of the Family Practice Residency Program in Gainesville. Both are experienced teachers of ECG interpretation to physicians and students.

The text is divided into three major sections plus an appendix: Part I deals with basic principles, part II with clinical applications, and part III with review exercises in ECG interpretation. The section on basic principles includes a description of the conduction system of the heart, the ECG waveforms and intervals, a suggested systematic approach to ECG interpretation, rate and rhythm, some major dysrhythmias, and QRS axis in the frontal plane. Also included is a theoretical consideration of normal and abnormal depolarization and repolarization, cardiac hypertrophy, atrial enlargement, genesis of pathologic Q waves, and coronary circulation. The section on clinical application deals with the ST segment in health and disease, intraventricular conduction delays, accessory pathways, myocardial infarction, chamber enlargement, and pulmonary embolism. The third section, which is a review in ECG interpretation, is grouped into metabolic disturbances, atypical ECGs in healthy asymptomatic subjects, ECGs in patients who have cardiomegaly by chest film, ECGs with tall R waves in lead V_1, ECGs in patients with chest pain, and ECGs with pseudoinfarction patterns. The appendix contains a list of abbreviations used in the text, a "handy references guide," and a short bibliography.

The illustrations and tables are generally of good quality and are germane. The section on dysrhythmia is limited. The authors agree that the fundamental defect in right bundle branch block is one of delay of the terminal QRS conduction, but still call an ECG (Figure F-4) with an RSr′ pattern in V_1 that does *not* have a broad S wave in leads I, aV_L, and V_6, an incomplete RBBB.

The use of the term *strain* when referring to the repolarization changes associated with myocardial hypertrophy is unphysiologic. It is better to call them what they are, ie, ST-T changes associated with cardiac hypertrophy.

No mention was made of the Mahaim type of accelerated conduction during the discussion of accessory pathways (but Figure 8-21 may be an example of it) although both Wolff-Parkinson-White and Levine-Ganong-Lown types were covered. The authors stated that the T-wave inversions in leads II, V_5, and V_6 of Figures 5–13 are deep and symmetric due to ischemia. On the contrary, they are asymmetric, suggesting left ventricular hypertrophy.

The important topic of pacemaker electrocardiography was not discussed at all.

Despite the obvious flaws, the authors are talented teachers, are well organized, have a sense of humor, and have added a useful text to the medical literature. I have a strong feeling that it is impossible to write a book to reach such a broad audience as these authors attempt. The text should be useful to those who are beginning their studies of electrocardiography and to those experienced ECG readers who have not recently read other books on ECG or kept up with the current literature and wish to refresh themselves.

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MANUAL OF EXERCISE ECG TESTING

By Edward K. Chung, MD. 178 pp, illustrated. New York, Yorke Medical Books, 1986. $30.00 (hardcover)

Dr Chung has written another excellent text on exercise electrocardiography. Using an outline format, with the content presented in a list form, he provides extensive technical, physiologic, and procedural information. The author's expertise is well known and the text reflects a careful review of the literature. The book presents the major options available to the professional in regard to methods, protocols, and interpretive criteria. The logical format allows for easy access to required information.

The content of the book is extensive and covers the critical areas relevant to an ECG testing laboratory, such as indications for and contraindications to testing, selection of protocols, and interpretation of exercise ECG tests. Specific chapters on testing of individuals prior to their beginning an exercise program, and on exercise prescription for both healthy individuals and patients with coronary heart disease, are comprehensive and provide valuable, specific "how to" information. The chapter on exercise testing with myocardial imaging provides a concise basic review of the principles and technical limitations of such tests. These will be of help to physicians performing or ordering exercise thallium studies and exercise radionuclide angiocardiograms. The book contains numerous tables and illustrations that enhance the value of the text.

The author devotes appropriate attention and space to the testing of post-myocardial infarction patients prior to the hospital discharge. This section is especially informative and will provide the physician supervising the exam with the necessary information to optimize both safety and the value of the test.

One limitation of the format chosen by the author is the absence of references for specific statements (eg, sensitivity of various ECG lead configurations) and the inability to provide an in-depth review of certain specific issues. I would have preferred the inclusion of a discussion of computer-generated ST segment variables and of newer methods of ST segment analysis. Omitted is a section on indexes that correct ST segment magnitude for R wave amplitude. It would also have been useful if the author had included a discussion of methods of analysis that measure changes in ST depression with respect to increases in heart rate during exercise.

Even with these limitations, the *Manual of Exercise ECG Testing* is an excellent basic text to have available in the testing area, and a good resource for professionals who wish to review a large body of accurate information in a concise and logical format.

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CARDIOVASCULAR DISEASE AND BEHAVIOR

Edited by Jeffrey W. Elias, PhD, and Philip Howard Marshall. 196 pp, illustrated. New York, Hemisphere Publishing Corp, 1987. $32.00 (hardcover)

Most physicians are aware that intense emotions (or even mental arithmetic) can produce myocardial ischemia in patients with severe coronary disease. However, there are both strong and conflicting opinions regarding the importance of behavior on the genesis of coronary disease, its prognosis, and on the precipitation of sudden death and myocardial infarction. The very nature of behavior makes its definition for statistical purposes difficult, and this difficulty is further compounded by the multicausal nature of coronary disease. Statistical correlations with types of behavior or responses to stress are consequently low, even in the most enthusiastic studies. Any publication that can shed light on this association, in identifying either the types of stress response to avoid or the mechanisms involved, is welcome. This goal, however, is not achieved in this book.

This volume is the result of the Third Annual Interfaces in Psychology Conference held at Texas Tech University. It uses a multidisciplinary approach to examine the relation of behavior to both coronary disease and hypertension with a focus on the effects of aging. There are seven chapters of widely varying pertinence and value for this purpose. The book is apparently directed toward a nonmedical or psychological group, since basic medical terms are often defined or placed in a glossary. There is considerable material regarding tests for the classification of behavior and personality and their statistical interrelationships. The material in a review chapter by Redford Williams, of most potential interest for physicians, has frequently been presented before, although he now identifies cynicism rather than hostility as the factor tested for in his prior studies. Two chapters of particular interest, in view of the current emphasis on silent ischemia, show that the frequency of angina relates to personality type. Other material on the effect of aging on behavior and symptomatology is of unique interest. In general there is neither a complete nor a clear view of what is known about the effects of behavior on coronary disease, and the chapters often stand separately. More thorough reviews of this subject are available elsewhere (Conference on Behavioral Medicine and Cardiovascular Disease. *Circulation* 1987; 76:1–226). This book would probably be of most value for those interested in the methodology of behavior analysis in relation to clinical disease. It supplies many important references.

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THE BASIC SCIENCE OF ONCOLOGY

Edited by Ian F. Tannock, MD, and Richard P. Hill, PhD. 398 pp, illustrated. Elmsford, NY, Pergamon Press, Inc, 1987. $37.50 (softcover)

This book is a multiauthored text written by various faculty members of the University of Toronto, and based upon a course of the same title given by the Ontario Cancer Institute/Princess Margaret Hospital. Besides stringent editing by Drs Tannock and Hill, each chapter had at least two reviews by international experts on the topic. The target audience is primarily undergraduate or graduate students and clinical or research fellows in the oncologic specialties. The goal of the book is to provide a comprehensive overview of the fundamental science of oncology.

The text is divided into three primary sections. The section on cancer causation discusses the genetic, viral, and carcinogenic etiologies of cancer and also provides an excellent overview of cancer epidemiology. The section on cancer biology includes seven chapters which give an overview of unique tumor cell properties and kinetics. The final section, entitled "Biology Underlying Cancer Treatment," is concerned with the basic science of cancer therapies. It includes discussions of radiation therapy, chemotherapy, immunotherapy, and hyperthermia.

The format of the text is well planned. Each chapter has its own brief table of contents, helpful figures or tables, a summary, and reference sections. The latter were somewhat disappointing because, with rare exception, references were from 1984 or earlier. A helpful glossary and index are provided.

I believe the editors accomplished their task well and provide an excellent overview of cancer basic science for the student. Although most of the text is quite understandable, with excellent illustrative examples, some parts would require a refresher course in mathematical models or other very basic science disciplines. This text would be of limited value for the practicing oncologist other than as an occasionally used reference or source for basic science lectures. The chapters that discuss clinical issues such as tumor markers, hormones and cancer, chemotherapy, or radiation therapy are too superficial to be useful in treatment planning. In short, this book can be recommended primarily for its target audience.

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A MULTIMODALITY APPROACH TO BREAST IMAGING

By Saar Porrath, MD. 333 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $63.50 (hardcover)

This book was written by a physician with many years of experience in dealing with women confronted with breast problems. It presents an overview of the diagnostic modalities used for all types of breast conditions, both benign and malignant, and succeeds in its purpose. Material presented does not constitute an exhaustive description of all modalities and their results. Rather, the book presents a picture of the potential value of these techniques by means of typical case reports. It is directed not to breast specialists, surgeons, or radiologists but to primary care physicians, internists, and gynecologists and to paramedical personnel who care for women with potential breast problems.

The format and organization of the book are excellent and appealing. Many illustrations are provided throughout, which are clear and well done, and which will be appreciated by readers. The book is concise, to the point, and potentially most helpful to the audience to which it is directed. Of particular interest is an emphasis on the psychological problems involved in breast conditions, especially as they relate to screening.

The book is especially recommended for general physicians who care for women with signs or symptoms of breast disease. It will assist physicians in caring for those women who may require diagnostic procedures and who may need emotional support for dealing with a variety of breast conditions.

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COCAINE ABUSE: NEW DIRECTIONS IN TREATMENT AND RESEARCH

Edited by Henry I. Spitz, MD, and Jeffrey S. Rosecan, MD. 352 pp, illustrated. New York, Bruner/Mazel Publishers, 1987. $35.00 (hardcover)

The increasing prevalence of cocaine abuse has resulted in a rapidly expanding literature on the subject, ranging from self-help guides to technical treatises. Towards the latter end of this spectrum lies *Cocaine Abuse: New Directions in Treatment and Research*, edited by Henry I. Spitz and Jeffrey S. Rosecan. This volume provides an overview of current knowledge about cocaine abuse, and poses questions for future researchers to explore. The book begins by discussing basic science issues, reviewing animal research on cocaine and the human neurobiology of the drug. The next section focuses on treatment, discussing the roles of individual, group, and family therapy, hospitalization, and pharmacological treatment of cocaine abusers. Current public health issues are reviewed, including the effects of cocaine on pregnant women, the debate over urine testing in the workplace, and cocaine use by special populations such as health care professionals, women, and athletes. Despite the fact that the book is authored exclusively by faculty members at Columbia University, it lacks the parochial quality sometimes associated with edited volumes that emanate from a single institution.

The strengths and weaknesses of the book reflect the imbalance in the state of current knowledge about cocaine abuse. Much more, for instance, is known about the effects of cocaine on animals and human neurotransmitters than about effective ways to help cocaine abusers and their families. Thus, the chapter entitled "Human Neurobiology of Cocaine," by Edward Nunes and Jeffrey Rosecan, presents a superbly organized overview of a prodigious and sometimes confusing literature. Chapters on treatment, on the other hand, are forced to rely more heavily on theoretical speculation and anecdotal material, since rigorous studies in this area have been relatively lacking.

Two problems with the book are its repetitiousness and the excessive number of typographical errors. Although this is an edited book, some subjects are discussed in several different chapters, sometimes with no cross references. While this makes each chapter more comprehensive and able to stand on its own merit, it makes the book somewhat longer than necessary. In general, however, this is a very solid and thorough book, which should be useful to people with a serious interest in the field of substance abuse.

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COCAINE: CLINICAL AND BEHAVIORAL ASPECTS

Edited by Seymour Fisher, PHD, Allen Raskin, PHD, and E. H. Uhlenhuth, MD. 256 pp, illustrated. New York, Oxford University Press, Inc, 1987. $24.95 (hardcover)

Is there truly a withdrawal syndrome associated with abrupt cessation of cocaine? You may not get a definitive answer to that question from most medical textbooks today. At last, there is a short medical text on cocaine which clearly states that "... abrupt cessations of dosing after many days of cocaine use does produce a mild abstinence syndrome..." Dr Seymour Fisher, professor of psychiatry and behavioral sciences at the University of Texas Medical Branch, Galveston, Texas, and his fellow editors have brought together carefully selected presentations on cocaine from the 1984 Annual Meeting of the American College of Neuropsychopharmacology. They show the complexity of the cocaine problem while hoping to stimulate much-needed multidisciplinary, well-controlled research.

The book contains seven chapters. A short history of cocaine is presented in the first chapter. Chapters 2 through 6 deal with various aspects of the neurochemistry and neurophysiology of cocaine. Much of the information presented comes from animal research. There is discussion of the behavioral and neuronal actions of cocaine which lead to its abuse potential through both reinforcing and discriminative effects. Some contributing authors believe that the primary basis for abuse is the rewarding effect of cocaine mediated by areas of the brain that subserve intracranial self-stimulation. The final chapter presents information on cocaine related to its magical and religious roles in primitive societies.

Primary care physicians will likely find that chapter 7 ("Clinical Issues in Cocaine Abuse") is the most helpful with regard to the day-to-day practice of medicine. This chapter contains a brief case presentation on cocaine abuse and information on predisposing factors, epidemiology, signs and symptoms, and treatment of both cocaine overdose and cocaine abuse. However, this chapter is by no means exhaustive. Physicians are well advised to couple the information in this chapter with some recent, well-chosen journal articles on the topic.

The text is organized, readable, and certainly not burdensome to the eyes, hands, or wallet. The frequent use of charts, figures, tables, and photographs throughout the book helps keep the reader's interest. In addition, the editors and contributing authors are well-qualified authorities on cocaine. The text itself is published under the imprimatur of the American College of Neuropsychopharmacology. Currently, this text represents a remarkable and much awaited contribution to the field of addictionology. Yet I expect that, as with some other scientific writings, this book will slowly fall into obscurity as our basic science knowledge and clinical abilities advance over time. For now, and for those colleagues who are truly interested in cocaine addiction, this book makes good reading.

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Aspirin's beneficial effects on myocardial infarction risk documented

In December 1987, an independent group monitoring the data from the Physicians' Health Study recommended terminating the randomized aspirin component of the trial more than two years ahead of schedule because of the significantly beneficial effects of aspirin treatment on myocardial infarction risk. A preliminary report of the study's findings appeared in the January 28, 1988, issue of the *New England Journal of Medicine* (318:262–264), with a more comprehensive report expected within several months.

The Physicians' Health Study is an ongoing randomized, double-blind, placebo-controlled trial aimed at determining the primary preventive effects of two distinct treatment protocols: aspirin to reduce mortality from cardiovascular disease; and beta carotene to decrease the incidence of cancer. While the beta carotene component of the trial is continuing, until a scheduled conclusion in 1990, the Data Monitoring Board of the study recommended informing participants in the aspirin study of their treatment group, as well as not delaying public disclosure of the data from that portion of the trial.

Randomization of the 22,071 participating male physicians in the US, aged 40–84 years, resulted in 11,037 physicians receiving 325 mg of aspirin every other day, and 11,034 physicians receiving a placebo. On alternate days, these same subjects received either beta carotene or a placebo. Those physicians who reported side effects, poor compliance with the protocol, or unwillingness to continue during a prestudy trial were excluded from the final study group. The distributions of baseline characteristics for the two treatment groups were virtually identical.

The board based its recommendation to terminate the aspirin trial on the findings of aspirin's "extreme beneficial effects on nonfatal and fatal myocardial infarction." In the aspirin treatment group, there was a 47% reduction in the risk of total myocardial infarction, which includes both nonfatal and fatal events. This benefit of aspirin was statistically significant ($p = 0.00001$). For this same group, there was a nonsignificant 15% increased risk for total stroke. To make the findings more specific, strokes were divided into ischemic and hemorrhagic events and then subdivided into those resulting in mild disability versus moderate to severe disability or death. Based on these subdivisions, the only statistically significant ($p = 0.02$) finding was an increased risk of moderate to severe or fatal hemorrhagic strokes among the physicians taking aspirin. The authors note that this finding was based on relatively small numbers of events. By combining nonfatal myocardial infarction and stroke with cardiovascular death, the authors sought to clarify the risk-to-benefit ratio of aspirin, revealing a significant ($p = 0.006$) 23% reduction in risk among the subjects in the aspirin group.

The authors address the question of the generalizability of these findings, due to the very low cardiovascular mortality rate among the study participants compared to white men in the US with the same age distribution. They believe that "there seems little reason to suspect that the biologic effects of aspirin would be materially different in other populations with comparable or higher risks of cardiovascular disease."

The Steering Committee of the Physicians' Health Study Research Group concluded that individual judgment must still play a major role in deciding whether to prescribe aspirin for the primary prevention of cardiovascular disease. Aspirin's benefits demonstrated here must be weighed against its associated risks of gastrointestinal discomfort and bleeding and the dangers of hemorrhagic strokes. In an accompanying editorial (pp 245–246), Arnold S. Relman, MD, stated that although the trial results are supportive of aspirin's benefits, they "do not demonstrate the value and safety of aspirin in the general population—particularly in those at risk for hemorrhagic strokes or other hemorrhagic complications of aspirin therapy."

Relman concluded that "if its highly promising preliminary results withstand the test of subsequent full reporting and further peer review, the Physicians' Health Study will be regarded as a milestone in the continuing struggle against myocardial infarction. That it may still leave many important questions unresolved should not diminish the importance of its achievement."

Drugs to combat surgical blood loss

Pharmaceutical agents that can reduce blood loss during surgery have taken on increased importance with the rising fears of infection transmission through transfusions and continuing shortages of donor blood. A recent article in the *Lancet* (1988; 1:155–156) describes new evidence that, in some types of surgery, drug therapy may reduce perioperative hemorrhage.

One such drug, epoprostenol, may reduce blood loss following cardiac surgery. Cardiopulmonary bypass can cause a decrease in platelet numbers as well as damage to remaining platelets. When administered at the time of heparinization before bypass, epoprostenol causes the number of platelets to increase during bypass and lessens the loss of platelets in the first postoperative day. However, the clinical effectiveness of this drug in reducing surgical blood loss has not been shown.

Desmopressin, which increases the concentration and activity of factor VIII:von Willebrand factor in plasma, can reduce bleeding and partial thromboplastin times. The results of a double-blind study of patients undergoing cardiac surgery were impressive. There was a 40% reduction in total drainage loss associated with a 0.3 μg/kg dose of desmopressin given during cardiac surgery but after cardiopulmonary bypass. "On average, patients who received desmopressin required transfusion of 30% less blood than those given placebo." The drug's effectiveness has also been demonstrated in noncardiac surgery.

A third drug, aprotinin, has produced dramatic reductions in perioperative blood loss. High doses of aprotinin given to patients undergoing reoperation for cardiac sur-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

gery involving cardiopulmonary bypass reduced postoperative drainage loss by an average of 81% and total hemoglobin loss by 89%. In addition, there was a 91% decrease in the mean blood transfusion requirement.

All three drugs appear to reduce surgical blood loss through their effects on platelet function, although their mechanisms of action are not well understood. Potential adverse effects of these drugs include hypotension, hyponatremia and water intoxication, and increased risks of arterial or deep venous thrombosis.

The concluding remarks question the clinical value of epoprostenol in reducing perioperative blood loss, and limit the usefulness of desmopressin to noncardiac surgery involving significant blood loss. High-dose aprotinin appears to hold the most hope for substantially reducing blood loss and transfusion needs during cardiac surgery, and "its role in non-cardiac surgery is uncertain, but warrants urgent investigation."

Financial incentives in HMOs: Is there a conflict of interest?

Alan L. Hillman, MD, MBA, from the University of Pennsylvania (Philadelphia, PA), poses the following question in a special report in the *New England Journal of Medicine* (1987; 317:1743–1748): "At what point does a financial incentive create a conflict of interest, in which physicians' behavior may be motivated substantially by pecuniary self-interest rather than by the patient's best interest?" The financial incentives in question refer to those offered to physicians in some health maintenance organizations (HMOs).

In the HMO system, the purchaser of health care, the providers of care, and the insurers share the financial risk. There are various types of HMOs, with different ownership and staff structures, varying methods of paying participating physicians, and different systems of distributing financial risk and reward. The potential effects of financial incentives may depend on these variables.

An HMO guarantees comprehensive health care services to its members for a fixed price. Therefore, the financial success of an HMO depends on the organization's ability to control the use of health care resources by encouraging a cost-conscious approach to medicine among its health care providers. This system is in stark contrast to traditional fee-for-service payment, in which a physician receives financial rewards for using more health care resources. Hillman outlines the various mechanisms used by HMOs to control the use of resources: "utilization review, education, requirements for prior approval of certain procedures, and financial incentives." Of these, he contends that financial incentives "are a particularly important influence on the behavior of physicians," noting that "it is human nature to respond to financial incentives."

Opponents of the HMO system argue that such incentives to reduce the use of health care resources may compromise the quality of care. When do cost-cutting measures take precedence over the patient's best interest, they inquire? Hillman used a survey to study the different types of financial incentives used by HMOs. These incentives may be in the form of penalties—through which physicians may lose a percentage of their payment due to overall deficits—or rewards—whereby physicians receive bonuses as a share of surpluses.

The author found that in 30% of HMOs surveyed, primary care physicians are at risk of financial penalties beyond the percentage of payment typically withheld by the HMO. Some groups set limits on these additional penalties, whereas some hold the physicians responsible for repaying any deficit. "Older plans are more likely to impose penalties beyond the withheld amount," suggesting to the author that "experience leads to additional financial incentives aimed at modifying physicians' behavior."

In most plans, the risk for deficits is shared among all primary care physicians based on the collective performance of the group, whereas 18% of the HMOs polled assess financial risk based on individual performance. Fifty-three percent of the HMOs apply financial incentives to specialists. In 40% of responding HMOs, primary care physicians must pay for outpatient laboratory tests from their own capitation payments or from a fund that includes their capitation payments. This practice may encourage the use of fewer tests for cost-cutting purposes.

The various reward systems for distributing surplus funds include a fixed dollar amount for each practitioner, a fixed percentage of the surplus shared among the group, and a bonus based on individual productivity. In determining the latter bonus, 41% of the HMOs compare physician productivity with a group average, thus placing physicians in direct competition with each other.

Although Hillman refrains from concluding that the financial incentives offered in HMOs induce physicians to compromise patient care by acting in their own financial self-interest, he does state that "certain contractual arrangements seem to be of special concern. These include plans in which the number of doctors sharing the risk is small, the rates of withholding are excessive, there are penalties beyond the withheld amounts, or outpatient laboratory tests are paid from a primary care capitation fund." However, he adds, there are many HMOs that operate without any such financial arrangements.

Future studies should examine specifically how physicians' behavior is affected by the type and magnitude of various risk and reward mechanisms. It is important to determine whether specific financial incentives are advantageous or disadvantageous with respect to patient care. Encouraging physicians to conserve health care resources excessively could create a conflict of interest between financial self-interest and quality of care.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

George Bouton Andrews, MD, Syracuse. Died December 12, 1987; age 93. Dr Andrews was a 1918 graduate of the State University of New York Health Science Center at Syracuse. He was a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

William Jacob Bearman, MD, Deerfield Beach, Florida. Died December 26, 1987; age 90. He was a 1924 graduate of New York University School of Medicine, New York. Dr Bearman was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Joseph Donnell Beebe, MD, Brooklyn. Died December 16, 1987; age 65. Dr Beebe was a 1956 graduate of Universitaire Katholique de Louvain, Faculte de Medecine, Louvain, Belgium. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

William Boyd Bell, MD, Royal Oak, Maryland. Died January 27, 1988; age 69. He was a 1943 graduate of the University of Louisville School of Medicine, Louisville. Dr Bell was a member of the American Society of Abdominal Surgeons, the Nassau County Medical Society, and the Medical Society of the State of New York.

David Bernstein, MD, Brooklyn. Died January 14, 1988; age 78. Dr Bernstein was a 1935 graduate of the New York University School of Medicine. He was a Qualified Fellow of the International College of Surgeons, a Fellow of the American College of Chest Physicians, and a Diplomate of the American Board of Otolaryngology. His memberships included the Academy of Medicine, the American Academy of Otolaryngology, the American Academy of Facial Plastic and Reconstructive Surgery, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Hans Erich Broscheit, MD, Poughkeepsie. Died December 9, 1987; age 76. He was a 1939 graduate of Friedrich-Wilhelms-Universitat Medizinische Fakultaet, Berlin, Germany. Dr Broscheit was a member of the Aerospace Medical Association, the Dutchess County Medical Society, and the Medical Society of the State of New York.

Mortimer G. Brown, MD, Auburn. Died December 29, 1987; age 103. Dr Brown was a 1908 graduate of the State University of New York Health Science Center at Syracuse. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the Academy of Medicine, the American Academy of Otolaryngology, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Samuel S. Brown, MD, Hollywood, Florida. Died January 25, 1988; age 89. He was a 1923 graduate of the State University of New York Health Science Center at Syracuse. Dr Brown was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Antonio S. Carbonell, MD, Miami, Florida. Died October 23, 1987; age 81. He was a 1929 graduate of Facultad de Medicina de la Universidad de La Habana, Cuba. Dr Carbonell was a Fellow of the American Academy of Pediatrics and a member of the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Herman J. Christensen, MD, Hyde Park. Died December 29, 1987; age 85. Dr Christensen was a 1930 graduate of Cornell University Medical College, New York. He was a member of the Dutchess County Medical Society and the Medical Society of the State of New York.

Angelo Arthur Ciocca, MD, North Tarrytown. Died January 3, 1988; age 82. Dr Ciocca was a 1930 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Hamilton J. Clarke, MD, Buffalo. Died January 14, 1988; age 85. Dr Clarke was a 1930 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Raphael Joseph DiNapoli, MD, Durham, South Carolina. Died December 28, 1987; age 85. Dr DiNapoli was a 1928 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

William Archibald Dickson, MD, Jamestown. Died December 24, 1987; age 70. He was a 1943 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. Dr Dickson was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Chautauqua and the Medical Society of the State of New York.

Herman Drexler, MD, Margaretville. Died December 22, 1987; age 73. Dr Drexler was a 1940 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the New York Cardiological Society, the American Geriatrics Society, the Delaware County Medical Society, and the Medical Society of the State of New York.

Joseph Anthony Garcia, MD, Cornwall. Died December 13, 1987; age 46. He was a 1974 graduate of Facolta de Medicina e Chirurgia dell'Universita di

Roma, Rome, Italy. He was a member of the New York State Society of Internal Medicine, the Medical Society of the County of Orange, and the Medical Society of the State of New York.

Irwin Goldberg, MD, West Palm Beach, Florida. Died December 24, 1987; age 78. Dr Goldberg was a 1937 graduate of the Universite de Paris VI, Paris, France. He was a Fellow of the American Academy of Family Practice and a member of the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Joseph Mordecai Green, MD, Staten Island. Died January 12, 1988; age 50. Dr Green was a 1966 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the New York State Society of Internal Medicine, the New York Cardiological Society, the Richmond County Medical Society, and the Medical Society of the State of New York.

Milford M. Greenbaum, MD, Poughkeepsie. Died November 18, 1987; age 79. Dr Greenbaum was a 1934 graduate of the Medical College of Virginia Commonwealth University School of Medicine, Richmond. He was a member of the Dutchess County Medical Society and the Medical Society of the State of New York.

Irving Nathan Holtzman, MD, Miami Beach, Florida. Died October 18, 1987; age 78. He was a 1931 graduate of the State University of New York Health Science Center at Brooklyn. Dr Holtzman was a Fellow of the American College of Physicians and a Diplomate of the American Board of Dermatology, Inc. His memberships included the New York State Dermatological Society, the American Academy of Dermatology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Frederic B. Kleker, MD, Spencerport. Died December 25, 1987; age 84. Dr Kleker was a 1929 graduate of McGill University Faculty of Medicine, Montreal, Canada. He was a member of the Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Jonah D. Kosovsky, MD, New York. Died January 24, 1988; age 62. Dr Kosovsky was a 1950 graduate of New York University School of Medicine, New York. He was a Diplomate of the American Board of Internal Medicine and a member of the New York County Medical Society and the Medical Society of the State of New York.

Robert Lee Letcher, MD, Fletcher, Missouri. Died September 2, 1987; age 37. Dr Letcher was a 1974 graduate of Columbia University College of Physicians and Surgeons, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Solomon Perlroth, MD, Portola Valle, California. Died December 7, 1987; age 79. Dr Perlroth was a 1931 graduate of Universitaet Wien, Medizinische Fakultaet, Wien, Germany. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Edward Stephen Pniewski, MD, Hyde Park. Died January 21, 1988; age 64. He was a 1953 graduate of New York University School of Medicine, New York. Dr Pniewski was a member of the American Occupational Medical Association, the Dutchess County Medical Society, and the Medical Society of the State of New York.

Michael David Pollane, MD, Rockville Centre. Died January 4, 1988; age 50. Dr Pollane was a 1962 graduate of Georgetown University School of Medicine, Washington, DC. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Stephan Robert Rheingold, MD, Poughkeepsie. Died December 16, 1987; age 51. He was a 1963 graduate of Emory University School of Medicine, Atlanta. Dr Rheingold was a Fellow of the American College of Gastroenterology and a Diplomate of the American Board of Internal Medicine. His memberships included the American Gastro-enterological Association, the Dutchess County Medical Society, and the Medical Society of the State of New York.

Charles Hiram Richards, MD, Lake Worth, Florida. Died September 3, 1987; age 83. Dr Richards was a 1937 graduate of Cornell University Medical College, New York. He was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Edwin Willard Roberts, MD, Pulaski. Died January 18, 1988; age 78. He was a 1935 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. Dr Roberts was a Diplomate of the American Board of Surgery. His memberships included the International Academy of Proctology, the American Occupational Medical Association, the Medical Society of the County of Jefferson, and the Medical Society of the State of New York.

Demetrius A. Scalzo, MD, Pelham Manor. Died January 5, 1988; age 81. Dr Scalzo was a 1934 graduate of Facolta di Medicina e Chirurgia dell' Universita di Roma, Rome, Italy. He was a Fellow of the American College of Obstetricians and Gynecologists and a Qualified Fellow of the International College of Surgeons. His memberships included the Academy of Medicine, the New York State Surgical Society, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Ralph George Schilling, MD, Katonah. Died November 3, 1987; age 54. He was a 1959 graduate of Albany Medical College of Union University, Albany. Dr Schilling was a Diplomate of the American Board of Pediatrics and a member of the Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Charles C. Shepard, MD, Utica. Died January 8, 1988; age 74. He was a 1942 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. Dr Shepard was a member of the Academy of Medicine, the New York State Society of Internal Medicine, the Aerospace Medical Association, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Sigmund A. Siegel, MD, New York. Died January 4, 1988; age 79. He was a 1934 graduate of New York University School of Medicine, New York. Dr Siegel was a Fellow of the American College of Surgeons and the American College of Angiology and a Diplomate of the American Board of Surgery. He was a member of the American Academy of Compensation Medicine, Inc, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Joseph Raymond Strauss, MD, Port Washington. Died December 25, 1987; age 79. Dr Strauss was a 1935 graduate of Cornell University Medical College, New York. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Peter Vitulli, MD, Garden City. Died January 18, 1988; age 88. Dr Vitulli was a 1925 graduate of Eclectic Medical College, Cincinnati. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Elias Weinstein, MD, Carmel. Died March 9, 1987; age 88. He was a 1925 graduate of the State University of New York Health Science Center at Brooklyn. Dr Weinstein was a 1925 graduate of the State University of New York Health Science Center at Brooklyn, the Putnam County Medical Society, and the Medical Society of the State of New York.

Before prescribing, see complete prescribing information in SK&F LAB CO. literature or PDR. The following is a brief summary.

Contraindications: There are no known contraindications to the use of 'Tagamet'.

Precautions: While a weak antiandrogenic effect has been demonstrated in animals, 'Tagamet' has been shown to have no effect on spermatogenesis, sperm count, motility, morphology or in vitro fertilizing capacity in humans.

In a 24-month toxicity study in rats at dose levels approximately 9 to 56 times the recommended human dose, benign Leydig cell tumors were seen. These were common in both the treated and control groups, and the incidence became significantly higher only in the aged rats receiving 'Tagamet'.

Rare instances of cardiac arrhythmias and hypotension have been reported following the rapid administration of 'Tagamet' HCl (brand of cimetidine hydrochloride) injection by intravenous bolus.

Symptomatic response to 'Tagamet' therapy does not preclude the presence of a gastric malignancy. There have been rare reports of transient healing of gastric ulcers despite subsequently documented malignancy.

Reversible confusional states have been reported on occasion, predominantly in severely ill patients.

'Tagamet' has been reported to reduce the hepatic metabolism of warfarin-type anticoagulants, phenytoin, propranolol, chlordiazepoxide, diazepam, lidocaine, theophylline and metronidazole. Clinically significant effects have been reported with the warfarin anticoagulants; therefore, close monitoring of prothrombin time is recommended, and adjustment of the anticoagulant dose may be necessary when 'Tagamet' is administered concomitantly. Interaction with phenytoin, lidocaine and theophylline has also been reported to produce adverse clinical effects.

However, a crossover study in healthy subjects receiving either 'Tagamet' 300 mg. q.i.d. or 800 mg. h.s. concomitantly with a 300 mg. b.i.d. dosage of theophylline (Theo-Dur®, Key Pharmaceuticals, Inc.), demonstrated less alteration in steady-state theophylline peak serum levels with the 800 mg. h.s. regimen, particularly in subjects aged 54 years and older. Data beyond ten days are not available. (Note: All patients receiving theophylline should be monitored appropriately, regardless of concomitant drug therapy.)

Lack of experience to date precludes recommending 'Tagamet' for use in pregnant patients, women of childbearing potential, nursing mothers or children under 16 unless anticipated benefits outweigh potential risks; generally, nursing should not be undertaken in patients taking the drug since cimetidine is secreted in human milk.

Adverse Reactions: Diarrhea, dizziness, somnolence, headache, rash. Reversible arthralgia, myalgia and exacerbation of joint symptoms in patients with preexisting arthritis have been reported. Reversible confusional states (e.g., mental confusion, agitation, psychosis, depression, anxiety, hallucinations, disorientation), predominantly in severely ill patients, have been reported. Gynecomastia and reversible impotence in patients with pathological hypersecretory disorders receiving 'Tagamet', particularly in high doses, for at least 12 months, have been reported. Reversible alopecia has been reported very rarely. Decreased white blood cell counts in 'Tagamet'-treated patients (approximately 1 per 100,000 patients), including agranulocytosis (approximately 3 per million patients), have been reported, including a few reports of recurrence on rechallenge. Most of these reports were in patients who had serious concomitant illnesses and received drugs and/or treatment known to produce neutropenia. Thrombocytopenia (approximately 3 per million patients) and a few cases of aplastic anemia have also been reported. Increased serum transaminase and creatinine, as well as rare cases of fever, interstitial nephritis, urinary retention, pancreatitis and allergic reactions, including hypersensitivity vasculitis, have been reported. Reversible adverse hepatic effects, cholestatic or mixed cholestatic-hepatocellular in nature, have been reported rarely. Because of the predominance of cholestatic features, severe parenchymal injury is considered highly unlikely. A single case of biopsy-proven periportal hepatic fibrosis in a patient receiving 'Tagamet' has been reported.

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Prefilled Syringes: 300 mg./2 ml. in single-dose prefilled disposable syringes.

Plastic Containers: 300 mg. in 50 ml. of 0.9% Sodium Chloride in single-dose plastic containers, in packages of 4 units. No preservative has been added.

ADD-Vantage®* Vials: 300 mg./2 ml. in single-dose ADD-Vantage® Vials, in packages of 25.

Exposure of the premixed product to excessive heat should be avoided. It is recommended the product be stored at controlled room temperature. Brief exposure up to 40°C does not adversely affect the premixed product.

'Tagamet' HCl (brand of cimetidine hydrochloride) injection premixed in single-dose plastic containers is manufactured for SK&F Lab Co. by Travenol Laboratories, Inc., Deerfield, IL 60015.

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BRS-TG:L73B Date of Issuance Apr. 1987

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(*continued from p 4A*)

May 26–27. **Current Concepts of Anatomic Pathology.** San Francisco. Contact: Extended Programs in Medical Education, University of California, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

NEW JERSEY

May 13–14. **Eastern Region Pathology Meeting.** Resorts International Casino Hotel, Atlantic City. Contact: Cathy Gillmer, Executive Secretary, Eastern Regional Pathology Meeting, 2 Princess Rd, Lawrenceville, NJ 08648. Tel: (609) 896-1717.

NEBRASKA

May 9–20. **Family Practice Review.** Omaha. Contact: Marge Adey or Brenda Ram, Center for Continuing Education, University of Nebraska Medical Center, 42nd and Dewey Avenue, Omaha, NE 68105. Tel: (402) 559-4152.

PENNSYLVANIA

May 23–25. **Common Clinical Challenges in the Elderly.** Hershey Hotel, Philadelphia. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

SOUTH CAROLINA

May 26–29. **14th Annual Family Medicine Update.** 30 Cat 1 Credits. Francis Marion Radison Hotel, Charleston. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

May 26–29. **AIDS, Hepatitis, and Blood Transfusion: Impact on Surgical Practice.** The Omni at Charleston Place, Charleston. Contact: Cardiothoracic Research and Educational Foundation, PO Box 33185, San Diego, CA 92103. Tel: (619) 692-9115.

VIRGINIA

May 4–6. **Regions II and III High Blood Pressure Conference.** Omni Hotel, Richmond. Contact: Paula Ciavarella or Joann Richardson, Conference Co-Chairpersons, Virginia Department of Health, Richmond, VA 23219. Tel: (804) 786-4065.

AROUND THE WORLD

CANADA

May 30–June 3. **Internal Medicine Review.** Four Seasons Hotel, Vancouver, BC. Contact: American College of Physicians, 4200 Pine Street, Philadelphia, PA 19104.

ORIENT

May 13–June 3. **Risk Management and Comparative Medicolegal Issues.** 28 Cat 1 Credits. Hong Kong, China, and Japan. Contact: International Conferences, 189 Lodge Ave, Huntington Station, NY 11746. Tel: (516) 549-0869. □

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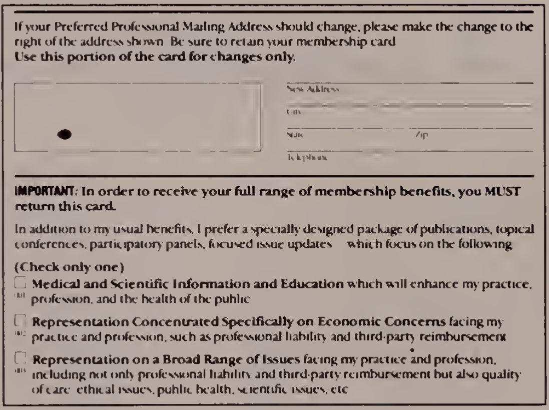

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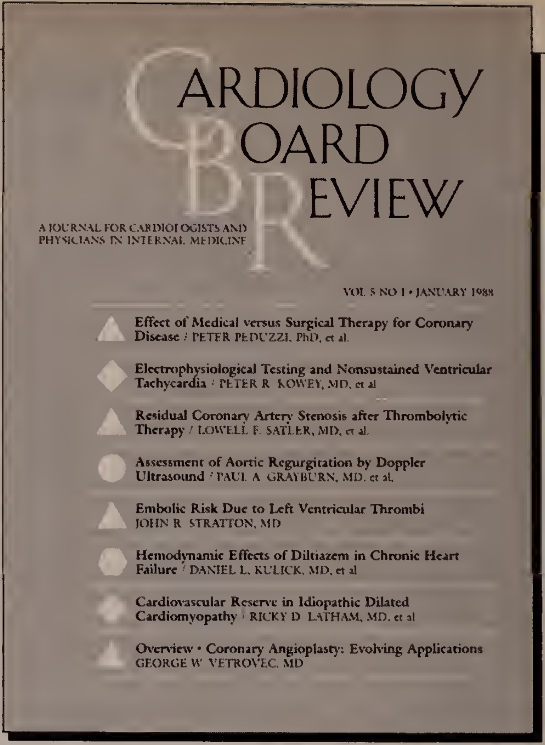

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1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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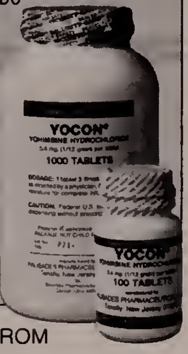

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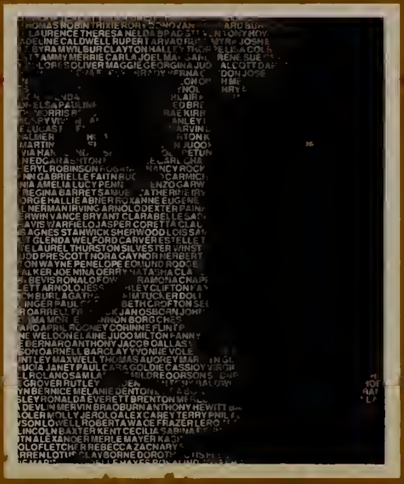

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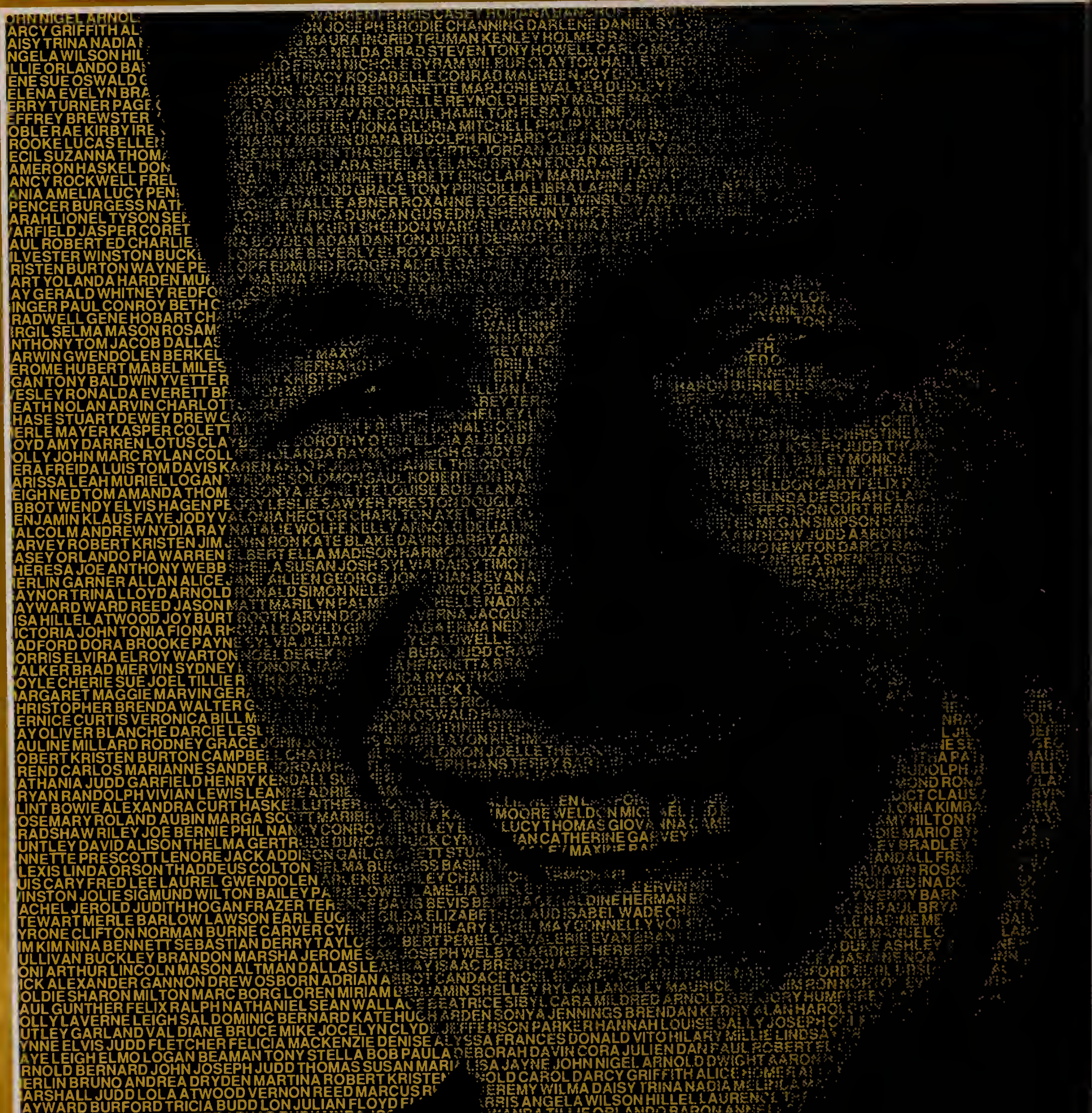
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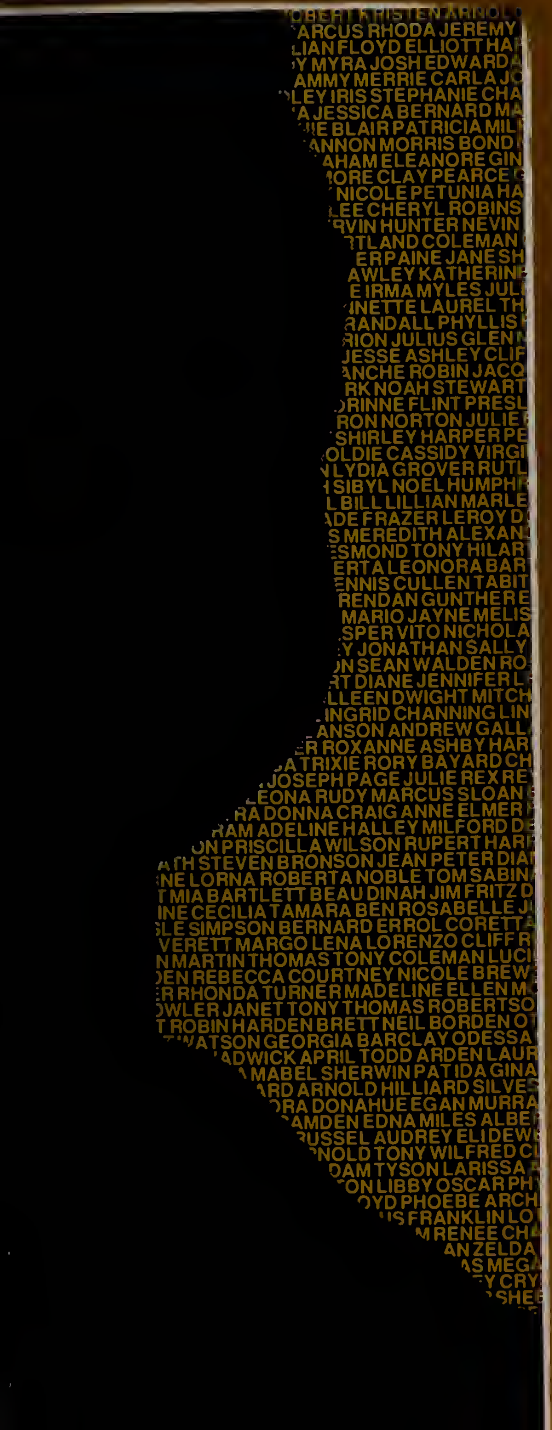

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...Virtually every responding physician rated patient satisfaction with INDERAL LA to be as good as, or better than, other beta blockers.

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree and bronchial asthma.

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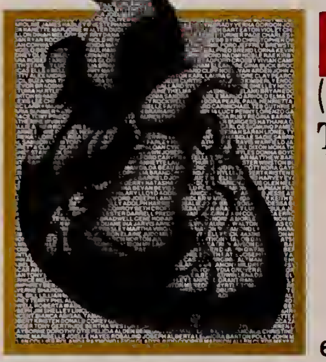

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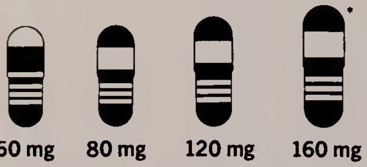

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride. INDERAL LA is available as 60 mg, 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure, and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first-degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected. Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL (propranolol HCl) is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Caution should be exercised when patients receiving a beta blocker are administered a calcium-channel-blocking drug, especially intravenous verapamil, for both agents may depress myocardial contractility or atrioventricular conduction. On rare occasions, the concomitant intravenous use of a beta blocker and verapamil has resulted in serious adverse reactions, especially in patients with severe cardiomyopathy, congestive heart failure, or recent myocardial infarction.

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol.

Ethanol slows the rate of absorption of propranolol.

Phenytoin, *phenobarbitone*, and *rifampin* accelerate propranolol clearance.

Chlorpromazine, when used concomitantly with propranolol, results in increased plasma levels of both drugs.

Antipyrine and *lidocaine* have reduced clearance when used concomitantly with propranolol.

Thyroxine may result in a lower than expected T_3 concentration when used concomitantly with propranolol.

Cimetidine decreases the hepatic metabolism of propranolol, delaying elimination and increasing blood levels.

Theophylline clearance is reduced when used concomitantly with propranolol.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS: INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type.

Central Nervous System: Light-headedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; vivid dreams; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. For immediate formulations, fatigue, lethargy, and vivid dreams appear dose related.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL Tablets to INDERAL LA Capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg-for-mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary, especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—*Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three- to seven-day intervals until optimal response is obtained. Although individual patients may respond at any dosage level, the average optimal dosage appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE—*Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimal migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximal dose, INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

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Reference:

1. Data on file, Ayerst Laboratories.

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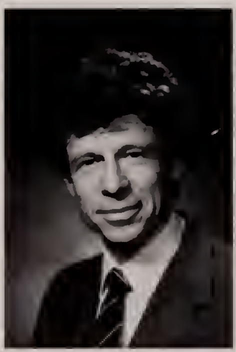
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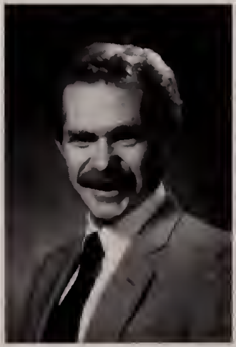
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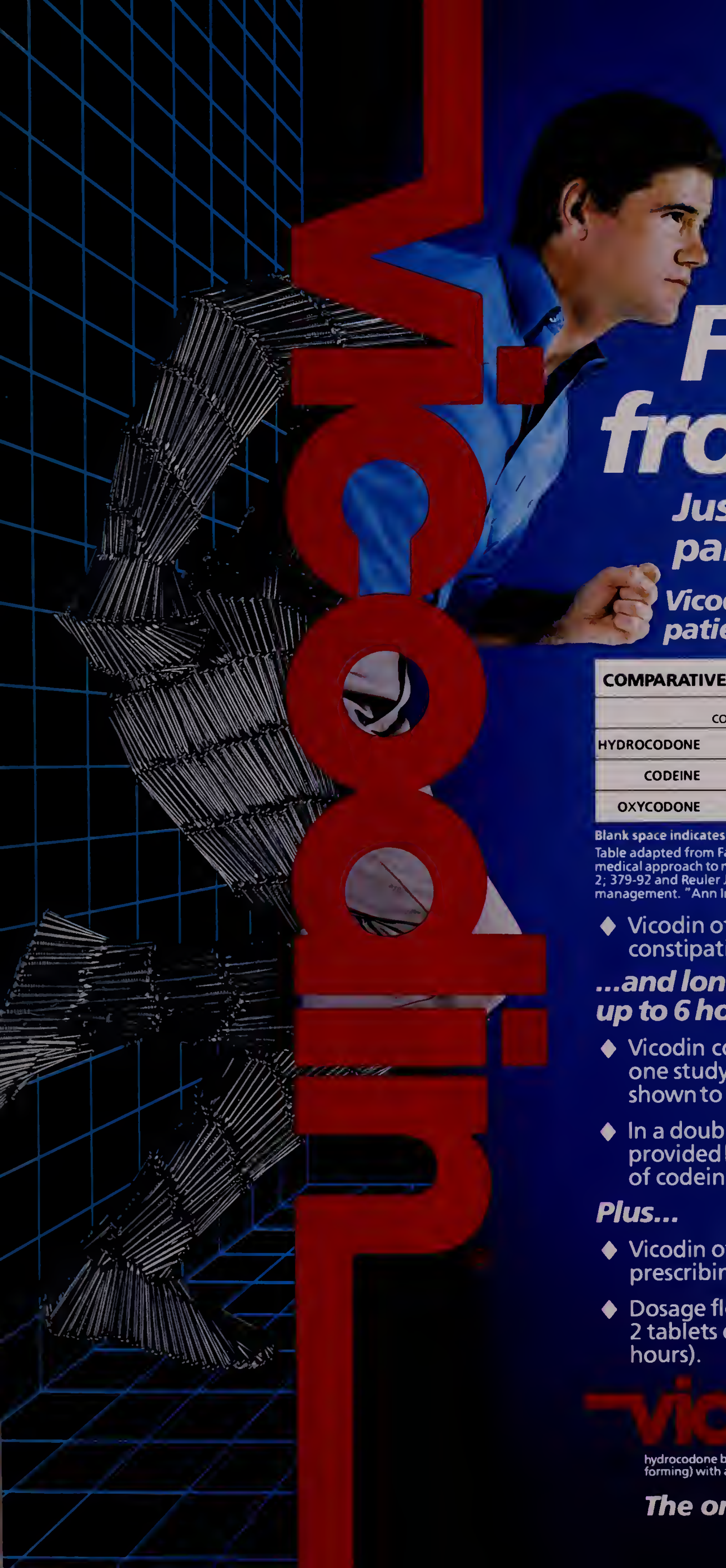

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HYDROCODONE		X			X
CODEINE	X	X	X	X	X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975, 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

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INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.
WARNINGS:
Drug Abuse and Dependence: VICODIN® is subject to the Federal Controlled Substances Act (Schedule III). Psychic dependence, physical dependence and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN should be prescribed and administered with the same caution appropriate to the use of other oral-narcotic-containing medications.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers. Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing.
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS:
Special Risk Patients: VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Information For Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
Cough Reflex: Hydrocodone suppresses the cough reflex; caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants. When combined therapy is contemplated, the dose of one or both agents should be reduced. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic Effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
Labor and Delivery: Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
Nursing Mothers: It is not known whether this drug is excreted in human milk; therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS:
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients. Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.
Respiratory Depression: (See WARNINGS.)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. However, tolerance to hydrocodone can develop with continued use, and the incidence of untoward effects is dose related.
The usual dose is one tablet every six hours as needed for pain. (If necessary, this dose may be repeated at four-hour intervals.) In cases of more severe pain, two tablets every six hours (up to eight tablets in 24 hours) may be required.

Revised, April 1982. S6BS

1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med*, 141:293-300, 1981.

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MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

JUNE 1988

AROUND THE STATE

ALBANY

June 9–10. **The Use of the Saphenous Vein In-Situ as an Arterial Bypass.** Americana Inn and Albany Medical College. *June 16.* **Family Practice—Preventive Medicine.** Turf Inn, Albany. Contact: Lynne Wechsler, Albany Medical College, Office of Continuing Medical Education, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

BOLTON LANDING ON LAKE GEORGE

June 17–19. **Ophthalmology Conference '88.** Sagamore Hotel, Bolton Landing. Contact: Diane M. Martin, Albany Medical College, Office of Continuing Medical Education, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

BRONX

June 3. **14th Ophthalmology Symposium.** 6 Cat 1 Credits. Albert Einstein College of Medicine. Contact: Danny Flood, Director, Public Relations, Albert Einstein College of Medicine/Montefiore Medical Center, Office of Continuing Education, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674.

BUFFALO

June 12–16. **11th International Convocation on Immunology.** Hyatt Regency Buffalo Hotel. Contact: Dr James F. Mohn, Director, The Ernest Witebsky Center for Immunology, 240 Sherman Hall, State University of New York at Buffalo, Buffalo, NY 14214. Tel: (716) 831-2848.

(*continued on p 13A*)

For urinary tract infection

Illustration of Bactrim power in urinary tract infection.

Bactrim Power

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- Persists

Bactrim penetrates all tissues[1] to overpower most common susceptible uropathogens including *E. coli*, *Klebsiella* species, *Enterobacter* species, *Morganella morganii*, *Proteus (in vitro)* year after year.[2] B.i.d. dosing, easy transition from IV to oral, and economy help keep successful therapy within your power. Especially when you remember to protect your prescribing decision by specifying D.A.W.

Please note that *in vitro* data may not correlate with clinical experience. Bactrim is contraindicated in infants less than two months of age, in pregnancy at term, during lactation, and in documented megaloblastic anemia due to folate deficiency. Maintain adequate fluid intake.

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Bactrim™ DS

(160 mg trimethoprim and 800 mg sulfamethoxazole/Roche)

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Please see references and summary of product information on following page.

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References: 1. Rubin RH, Swartz MN *N Engl J Med 302*:426-432, Aug 21, 1980. 2. BAC-DATA Medical Information Systems, Inc , Volume I, 1986

Bactrim™

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

CONTRAINDICATIONS: Hypersensitivity to trimethoprim or sulfonamides, documented megaloblastic anemia due to folate deficiency; pregnancy at term and during the nursing period, infants less than two months of age

WARNINGS: FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS.

BACTRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. Clinical signs, such as rash, sore throat, fever, arthralgia, cough, shortness of breath, pallor, purpura or jaundice, may be early indications of serious reactions In rare instances a skin rash may be followed by more severe reactions, such as Stevens-Johnson syndrome, toxic epidermal necrolysis, hepatic necrosis or serious blood disorder Perform complete blood counts frequently

BACTRIM SHOULD NOT BE USED IN THE TREATMENT OF STREPTOCOCCAL PHARYNGITIS Clinical studies show that patients with group A ß-hemolytic streptococcal tonsillopharyngitis have a greater incidence of bacteriologic failure when treated with Bactrim than with penicillin.

PRECAUTIONS: *General* Give with caution to patients with impaired renal or hepatic function, possible folate deficiency (e g , elderly, chronic alcoholics, patients on anticonvulsants, with malabsorption syndrome, or in malnutrition states) and severe allergies or bronchial asthma In glucose-6-phosphate dehydrogenase deficient individuals, hemolysis may occur, frequently dose-related

Use in the Elderly May be increased risk of severe adverse reactions in elderly, particularly with complicating conditions, *e g* , impaired kidney and/or liver function, concomitant use of other drugs Severe skin reactions, generalized bone marrow suppression (see WARNINGS and ADVERSE REACTIONS) or a specific decrease in platelets (with or without purpura) are most frequently reported severe adverse reactions in elderly In those concurrently receiving certain diuretics, primarily thiazides, increased incidence of thrombocytopenia with purpura reported Make appropriate dosage adjustments for patients with impaired kidney function (see DOSAGE AND ADMINISTRATION)

Use in the Treatment of Pneumocystis Carinii Pneumonia in Patients with Acquired Immunodeficiency Syndrome (AIDS). AIDS patients may not tolerate or respond to Bactrim in same manner as non-AIDS patients Incidence of side effects, particularly rash, fever, leukopenia, elevated aminotransferase (transaminase) values, with Bactrim in AIDS patients treated for *Pneumocystis carinii* pneumonia reported to be greatly increased compared with incidence normally associated with Bactrim in non-AIDS patients

Information for Patients Instruct patients to maintain adequate fluid intake to prevent crystalluria and stone formation

Laboratory Tests Perform complete blood counts frequently; if a significant reduction in the count of any formed blood element is noted, discontinue Bactrim Perform urinalyses with careful microscopic examination and renal function tests during therapy, particularly for patients with impaired renal function

Drug Interactions In elderly patients concurrently receiving certain diuretics, primarily thiazides, an increased incidence of thrombocytopenia with purpura has been reported Bactrim may prolong the prothrombin time in patients who are receiving the anticoagulant warfarin Keep this in mind when Bactrim is given to patients already on anticoagulant therapy and reassess coagulation time Bactrim may inhibit the hepatic metabolism of phenytoin Given at a common clinical dosage, it increased the phenytoin half-life by 39% and decreased the phenytoin metabolic clearance rate by 27%. When giving these drugs concurrently, be alert for possible excessive phenytoin effect Sulfonamides can displace methotrexate from plasma protein binding sites, thus increasing free methotrexate concentrations

Drug/Laboratory Test Interactions Bactrim, specifically the trimethoprim component, can interfere with a serum methotrexate assay as determined by the competitive binding protein technique (CBPA) when a bacterial dihydrofolate reductase is used as the binding protein No interference occurs if methotrexate is measured by a radioimmunoassay (RIA). The presence of trimethoprim and sulfamethoxazole may also interfere with the Jaffe alkaline picrate reaction assay for creatinine, resulting in overestimations of about 10% in the range of normal values

Carcinogenesis, Mutagenesis, Impairment of Fertility: Carcinogenesis. Long-term studies in animals to evaluate carcinogenic potential not conducted with Bactrim *Mutagenesis* Bacterial mutagenic studies not performed with sulfamethoxazole and trimethoprim in combination. Trimethoprim demonstrated to be nonmutagenic in the Ames assay No chromosomal damage observed in human leukocytes *in vitro* with sulfamethoxazole and trimethoprim alone or in combination, concentrations used exceeded blood levels of these compounds following therapy with Bactrim Observations of leukocytes obtained from patients treated with Bactrim revealed no chromosomal abnormalities *Impairment of Fertility* No adverse effects on fertility or general reproductive performance observed in rats given oral dosages as high as 70 mg/kg/day trimethoprim plus 350 mg/kg/day sulfamethoxazole.

Pregnancy Teratogenic Effects Pregnancy Category C Trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefit justifies potential risk to fetus Nonteratogenic Effects. See CONTRAINDICATIONS section

Nursing Mothers See CONTRAINDICATIONS section

Pediatric Use Not recommended for infants under two months (see INDICATIONS and CONTRAINDICATIONS sections)

ADVERSE REACTIONS: Most common are gastrointestinal disturbances (nausea, vomiting, anorexia) and allergic skin reactions (such as rash and urticaria) **FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS (SEE WARNINGS SECTION)** *Hematologic.* Agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, neutropenia, hemolytic anemia, megaloblastic anemia, hypoprothrombinemia, methemoglobinemia, eosinophilia *Allergic Reactions* Stevens-Johnson syndrome, toxic epidermal necrolysis, anaphylaxis, allergic myocarditis, erythema multiforme, exfoliative dermatitis, angioedema, drug fever, chills, Henoch-Schoenlein purpura, serum sickness-like syndrome, generalized allergic reactions, generalized skin eruptions, photosensitivity, conjunctival and scleral injection, pruritus, urticaria and rash Periarteritis nodosa and systemic lupus erythematosus have been reported *Gastrointestinal* Hepatitis (including cholestatic jaundice and hepatic necrosis), elevation of serum transaminase and bilirubin, pseudomembranous enterocolitis, pancreatitis, stomatitis, glossitis, nausea, emesis, abdominal pain, diarrhea, anorexia. *Genitourinary* Renal failure, interstitial nephritis, BUN and serum creatinine elevation, toxic nephrosis with oliguria and anuria, crystalluria *Neurologic* Aseptic meningitis, convulsions, peripheral neuritis, ataxia, vertigo, tinnitus, headache *Psychiatric* Hallucinations, depression, apathy, nervousness *Endocrine.* Sulfonamides bear certain chemical similarities to some goitrogens, diuretics (acetazolamide and the thiazides) and oral hypoglycemic agents, cross-sensitivity may exist Diuresis and hypoglycemia have occurred rarely in patients receiving sulfonamides *Respiratory* Pulmonary infiltrates *Musculoskeletal* Arthralgia, myalgia *Miscellaneous* Weakness fatigue insomnia

DOSAGE AND ADMINISTRATION: Not recommended for use in infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN *Usual adult dosage* for urinary tract infections is one DS tablet, two tablets or four teaspoonfuls (20 ml) *b i d* for 10 to 14 days Use identical daily dosage for 5 days for shigellosis *Recommended dosage for children* with urinary tract infections or acute otitis media is 8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses every 12 hours for 10 days Use identical daily dosage for 5 days for shigellosis *Renal Impaired* Creatinine clearance above 30 ml/min, give usual dosage, 15-30 ml/min give one-half the usual regimen, below 15 ml/min, use not recommended

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS Usual adult dosage is one DS tablet, two tablets or four teasp (20 ml) *b i d* for 14 days

PNEUMOCYSTIS CARINII PNEUMONIA Recommended dosage is 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days See complete product information for suggested children's dosage table

HOW SUPPLIED: *DS (double strength) Tablets* (160 mg trimethoprim and 800 mg sulfamethoxazole)—bottles of 100, 250 and 500, Tel-E-Dose* packages of 100, Prescription Paks of 20 *Tablets* (80 mg trimethoprim and 400 mg sulfamethoxazole)—bottles of 100 and 500, Tel-E-Dose* packages of 100; Prescription Paks of 40 *Pediatric Suspension* (40 mg trimethoprim and 200 mg sulfamethoxazole per teasp)—bottles of 100 ml and 16 oz (1 pint) *Suspension* (40 mg trimethoprim and 200 mg sulfamethoxazole per teasp)—bottles of 16 oz (1 pint)

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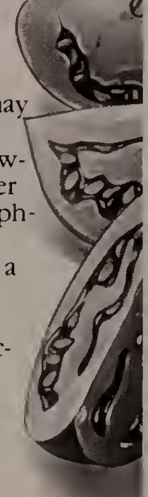


There is evidence that diet and cancer are related. Some foods may promote cancer, while others may protect you from it.

Foods related to lowering the risk of cancer of the larynx and esophagus all have high amounts of carotene, a form of Vitamin A which is in cantaloupes, peaches, broccoli, spinach, all dark green leafy vegetables, sweet potatoes, carrots, pumpkin, winter squash, and tomatoes, citrus fruits and brussels sprouts.

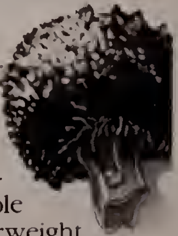
Foods that may help reduce the risk of gastrointestinal and respiratory tract cancer are cabbage, broccoli, brussels sprouts, kohlrabi, cauliflower.

Fruits, vegetables and whole-grain cereals such as oatmeal, bran and wheat may help lower the risk of colorectal cancer.

Foods high in fats, salt- or nitrite-cured foods such as ham, and fish and types of sausages smoked by traditional methods should be eaten in moderation.

Be moderate in consumption of alcohol also.

A good rule of thumb is cut down on fat and don't be fat. Weight reduction may lower cancer risk. Our 12-year study of nearly a million Americans uncovered high cancer risks particularly among people 40% or more overweight.



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Slow-K®

potassium chloride
slow-release tablets
8 mEq (600 mg)

It means "dependability" in almost any language

*Based on worldwide sales data on file, CIBA Pharmaceutical Company.
Capsule or tablet slow-release potassium chloride preparations should be reserved for patients who cannot tolerate, refuse to take, or have compliance problems with liquid or effervescent potassium preparations because of reports of intestinal and gastric ulceration and bleeding with slow-release KCl preparations.
Before prescribing, please consult Brief Prescribing Information on next page.

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☐ **It's safe**—unsurpassed by any other KCl tablet or capsule[2]*

☐ **It's acceptable vs liquids**—greater palatability, fewer GI complaints, lower incidence of nausea[2]

☐ **It's comparable to 10 mEq**—in low-dosage supplementation[3]†

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Slow-K®
potassium chloride
slow-release tablets 8 mEq (600 mg)

For patients who can't or won't tolerate liquid KCl.

*The most common adverse reactions to potassium salts are gastrointestinal side effects.

†Pooled mean serum potassium following oral administration of 30 mEq K-Tab compared to 24 mEq Slow-K in diuretic-treated hypertensives (n = 20) over 8 weeks.

C I B A

References: 1. Data on file, CIBA Pharmaceutical Company 2. Skoutakis VA, Acchiardo SR, Wojciechowski NJ, et al: Liquid and solid potassium chloride Bioavailability and safety. *Pharmacotherapy* 1980;4(6):392-397. 3. Skoutakis VA, Carter CA, Acchiardo SR: Therapeutic assessment of Slow-K and K-Tab potassium chloride formulations in hypertensive patients treated with thiazide diuretics. *Drug Intell Clin Pharm* 1987;21 436-440

Slow-K®
potassium chloride USP
Slow-Release Tablets
8 mEq (600 mg)

BRIEF SUMMARY (FOR FULL PRESCRIBING INFORMATION SEE PACKAGE INSERT)

INDICATIONS AND USAGE

BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS.

1 For therapeutic use in patients with hypokalemia with or without metabolic alkalosis; in digitalis intoxication and in patients with hypokalemic familial periodic paralysis

2 For prevention of potassium depletion when the dietary intake of potassium is inadequate in the following conditions: patients receiving digitalis and diuretics for congestive heart failure; hepatic cirrhosis with ascites; states of aldosterone excess with normal renal function; potassium-losing nephropathy; and certain diarrheal states.

3 The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases In more severe cases supplementation with potassium salts may be indicated.

CONTRAINDICATIONS

Potassium supplements are contraindicated in patients with hyperkalemia, since a further increase in serum potassium concentration in such patients can produce cardiac arrest. Hyperkalemia may complicate any of the following conditions chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e g , spironolactone, triamterene) (see OVERDOSAGE)

All solid dosage forms of potassium supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract In these instances, potassium supplementation should be with a liquid preparation. Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to an enlarged left atrium.

WARNINGS

Hyperkalemia (See OVERDOSAGE)

In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally Potentially fatal hyperkalemia can develop rapidly and be asymptomatic

The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment.

Interaction With Potassium-Sparing Diuretics

Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e.g , spironolactone or triamterene), since the simultaneous administration of these agents can produce severe hyperkalemia

Gastrointestinal Lesions

Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths. These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage, or perforation. Slow-K is a wax-matrix tablet formulated to provide a controlled rate of release of potassium chloride and thus to minimize the possibility of a high local concentration of potassium ion near the bowel wall. While the reported frequency of small-bowel lesions is much less with wax-matrix tablets (less than one per 100,000 patient-years) than with enteric-coated potassium chloride tablets (40-50 per 100,000 patient-years) cases associated with wax-matrix tablets have been reported both in foreign countries and in the United States. In addition, perhaps because the wax-matrix preparations are not enteric-coated and release potassium in the stomach, there have been reports of upper gastrointestinal bleeding associated with these products. The total number of gastrointestinal lesions remains approximately one per 100,000 patient-years. Slow-K should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs.

Metabolic Acidosis

Hypokalemia in patients with metabolic *acidosis* should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, or potassium acetate

PRECAUTIONS

General:

The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis *per se* can produce hypokalemia in the absence of a deficit in total body potassium, while acute acidosis *per* se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium.

Information for Patients

Physicians should consider reminding the patient of the following:

To take each dose without crushing, chewing, or sucking the tablets.

To take this medicine only as directed. This is especially important if the patient is also taking both diuretics and digitalis preparations.

To check with the physician if there is trouble swallowing tablets or if the tablets seem to stick in the throat.

To check with the doctor at once if tarry stools or other evidence of gastrointestinal bleeding is noticed

Laboratory Tests

Regular serum potassium determinations are recommended. In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis

Drug Interactions

Potassium-sparing diuretics: see WARNINGS

Carcinogenesis, Mutagenesis, Impairment of Fertility

Long-term carcinogenicity studies in animals have not been performed.

Pregnancy Category C

Animal reproduction studies have not been conducted with Slow-K. It is also not known whether Slow-K can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Slow-K should be given to a pregnant woman only if clearly needed.

Nursing Mothers

The normal potassium ion content of human milk is about 13 mEq/L. It is not known if Slow-K has an effect on this content Caution should be exercised when Slow-K is administered to a nursing woman.

Pediatric Use

Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

One of the most severe adverse effects is hyperkalemia (see CONTRAINDICATIONS, WARNINGS, and OVERDOSAGE). There also have been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see CONTRAINDICATIONS and WARNINGS); other factors known to be associated with such conditions were present in many of these patients.

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose.

Skin rash has been reported rarely.

OVERDOSAGE

The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia. However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see CONTRAINDICATIONS and WARNINGS). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration (6.5-8 0 mEq/L) and characteristic electrocardiographic changes (peaking of T waves, loss of P wave, depression of S-T segment, and prolongation of the Q-T interval). Late manifestations include muscle paralysis and cardiovascular collapse from cardiac arrest (9-12 mEq/L).

Treatment measures for hyperkalemia include the following: (1) elimination of foods and medications containing potassium and of potassium-sparing diuretics; (2) intravenous administration of 300-500 ml/hr of 10% dextrose solution containing 10-20 units of insulin per 1,000 ml; (3) correction of acidosis, if present, with intravenous sodium bicarbonate; (4) use of exchange resins, hemodialysis, or peritoneal dialysis.

In treating hyperkalemia in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity

DOSAGE AND ADMINISTRATION

The usual dietary intake of potassium by the average adult is 40-80 mEq per day Potassium depletion sufficient to cause hypokalemia usually requires the loss of 200 or more mEq of potassium from the total body store. Dosage must be adjusted to the individual needs of each patient but is typically in the range of 20 mEq per day for the prevention of hypokalemia to 40-100 mEq or more per day for the treatment of potassium depletion. Large numbers of tablets should be given in divided doses.

Note: Slow-K slow-release tablets must be swallowed whole and never crushed, chewed, or sucked

HOW SUPPLIED

Tablets—600 mg of potassium chloride (equivalent to 8 mEq) round, buff colored, sugar-coated (imprinted Slow-K)

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12 Bottles—100 tablets each	NDC 0083-0165-65
Accu-Pak® Unit Dose (Blister pack)	
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Dispense in tight, light-resistant container (USP).

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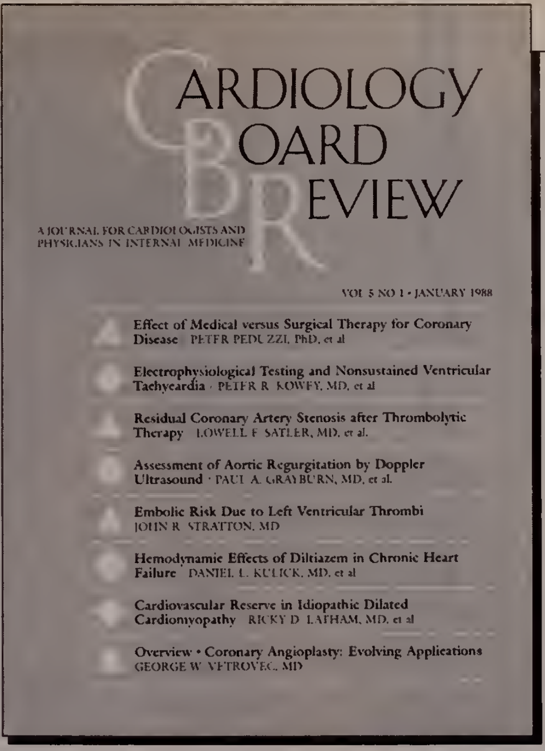

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A JOURNAL FOR CARDIOLOGISTS AND PHYSICIANS IN INTERNAL MEDICINE

VOL 5 NO 1 • JANUARY 1988

Effect of Medical versus Surgical Therapy for Coronary Disease · PETER PEDUZZI, PhD, et al

Electrophysiological Testing and Nonsustained Ventricular Tachycardia · PETER R KOWEY, MD, et al

Residual Coronary Artery Stenosis after Thrombolytic Therapy · LOWELL F SATLER, MD, et al.

Assessment of Aortic Regurgitation by Doppler Ultrasound · PAUL A. GRAYBURN, MD, et al.

Embolic Risk Due to Left Ventricular Thrombi
JOHN R STRATTON, MD

Hemodynamic Effects of Diltiazem in Chronic Heart Failure · DANIEL L. KULICK, MD, et al

Cardiovascular Reserve in Idiopathic Dilated Cardiomyopathy · RICKY D LATHAM, MD, et al

Overview • Coronary Angioplasty: Evolving Applications
GEORGE W VETROVEC, MD

*Journals reviewed include: *Circulation, American Heart Journal, Journal of the American College of Cardiology, British Heart Journal, Chest, The American Journal of Cardiology, The New England Journal of Medicine, Annals of Internal Medicine, American Journal of Medicine*, and *The Journal of the American Medical Association.*

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diltiazem HCl/Marion

60 mg
GREATER DOSAGE FLEXIBILITY
90 mg/120 mg

- **A remarkable safety profile**[1-6]
  The low incidence of side effects with Cardizem allows patients to feel better.
- **Protection against angina attacks**[1,5,7-9]
  The predictable efficacy of Cardizem in stable exertional* and vasospastic angina allows patients to do more.
- **A decrease in myocardial oxygen demand**
  Resulting from a lowered heart rate-blood pressure product.[5]
- **Compatible with other antianginals**[6]†
- **Safe in angina with coexisting hypertension, COPD, asthma, or PVD**[1,3,5,6]

*CARDIZEM® (diltiazem HCl) is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

†See Warnings and Precautions.

Please see brief summary of prescribing information on the next page. 0778A8

CARDIZEM® ANTIANGINAL PROTECTION PLUS SAFETY

diltiazem HCl/Marion

Usual maintenance dosage range: 180-360 mg/day

Rx

Cardizem®
(diltiazem HCl)

☐ 60 mg ☐ 90 mg
☐ 120 mg

Sig: tid

BRIEF SUMMARY
Professional Use Information

CARDIZEM®
(diltiazem HCl)
30 mg, 60 mg, 90 mg, and 120 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, (3) patients with hypotension (less than 90 mm Hg systolic), and (4) patients who have demonstrated hypersensitivity to the drug.

WARNINGS

1. **Cardiac Conduction.** *CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.*
2. **Congestive Heart Failure.** *Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.*
3. **Hypotension.** *Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.*
4. **Acute Hepatic Injury.** *In rare instances, significant elevations in enzymes such as alkaline phosphatase, LDH, SGDT, SGPT, and other phenomena consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)*

PRECAUTIONS

General. *CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.*

Dermatological events (see ADVERSE REACTIONS section) may be transient and may disappear despite continued use of CARDIZEM. However, skin eruptions progressing to erythema multiforme and/or exfoliative dermatitis have also been infrequently reported. Should a dermatologic reaction persist, the drug should be discontinued.

Drug Interaction. *Due to the potential for additive effects, caution and careful titration are warranted in patients receiving CARDIZEM concomitantly with any agents known to affect cardiac contractility and/or conduction. (See WARNINGS.)*

Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

As with all drugs, care should be exercised when treating patients with multiple medications. CARDIZEM undergoes biotransformation by cytochrome P-450 mixed function oxidase. Coadministration of CARDIZEM with other agents which follow the same route of biotransformation may result in the competitive inhibition of metabolism. Dosages of similarly metabolized drugs, particularly those of low therapeutic ratio or in patients with renal and/or hepatic impairment, may require adjustment when starting or stopping concomitantly administered CARDIZEM to maintain optimum therapeutic blood levels.

Beta-blockers: *Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities.*

Administration of CARDIZEM (diltiazem hydrochloride) concomitantly with propranolol in five normal volunteers resulted in increased propranolol levels in all subjects and bioavailability of propranolol was increased approximately 50%. If combination therapy is initiated or withdrawn in conjunction with propranolol, an adjustment in the propranolol dose may be warranted. (See WARNINGS.)

Cimetidine: *A study in six healthy volunteers has shown a significant increase in peak diltiazem plasma levels (58%) and area-under-the-curve (53%) after a one-week course of cimetidine at 1,200 mg per day and diltiazem 60 mg per day. Ranitidine produced smaller, nonsignificant increases. The effect may be mediated by cimetidine's known inhibition of hepatic cytochrome P-450, the enzyme system probably responsible for the first-pass metabolism of diltiazem. Patients currently receiving diltiazem therapy should be carefully monitored for a change in pharmacological effect when initiating and discontinuing therapy with cimetidine. An adjustment in the diltiazem dose may be warranted.*

Digitalis: *Administration of CARDIZEM with digoxin in 24 healthy male subjects increased plasma digoxin concentrations approximately 20%. Another investigator found no increase in digoxin levels in 12 patients with coronary artery disease. Since there have been conflicting results regarding the effect of digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing CARDIZEM therapy to avoid possible over- or under-digitalization. (See WARNINGS.)*

Carcinogenesis, Mutagenesis, Impairment of Fertility. *A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.*

Pregnancy. *Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/ postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.*

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. *Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.*

Pediatric Use. *Safety and effectiveness in children have not been established.*

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	*Angina, arrhythmia, AV block (first degree), AV block (second or third degree—see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.*
Nervous System:	*Amnesia, depression, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.*
Gastrointestinal:	*Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGDT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.*
Dermatologic:	*Petechiae, pruritus, photosensitivity, urticaria.*
Other:	*Amblyopia, CPK elevation, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.*

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established. *Issued 6/87*

See complete Professional Use Information before prescribing.

References: 1. Schroeder JS: Mod Med 1982;50(Sept):94-116. **2.** Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39. **3.** O'Rourke RA: Am J Cardiol 1985;56:34H-40H. **4.** McCall D, Walsh RA, Frohlich ED, et al: Curr Probl Cardiol 1985;10(8):6-80. **5.** Frishman WH, Chorlap S, Goldberger J, et al: Am J Cardiol 1985;56:41H-46H. **6.** Shapiro W: Consultant 1984;24(Dec):150-159. **7.** O'Hara MJ, Khurmi NS, Bowles MJ, et al: Am J Cardiol 1984;54:477-481. **8.** Strauss WE, McIntyre KM, Parisi AF, et al: Am J Cardiol 1982; 49:560-566. **9.** Feldman RL, Pepine CJ, Whittle J, et al: Am J Cardiol 1982;49:554-559.

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(*continued from p 4A*)

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MANHATTAN

June 4. **Vitrectomy in Anterior and Posterior Segment Surgery.** *June 11.* **Workshop: Diagnosis and Management of Patients with Oculo-Plastic Problems.** Manhattan Eye, Ear & Throat Hospital, 210 East 64th St, New York, NY 10021. Tel: (212) 605-3762.

June 13–16. **Biological Membranes in Cancer Cells.** Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

June 13–17. **New York Anesthesiology Review 1988.** 34 Cat 1 Credits. Waldorf-Astoria Hotel. Contact: Danny Flood, Director, Public Relations, Albert Einstein College of Medicine/Montefiore Medical Center, Office of Continuing Medical Education, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674.

June 17–19. **Acupuncture and Electrotherapeutics in Clinical Practice.** 25 Cat 1 Credits; accredited by New York State Boards for Medicine and Dentistry for 25 credit hours toward 300-hour Acupuncture Certificate requirement. Contact: Y. Omura, MD, 800 Riverside Dr (8-I), New York, NY 10032. Tel: (212) 781-6262.

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June 9–12. **17th Annual Scientific Assembly of the American College of Emergency Physicians/State Chapter of California, Inc.** Mission Hills Resort Hotel, Rancho Mirage, Contact: CAL/ACEP, 50 N Sepulveda Blvd, #12–14, Manhattan Beach, CA 90266. Tel: (213) 374-4039.

June 25–26. **Tenth Annual Poison Control Center Symposium on Toxicology and Poisoning.** 14 Cat 1 Credits. San

(*continued on p 24A*)

YOCON®
YOHIMBINE HCI

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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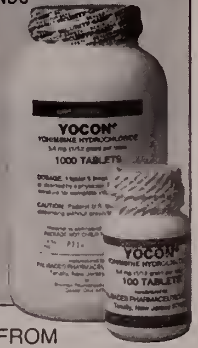

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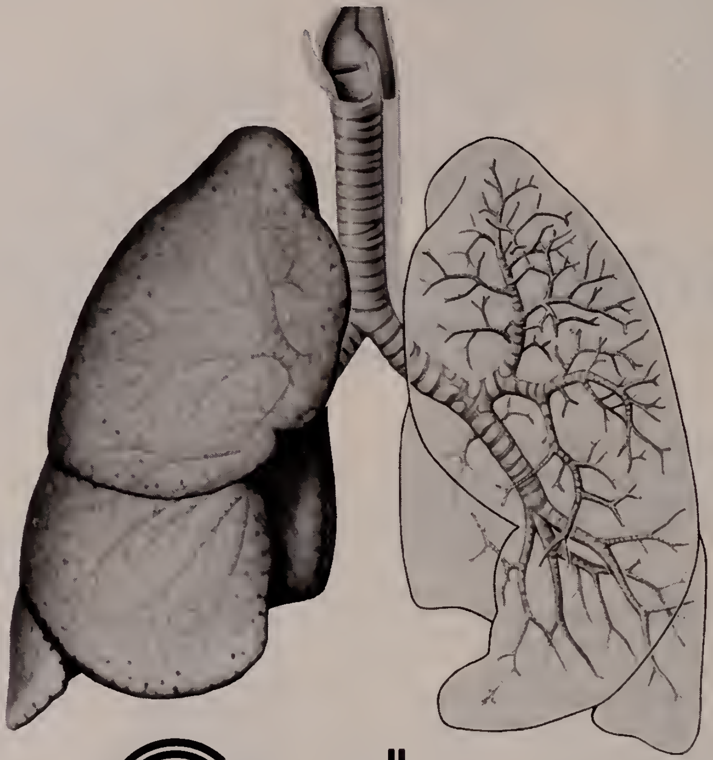

Ceclor®
cefaclor

250-mg Pulvules® t.i.d.
offers effectiveness against the major causes of bacterial bronchitis

Haemophilus influenzae and *Streptococcus pneumoniae*
(ampicillin-susceptible and ampicillin-resistant)

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

Ceclor® (cefaclor)

Summary. Consult the package literature for prescribing information.

Indication: Lower respiratory infections, including pneumonia, caused by *Streptococcus pneumoniae, Haemophilus influenzae,* and *Streptococcus pyogenes* (group A β-hemolytic streptococci).

Contraindication:
Known allergy to cephalosporins.

Warnings:
CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever): 1.5%; usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%; and, rarely, thrombocytopenia.

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly). [061787L]

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COMMENTARIES

Human immunodeficiency virus antibody testing: Time for clinicians to use it

In 1985 the enzyme-linked immunosorbent assay (ELISA) to detect antibody to the human immunodeficiency virus (HIV) was licensed by the Food and Drug Administration. Used in conjunction with appropriate confirmatory tests such as the Western blot, it was now possible to identify individuals who had been infected with this virus. The use of these tests was immediately and universally adopted by blood banks in this country. However, their use in the clinical setting was limited by a number of unanswered questions raised by clinicians, advocacy groups, and public health officials. These issues are nearing resolution, and it is now time to place HIV testing in wider clinical use.

There were four critical issues that impeded the wider use of HIV antibody testing. First, effective pre- and post-test counseling programs had yet to be developed and implemented. The accuracy of HIV testing in various populations needed to be defined. The meaning (clinical implications) of a confirmed positive test needed to be completely understood. Finally, no clear therapeutic options were available to clinicians caring for the seropositive patient who had not yet developed clinical acquired immunodeficiency syndrome (AIDS). Our clearer understanding of these issues is now a compelling reason for using HIV testing on a more active basis.

The need for effective counseling in conjunction with HIV testing has now been recognized.[1] Effective counseling programs, including both pre- and post-test counseling, have now been developed and implemented around the country. These programs have been instituted in diverse settings including hospitals, family planning clinics, methadone maintenance clinics, anonymous testing sites, and sexually transmitted disease clinics.[2]

The greatest impediment to the development of counseling programs is the lack of financial resources and an inadequate pool of trained counselors. This was demonstrated most clearly in Illinois when the introduction of required premarital HIV testing overwhelmed the few established public programs.[3] The Illinois experience is also instructive in reinforcing the concept of concentrating testing and counseling resources on high-risk groups. Diffusing scant resources to large populations delays their use for those populations that need it most.

The accuracy of HIV testing in various populations has been clarified in the past two years. Controversy around the issue of false-positives has really surrounded the use of the ELISA, an initial screening exam.[4] Under standard testing protocols all positive ELISAs are repeated, then confirmed by a Western blot (a far more specific if more costly test) prior to the patient's being notified that he or she is antibody positive. A positive confirmatory test is considered evidence of HIV infection. The sensitivity of the currently licensed ELISA tests is 99% or greater.[5] Given this performance, the probability of a false-negative test result is remote, except during that period prior to the development of antibodies. The specificity of a repeatedly reactive ELISA is approximately 99%.[5] The use of a supplemental test such as the Western blot markedly increases this specificity. Under ideal conditions, the probability that a testing sequence will result in a false positive in a population with a low rate of infection ranges from less than one in 100,000 to five in 100,000.[5] While some concern has been expressed about the rate of false positives in such low risk populations, it is important to recognize that in higher risk populations (where the seroprevalence of HIV rises above 5%), a positive test sequence should be considered exactly that, positive for HIV antibody.

The clinical implications of a confirmed positive antibody test have been clarified during the last 24 months by several studies. A confirmed positive test means that an individual is infected, potentially infectious, and has a high probability of developing symptomatic illness within seven years of infection. In adults, the best data have been generated from the San Francisco City Clinic cohort study, comprising a group of homosexual and bisexual men who were enrolled in a hepatitis B virus study in the late 1970s. Of 155 men with long term HIV infection (approximately 88 months), 36% have progressed to AIDS, while more than 40% had other signs or symptoms of infection; only 20% have remained completely asymptomatic.[6] These epidemiologic observations have been supported by other studies which have noted a clear and progressive decline in T-helper cells in 90% of individuals after at least three years of HIV infection.[7] Hence it appears that HIV infection is associated with an inexorable decline in cell-mediated immunity.

One of the initial arguments against HIV testing was the lack of therapeutic options for the seropositive patient. Clinical developments have now progressed to the point that practitioners can intervene to avoid some of the complications of HIV-induced immunosuppression. The clinical use of T4 (helper) lymphocyte enumeration plays an important role in this evaluation. As the number of T4-cells decreases, one's chance of developing one of the opportunistic infections increases. For example, it appears that when T cells decrease below a level of $200/\text{mm}^3$, one is at increased risk of developing *Pneumocystis carinii* pneumonia (PCP).[8] Several studies have demonstrated the effectiveness of various regimens in preventing PCP.[9,10] At least one additional study has demonstrated its use in asymptomatic seropositive individuals with T4 cell depletion.[11] The approach to patients who are tuberculin positive will also vary depending on the patient's HIV status. For example, the American Thoracic Society has recommended that isoniazid (INH) prophylactic therapy should be instituted in patients who are seropositive and have positive tuberculin skin tests.[12] Tuberculosis is a serious problem in many patients with HIV infection. Although most patients with severe HIV-induced immunosuppression are anergic, many are not. Application of tuberculin skin tests prior to the development of anergy can allow the clinician to determine whether to institute INH prophylaxis in hosts that may undergo further depression of cellular immunity. An additional clinical indication is based on recently completed studies in New York State which have shown a rate of HIV seropositivity in newborns of 1.5 to 2.5%.[13,14] In light of these data, the Centers for Disease Control (CDC) has suggested that women living in communities where there is known high prevalence of HIV infection be offered information and education about AIDS as well as testing. Finally, and most critically, even though all individuals should examine and alter their own personal behavior, an awareness of serostatus serves as an additional incentive to protect loved ones.

In New York State, the state with the highest incidence of AIDS, utilization of HIV testing is among the lowest of any state.[15] Clearly testing can be used to prevent further spread, to anticipate clinical problems in specific patients, as well as to help women make informed decisions about pregnancy. All testing should continue to be provided in a manner that ensures confidentiality. As physicians we need to understand the full implications of HIV testing so that we can adequately counsel our patients. Finally, we must reject all calls for mandatory (or routine) testing of populations. In an era of limited resources HIV testing should be done for clinical indications, not to pursue ideological goals. Nonetheless, it is now clear that all clinicians must become comfortable in nonjudgmentally inquiring about both a patient's drug and sexual history in order to determine whether they might benefit from HIV testing. The application of broader, selective testing will contribute greatly to patient survival as well as to preventing the spread of HIV disease.

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The new horizon: Programmatic responses to the HIV epidemic

Although the current epidemic of human immunodeficiency virus (HIV) infection has been recognized for at least seven years, in many respects we are just beginning to appreciate the sobering consequences of its impact. There is hardly an analyst familiar with the demographics of HIV infection who has not predicted substantial outlays for the medical care of the many infected individuals who will become symptomatic within the next decade. Researchers persist in their attempts to develop effective medical treatments, but even the most optimistic of them believe that there is no cure or vaccine on the immediate horizon. It is clear that many of our citizens will suffer prolonged, expensive, and untimely deaths as a result of the immune damage wrought by this viral infection.

Dr Steven Joseph, New York City's Commissioner of Health, has been quoted as saying, "the way mankind responds to crisis is first disbelief, then denial, then the third stage is mobilization, and we're at the horizon of that now.[1] Perhaps the surest proof of that statement is the influence that epidemic HIV infection is having on organizations within our society. The media, business corporations, public and private educational institutions, correctional facilities, religious institutions, and health care organizations of all types, regardless of their source of funding, scope of services, or geographic location, are mobilizing to confront the issue of acquired immunodeficiency syndrome (AIDS). While such activity is preferable to the tepid organizational response that characterized the early years of the epidemic, this activity, in and of itself, will not insure a comprehensive or coordinated response to the problem of HIV infection. Because of the federal government's lack of a comprehensive plan of action in response to the AIDS epidemic in its early years, much of the burden for strategic planning and programmatic development fell to state and local governments, health care facilities located in high incidence areas, and a host of community organizations which sprang up to meet the needs of infected clients. Although the manner in which these entities confronted the epidemic—often with little support, sympathy, or funding—is laudable, it was not a substitute for a comprehensive, coordinated national plan of action against the virus. The federal coordination of a programmatic response to HIV infection is no less important than the coordination of a similar response with regard to biomedical research and vaccine development. This response should include initiatives to prevent the further transmission of the virus, to expand health care and related services in areas of the country with substantial numbers of cases, to modify existing systems of health care financing to meet the needs of individuals who have lost all of their assets to this infection, and to create mechanisms which would subsume a share of the financial burden for the out-of-hospital care of AIDS patients, which has, in many instances, been placed upon the nonprofit sector.[2]

As noted by Novick et al,[3] the proportion of incident AIDS cases reported in "high incidence" areas will continue to decrease as more cases are reported in "low incidence" areas. Centralizing our attack on AIDS will facilitate information and resource sharing such that providers in areas currently designated as "low incidence" can develop adequate programmatic structures in advance of the epidemic. They will not have to relive all of the frustration endured by "high incidence" communities in the first years of the epidemic in order to profit from their experience. Not only will centralization of strategy help to insure a similar level of quality across disparate communities, it will also minimize the local, often untoward, political response which AIDS-related programs sometimes generate, especially in the area of prevention services. Of all the programmatic challenges we face as a result of the AIDS epidemic, the creation of effective, targeted prevention programs is among the most difficult.

At least in theory, there appears to be broad-based support for the concept of preventing the further transmission of HIV. There are, however, important variables that influence the manner in which we approach the subject of HIV prevention, and which subsequently have an impact on the way in which we mount programs to actualize these objectives. Because substantial resources will be consumed in the process of providing care for those already infected, it is conceivable that funding for prevention initiatives will have to compete with funding for treatment. Culturally, we have a strong bias toward biomedical solutions to health problems, and this may also serve to minimize the resources that are designated for serious prevention efforts. It is readily apparent that it is far easier to reach a consensus on the appropriateness of developing an effective antiretroviral chemotherapeutic agent than it is to obtain endorsement of the aggressive promotion of an explicit message about sexual prophylaxis, or an evaluation of programs that would enable intravenous drug abusers to participate in sterile needle exchanges.[4] In the absence of an effective treatment or vaccine against HIV, programs for the prevention of infection must not limit themselves to the "seek and treat" mentality which has become the bulwark of disease control within the public health establishment for the past two decades. If our objective is to interrupt viral transmission, we must be willing to design programs that have a reasonable chance of achieving that goal.

There are other barriers to prevention as well. AIDS is a burden which is not equally distributed across society. The burgeoning analysis of this epidemic indicates that it is actually a series of "subepidemics" which are related but unequal.[5] Specific groups are overrepresented within the "dole," groups whose behaviors may not conform to societal notions of propriety or whose day-to-day living circumstances may be completely alien to the average citizen. In order to effectively prevent transmission of virus within these subpopulations, we must develop messages and modalities that are acceptable to them, which they can understand, and which they can ultimately come to accept as normative. Risk-reduction campaigns that have been effective among homosexual men are not ipso facto acceptable to sexually active inner-city youths, nor to the female sexual partners of intravenous drug abusers. To promote HIV prevention within these populations we must develop strategies that specifically "reflect the special characteristics, needs, and preferences of these target groups."[6] When AIDS prevention messages are homogenized to a level that makes them acceptable to a general audience, or are arbitrarily transferred from one "risk group" to the next, they may lose their appeal to the target audience for which they were designed.

An ongoing misunderstanding appears to be in the differentiation of public education campaigns from targeted prevention programs. Although the general public requires information about AIDS, and how the virus is transmitted, individuals who are at particular risk of infection will require more substantial, more intensive efforts to promote the modification of those behaviors that place them at risk for HIV infection. While the frequent airing of public service announcements about AIDS and media attention to the subject of HIV infection are integral components of the basic education we must undertake, they should not be mistaken for risk reduction interventions. The latter, while they incorporate an informational component, also address germane psychological, behavioral, and situational characteristics of the target population in an effort to promote behavioral change. Legislators, educators, and the panoply of individuals who are positioned to influence the development of prevention programs must be made to understand that risk reduction programs are based on social science theory and sound public health practice—not on whether or not they will be palatable to the public at large. This is not to negate or minimize the controversy that often attends such proposals, only to underscore the fact that we cannot afford to "politicize" the process of program development, especially in the area of prevention services.[7]

At this phase of the epidemic, AIDS has become a ubiquitous acronym, and for many of us, it is difficult to remember a time when it was not part of our consciousness or our vocabulary. Clearly there are those who still view it as the disease of "outsiders," as something they know of only indirectly. But as more people succumb to their infections, as more households and families become touched by the reality of sickness and death, this perception will become the luxury of the fortunate few. Certainly, AIDS is not the end of mankind, and those individuals or groups who have chosen to describe it as the 20th century bubonic plague have fortunately been proven wrong. Nonetheless, the epidemic is bound to have a lasting impact on our society. Not only will it influence the scope and direction of current biomedical research and medical therapeutics—it will also affect our cultural mores, our attitudes about death and dying, and many of our basic assumptions about the primacy of technology as a solution to problems. Although epidemics are first and foremost medical phenomena, they are also extraordinary catalysts for societal change. At this juncture, as expenditures are being designated for programs to meet the needs of the infected and the at-risk, it is essential that we plan our strategy carefully, with special attention to the circumstances and needs of the clients whom we wish to serve, and that we capitalize on the lessons we have learned in the past seven years.

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Acquired immunodeficiency syndrome as a paradigm for medicolegal education

In the fall semester of 1987, I attempted to introduce preclinical second-year medical students at the State University of New York Health Science Center at Brooklyn to the range of medicolegal issues that would confront them as they entered the clinical phase of their training. For many medical students, and for many in medical practice, "medicolegal issues" previously meant the issue of medical malpractice, in particular the risk that one might be sued. All students in the entire class (220 students) were introduced to this topic with two one-hour lectures which are part of a required course in health services taught by the Department of Preventive Medicine and Community Health.

By means of a lottery, students are assigned to seminar groups that meet for two hours once a week for eight weeks. A total of 14 seminar topics are offered, and most students are given their first choice.[1]

The seminar under discussion, "Legal Issues in Medicine," was specifically designed to introduce a small group of students to some other legal issues in medicine. A total of 13 students participated in the seminar.

The initial seminar session was devoted to legal issues raised by acquired immunodeficiency syndrome (AIDS), including concerns regarding antibody testing programs, confidentiality, and discrimination. The second seminar session addressed issues of biomedical research, including how studies might be designed to be both ethical and scientifically useful. The remaining six seminar sessions dealt with a variety of other topics, including the regulation of unsafe manufactured products, issues in forensic psychiatry, access to health care, legal protection of children's welfare, and patients' refusal of treatment.

Several of the seminar discussions revolved around hypothetical fact situations, similar to the "case method" often used in legal education. This method was chosen to allow the students to get a feel for the task of applying abstract rules to particular cases, to help them anticipate how difficult this might prove in the future in dealing with actual rather than hypothetical patients.

The written assignment for the seminar drew on the material presented in the first two seminar sessions. It called on the student to consider how to design a research proposal to study an AIDS vaccine. At the same time, initial reports of the first US trial of an AIDS vaccine were appearing in the press, and it was hoped that this would lend a further sense of immediacy and reality to the assignment.

One of the strongest impressions I carried away from the seminar was of the desire of many of the students for rules and certainty. Attempts to discuss what they thought would happen in a given hypothetical situation sometimes led to requests for more information about the relevant medical facts, and/or what the law said should be done. I think that here, as in other aspects of clinical medicine, one of the most difficult things for students to deal with is the need to decide and act in the face of uncertainty.

It was not my goal to try to produce legal experts, merely to alert the students to the fact that they might in the future sometimes benefit from seeking legal advice. Quotations from New York statutes and court cases were sometimes helpful in illustrating one state's attempts to grapple with some of these issues, and also in demonstrating how broadly stated rules can have considerable ambiguity when the attempt is made to apply them to particular cases.

In the end, much of our discussion of the law focused not so much on what the law is, but on what the students thought it should be. One useful exercise in this regard was to move from discussion of a hypothetical fact situation and how the students thought it should be handled, to discussion of how to formulate a general rule that would produce a just result in the given hypothetical case as well as in other future cases. My hope was that this would give the students a better appreciation of how difficult a task this can be.

In the process of making therapeutic decisions for patient care, the medical students in their clinical years will be exposed to the difficulty of choosing a course of action in the midst of uncertainty. One of the main points I hoped to convey was that some of these same problems arise as the law interacts with medicine.

There are several sources of uncertainty. In some cases, the law is reasonably clear, and the problem is primarily the physician's lack of information. As such, the solution is the clearcut one of seeking legal advice. In other cases, the law is phrased in terms of a general rule (eg, exercise "reasonable care"), while the application of that rule to the facts of a given case is ambiguous.

There are also more fundamental sources of uncertainty in the law, which arise primarily because legislators have chosen not to address issues that have long been recognized in medicine. These may range from defining the circumstances under which a patient may refuse life-sustaining treatment to the mechanics for making health care decisions on behalf of incompetent patients.[2] In many jurisdictions the trend has been away from unbridled discretion by the attending physician toward greater legal involvement in these matters, but this trend has not always been accompanied by clear guidance on what legal rules are to be applied.

Just as many courts and state legislatures have begun to deal with some of the thorniest medical-legal issues of the 1960s and 1970s, the AIDS epidemic has erupted. AIDS has intensified the concern over some of these issues, as AIDS patients consider how vigorously they wish to be treated, as well as who will make medical decisions for them if their mental status should be impaired by AIDS dementia, lymphoma of the brain, or opportunistic CNS infection. AIDS has also sparked a revival of interest in some issues that had nearly been forgotten in this country, particularly how one should balance the individual patient's rights against a societal interest in infection control measures.[3]

In summary, there are useful purposes that can be served by instruction in legal issues in medicine. First, it can afford an appreciation that there is more to the topic than malpractice law. Second, it is another way of introducing preclinical students to the difficulty of making decisions in the face of incomplete information. Third, by focusing not just on the question of what the law is but on what it should be, discussions such as these introduce medical students to society's role in addressing questions of medical ethics. Finally, it is my hope that this background will prove useful to medical students, as they prepare to begin clinical training and to confront the legal and ethical issues raised by AIDS.

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Medical students' attitudes towards caring for patients with AIDS in a high incidence area

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ABSTRACT. Second-year medical students (N = 174) at a medical school located in an area of high incidence for acquired immunodeficiency syndrome (AIDS) were surveyed for their attitudes and perceived risk of different degrees of contact with AIDS patients. Fifty percent of the class were surveyed prior to a 60-minute lecture on the epidemiology of AIDS; the other half were surveyed immediately thereafter. Data were analyzed by multivariate and univariate analyses of covariance and logistic regression. The lecture had no measurable impact on students' attitudes and perception of risk. More than 60% of students believed that drawing blood from an AIDS patient carried a moderate to high risk. More than 22% thought that performing a physical examination was associated with a moderate to high risk. Perceptions of risk associated with various types of patient contact generally correlated with views supporting the prerogative of declining care to AIDS patients. A large number of students expressed the view that physicians in private practice should have the prerogative of declining to care for new patients with AIDS (48.3%) and for longstanding patients who develop AIDS (41.4%) provided that care is insured elsewhere. Perception of risk correlated with choice of location of future residency training programs. These data suggest that medical students in the early years of training may have misperceptions of the risk of acquiring human immunodeficiency virus (HIV) infection not corrected by merely receiving scientific facts. These misperceptions may influence both career choices and site of graduate training if not modified by subsequent corrective experiences in the third and fourth years of medical school.

(NY State J Med 1988; 88:223-227)

As of February 1988, some 52,000 cases of acquired immunodeficiency syndrome (AIDS) had been reported to the US Centers for Disease Control.[1] Of these, 13,000 cases (25%) occurred in New York City, the two boroughs of Manhattan and Brooklyn accounting for the majority of cases.[1-3] While homosexual/bisexual men still account for the majority of cases in the city, intravenous (IV) drug abusers account for close to 30%.[4] Of the estimated 200,000 IV drug abusers in New York City, close to 60% are thought to be seropositive for the human immunodeficiency virus (HIV).[5] AIDS is currently the leading cause of death in New York City among men aged 25-44 years and women aged 25-34 years.[6]

HIV-infected IV drug abusers have progressively spread their infections to their heterosexual and/or homosexual sex partners.[7] A large number of women of childbearing age in New York City who contract AIDS do so by sharing contaminated injection equipment during drug abuse or by sexual contact with a male drug user.

From late November 1987 through late December 1987, the New York State Department of Health tested the blood of every infant born in the state for HIV.[8] A total of 19,157 infants were tested, and overall 164 were found to be positive. Of the 9,047 infants born in New York City during the month-long period, 148 were positive.[8] Thus the rate of positivity for New York City was 1.64%, or 1 in 61.[8] The highest rate of positivity was in the borough of the Bronx (2.24%) followed by Manhattan (1.96%), Brooklyn (1.74%), Queens (0.81%), and Staten Island (0.76%).[8] These rates of positivity are thought to reflect the rate of HIV infection among the city's drug users and their sexual partners.

The State University of New York Health Science Center at Brooklyn (SUNY HSCB) is geographically located in a high AIDS incidence area in the city. Its major teaching hospital affiliate, Kings County Hospital Center (KCHC), is a 1,200-bed municipal hospital situated across the street from the SUNY HSCB. Both institutions are situated in an inner city minority area where intravenous drug abuse is a very significant problem.

There are a total of 5,000 deliveries a year at KCHC and an equal number of planned abortions.[9] Some 500 deliveries (10%) are among Haitian women.[9] In a recent study at KCHC it was found that 12 (2%) of 602 core blood samples were HIV positive.[9] Approximately half of the beds on the medical service at KCHC are currently occupied by AIDS patients.[10]

The high incidence of AIDS in the community served by SUNY HSCB has greatly increased the frequency

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with which physicians, house staff, and clinical clerks must care for patients who have the disease or who are HIV positive. The presence of large numbers of AIDS patients on a teaching service creates numerous medical, legal, ethical, and social issues which must be dealt with by these medical practitioners on an ongoing basis.

Other investigators have reported on medical students' attitudes towards AIDS and homosexual patients in a geographic area of low incidence.[11] However, the current study evaluated both attitudes and perceived personal risks associated with the care of AIDS patients as well as projected medical care behavior in one of the highest incidence areas in the United States. The study had several purposes, among which were to document second-year medical students' attitudes towards AIDS patients, to assess their perceptions of risk to self in caring for these patients, to examine correlations, if any, between risk perception and projected medical care behavior, and to see if a detailed lecture on AIDS and its risks would alter preexisting perceptions of risk to self in providing medical care.

METHODS

The survey was conducted among second-year medical students in December 1987. This is a time in the curriculum when the students have not yet received any systematic training in the clinical sciences. The questionnaire contained 25 questions that were of the fixed-alternate type, with space being provided for individual open-ended responses at the end. The survey instrument was administered during the final small group seminar session of the second-year course in preventive medicine and community health. Student participation was completely voluntary, and students were told not to identify themselves on the survey in any manner. To insure anonymity, students collected the questionnaires and brought them to the departmental offices. A total of 174 of 209 (83.2%) students completed the questionnaire. In order to assess the impact, if any, of the AIDS lecture, responses provided by 62 students on a Tuesday, two days before the lecture, were analyzed separately from those of 77 respondents who completed the questionnaire the afternoon following the lecture on Thursday. The course offered several seminars, two of which included discussions on some topics related to AIDS. All seminars consisted of eight two-hour weekly sessions. A total of 35 students were enrolled in these two seminar groups, and were excluded from the evaluation of the lecture.

Analysis of covariance was used to compare the Thursday lecture group with the Tuesday lecture group. Both multivariate and univariate analyses of covariance were used to assess differences in perceived risk of various behaviors and to ascertain a pattern of consistent responses. The impact of perceived risk on behaviors and attitudes was assessed using a polytomous logistic regression model with three outcomes: yes, no, or maybe.[12] Perceived risk was based on the sum of risk scores assigned to taking a medical history, performing a physical exam, performing invasive procedures, drawing blood, or being in the same room with an AIDS patient. The summary score could range from 5 to 30, with 30 indicating a very high perceived risk for each of the behaviors, and 5 indicating no risk associated with any of them.

RESULTS

Descriptive Data. The first four questions solicited basic information on age, sex, marital status, and number of children. Of the total group 77.0% were single, 12.6% were married, and 9.8% were engaged. Of the 174 students, 52.3% had personally seen a patient with AIDS, and 25.9% knew of someone (neighbor, acquaintance, relative, student, etc) with AIDS or who had died of AIDS. Results for the five questions that elicited perceptions of risk associated with a variety of activities are shown in Table I. The lowest perceived risk behavior was being in the same room with an AIDS patient, 93.1% rating this risk as low, very low, or none. The next lowest risk behavior was rated as taking a medical history from an AIDS patient, 90.8% rating this risk as low, very low, or none. By contrast, 76.9% rated performing a physical examination in these categories and 22.4% responded that performing a physical examination carried a very high, high, or moderate risk. Performing invasive procedures was rated as carrying the greatest risk, with 66.7% responding that this activity carried a very high, high, or moderate risk. This was followed by 62.0% who rated drawing blood as carrying a very high, high, or moderate risk (Table I).

TABLE I. Medical Students' Perceived Risk of AIDS Transmission by Activity (N = 174)

	Perceived Risk (%)						
Activity	Very High	High	Moderate	Low	Very Low	None	Don't Know
Taking medical history	1.2	0.6	6.9	7.5	20.1	63.2	0.6
Performing physical examination	2.9	4.0	15.5	21.8	40.2	14.9	0.6
Performing invasive procedures	13.8	17.8	35.1	18.4	11.5	1.1	2.3
Drawing blood	12.6	17.8	31.6	22.4	13.8	1.7	0.1
Being in same room	1.7	1.1	2.9	4.0	17.8	71.3	1.1

With regard to whether students would allow their own children to be in the same room as AIDS patients, 41.4% said yes, 17.8% said no, 26.4% said maybe, and 13.8% said they didn't know.

Medical students' beliefs regarding their prerogatives relating to medical care behaviors are presented in Table II. The two activity areas of concern to students were drawing blood and performing surgical procedures. With regard to the former, 31.6% replied yes and 14.9% replied maybe that they should have the prerogative of declining to draw blood. For surgical procedures the figures are 43.1% and 23.0%, respectively. Even with regard to performing physical examinations on AIDS patients, 13.8% said yes and 17.2% said maybe concerning the prerogative of declining to do so.

Whereas 21.8% of respondents were unsure or thought that medical students should have the prerogative of declining to take a medical history from an AIDS patient, only 10.9% thought interns and residents should (Table III). With regard to interns and residents not performing physical examinations, 5.2% said yes and 12.6% said maybe, much lower than the respective figures of 13.8% and 17.2% for medical students (Table II). While 31.6% of students thought they should have the prerogative of declining to draw blood from AIDS patients, only 14.4% thought interns and residents should (Table III). Yet 25.3% said interns and residents should be able to

TABLE II. Medical Students' Beliefs About Their Prerogatives Relating to the Care of AIDS Patients (N = 174)

	Belief				
Activity	Yes	No	Maybe	Don't Know	No Answer
Decline to take medical histories	6.9	78.2	10.9	3.5	0.6
Decline to perform physical examination	13.8	66.1	17.2	0	2.9
Decline to draw blood	31.6	50.0	14.9	3.5	0
Decline to perform surgical procedures	43.1	28.7	23.0	4.6	0.6

TABLE III. Medical Students' Beliefs About the Prerogatives of Interns and Residents Relating to the Care of AIDS Patients (N = 174)

	Belief				
Activity	Yes	No	Maybe	Don't Know	No Answer
Decline to take medical histories	3.5	89.1	6.3	0.6	0.6
Decline to perform physical examination	5.2	79.9	12.6	1.7	0.6
Decline to draw blood	14.4	71.3	10.3	2.9	1.2
Decline to perform surgical procedures	25.3	49.4	20.7	3.5	1.2

decline performing surgical procedures on AIDS patients and 20.7% said maybe (Table III). Overall, significantly more students thought that they should have the prerogative of declining to perform these activities compared to interns and residents.

When asked about the relative infectivity of AIDS patients and persons who are HIV positive, 27.0% replied that AIDS patients pose a greater risk, 56.9% said they posed the same risk, 14.9% did not know, and 1.6% gave no answer.

In response to whether a large census of AIDS patients in a teaching hospital would reduce the scope and value of training offered to interns and residents, 40.2% said yes and 28.5% said maybe. When asked about the effect of a high proportion of AIDS patients in a hospital respected for the quality of its teaching programs, 45.4% said it would make the hospital less appealing for training. Only 4.0% said it would make it more appealing and 37.9% said it would have no influence.

Students were asked if physicians in private practice should have the prerogative of declining to care for AIDS patients. This question was framed with regard to both new patients with AIDS and longstanding patients who develop AIDS. Concerning new patients, 10.3% said that physicians should have the prerogative of declining care outright with no questions asked. Only 4.0% gave this response for longstanding patients who subsequently develop AIDS. For new patients, 48.3% responded that physicians should have the prerogative of declining care provided the patient is insured care elsewhere. This response was given by 41.4% with regard to longstanding patients. Male and female students did not vary in their perceptions of risks or predicted behaviors.

Impact of the Lecture. There were no significant differences in perceptions of risk or beliefs regarding prerogatives relating to the care of AIDS patients between the students who received the one-hour lecture on AIDS and those who did not. The largest difference between groups was for perceived risk of being in the same room with an AIDS patient, which 8.1% of the nonlectured group viewed to be of moderate to higher risk compared to 3.9% of the lectured group ($p < 0.07$).

TABLE IV. Perceived Risk as a Percent of Maximum Risk Score by Beliefs Regarding Medical Students' Prerogatives

Prerogative to Decline for AIDS Patients:	Belief			Probability	Change in the Probability* of Response = No†
	Yes	No	Maybe		
Taking medical histories	60.2	42.4	55.1	0.001	−2.9%
Performing physical examination	57.5	41.1	51.9	0.001	−4.0%
Drawing blood	51.3	41.2	47.7	0.05	−3.4%
Performing surgical procedures	48.1	42.7	44.8	NS‡	

* Adjusted for sex and personal knowledge of an AIDS patient.
† Per unit increase in risk score.
‡ NS, not significant.

TABLE V. Perceived Risk as a Percent of Maximum Risk Score by Beliefs Regarding Interns' and Residents' Prerogatives

Prerogative to Decline for AIDS Patients:	Belief			Probability	Change in the Probability* of Response = No†
	Yes	No	Maybe		
Taking medical histories	61.1	44.6	50.6	NS	
Performing physical examination	56.3	43.5	53.6	0.01	−1.9%
Drawing blood	50.6	42.8	53.2	0.01	−2.4%
Performing surgical procedures	48.8	43.2	46.1	NS‡	

* Adjusted for sex and personal knowledge of an AIDS patient.
† Per unit increase in risk score.
‡ NS, not significant.

Influence of Perceived Risk on Predicted Behavior. The associations of perception of risk with students' beliefs regarding prerogatives to decline different types of contact with AIDS patients are shown in Tables IV–VI. The average risk score is indicated according to whether the student believed that medical practitioners should have the prerogative of declining service. The last column shows the percentage increase in the probability of a negative response for every unit increase in the risk score. Overall, the perception of risk score was 45.6% of the possible maximum.

There were significant differences in beliefs about medical student prerogatives in declining to take a medical history, perform a physical examination, and draw blood. In each instance, those students with a higher perception of risk were more likely to think that medical students in general should have the prerogative of declining care ($p < 0.01$). The exception was participation in surgical procedures where the trend was similar but the difference was not significant. Here, the majority thought that medical students should (43.1%) or maybe should (23%) have the prerogative to decline participation. With minor exception (being in the same room) the beliefs of the students carried over to expectations for interns and residents (Table V). Once again, the performance of surgery on AIDS patients was thought to carry high risk by a large number of students and was not influenced by the students' perception of risk score. There was no association between the risk score and beliefs about the prerogatives of private physicians to decline caring for an AIDS patient (Table VI).

Students who would not allow one of their own children in the same room with an AIDS patient had an average score of 61.7% compared to 39.6% for those who would ($p < 0.001$) (Table VII).

Students with a higher risk score thought that the presence of

TABLE VI. Average Percent Risk of Maximum Score* by Beliefs Regarding Private Practitioners' Prerogatives

	Belief			
Prerogative to Decline:	Unqualified Yes	Yes, if Alternative Care Is Assured	No	Probability
Care for new AIDS patients	48.4	45.4	44.3	NS†
Care for patients of some years who develop AIDS	51.3	44.7	46.4	NS

* Adjusted for sex and personal knowledge of an AIDS patient.
† NS, not significant.

TABLE VII. Perceived Risk as a Percent of Maximum Score by Attitudes Concerning AIDS Patients

	Belief				Change in the *Probability of
	Yes	No	Maybe	Probability	Response = No†
Allow own children to visit AIDS patients	39.6	61.7	44.9	0.001	3.0%
Risk of transmission is the same for AIDS patients and HIV-positive individuals	48.6	43.7	45.9	NS‡	
A large AIDS patient load would reduce the scope and value of postgraduate training	48.9	44.3	41.6	0.01	2.3%—Yes
A large AIDS patient load at a well-respected teaching hospital would make it less appealing for postgraduate training	48.4	44.3	42.9	0.01	2.6%

* Adjusted for sex and personal knowledge of an AIDS patient.
† Per unit increase in risk score.
‡ NS, not significant.

many AIDS patients reduced the scope and value of residency training and noted that this would make an otherwise acceptable training program less appealing.

DISCUSSION

The results of this study indicate that second-year medical students have definite perceptions of the risk of acquiring AIDS through various types of patient contact. These perceptions frequently correlated with beliefs regarding prerogatives related to medical care behavior. Neither the perceptions of risk nor beliefs were influenced by a lecture on AIDS which carefully set forth the epidemiology of the disease and the risks associated with various types of contact. Those students who expressed a higher perception of risk with regard to taking a medical history, performing a physical examination, or drawing blood were more likely to think that they should have the prerogative of declining care.

Students viewed the performance of invasive surgical procedures and the drawing of blood as carrying the greatest risks. Large numbers of students (43.1%) thought that they should have the prerogative of declining to participate in the performance of surgical procedures or the drawing of blood (31.6%). It is noteworthy that student views for these activity prerogatives for interns and residents were 25.3% and 14.4% respectively (Tables II, III). These differences may in part be explained by some of the subjective comments made by students on their response sheets. A number of them expressed the concern that they, unlike interns and residents, are not yet skilled in the performance of these procedures. Therefore they perceive that they would be more likely to accidentally infect themselves. Thus, declining to participate in these procedures with AIDS patients may reflect a concern about greater risk due to a lack of requisite skills. Such views might well change as students acquire these skills in their third year of training.

Medical educators have become increasingly concerned about the impact of the presence of large numbers of AIDS patients on teaching services. These concerns relate to recruitment of house officers and maintaining a mix of patients manifesting a broad scope of disease problems necessary for their education. It is clear from this study that many students (40.2%) think that having significant numbers of AIDS patients in a teaching hospital reduces the scope and value of training offered to house staff. Similarly, 45.4% were of the opinion that such hospitals would be less appealing for training. If these views persist throughout subsequent years of medical school, they may have an important influence on the choice of hospital for postgraduate training. This has very important implications for teaching hospitals in high AIDS incidence areas.

The refusal of a few private practice physicians to treat AIDS patients has in part led a number of professional organizations and regulatory bodies to reaffirm the legal and ethical responsibilities of physicians to treat these patients. In November 1987, the American Medical Association's (AMA) Council on Ethical and Judicial Affairs published a report, *Ethical Issues Involved in the Growing AIDS Crisis*.[13] The report addressed a range of issues and firmly stated that "a physician may not ethically refuse to treat a patient whose condition is within the physician's current realm of competence." [13-15] A number of other professional organizations have taken similar stands.[16]

The Associated Medical Schools of New York, a professional organization that represents all of New York State's 13 medical schools, took a strong stand on this issue in December 1987. In a statement, the association recommended that any student, resident, or intern who refused to treat HIV infected individuals should be dismissed.[17]

The New York State Department of Health, which is responsible for ensuring the care and treatment of all members of the public in facilities and programs licensed under New York State law, has also issued a strong policy statement on discrimination against AIDS patients.[18] The department does not officially recognize AIDS as either a sexually transmitted disease or as a communicable disease, but as "a physical impairment closely associated with the human immunodeficiency virus (HIV)." The reasons for this position relate to concerns that a sexually transmitted disease or communicable disease classification would require the implementation of public health measures (eg, contact tracing) which would lead to discrimination against AIDS patients in housing, employment, insurance, etc. In November 1987, the New York State Court of Appeals, the highest court in the state, ruled that AIDS is a communicable disease.[19] However, this ruling has limited application to the conjugal visits for prisoners who are HIV positive.

Invoking the provisions of the Federal Rehabilitation Act of 1973, the department has the statutory legal authority to prosecute and punish any medical facility that refuses to treat a patient solely by reason of their AIDS handicap.[18] Despite these position statements and the force of law, it is still possible for physicians in private practice to avoid treating AIDS patients if they so wish. Surgical specialists, at generally higher risk than primary care practitioners because of the procedures they perform, can simply decline referrals on the grounds of being overbooked. There are a number of anecdotal reports of surgical specialists using this mechanism.

It is significant that 48.3% of students stated that physicians in private practice should have the prerogative of declining to care for new patients with AIDS provided care was insured elsewhere; the figure for longstanding patients was 41.4%. Far fewer students gave interns and residents this prerogative (Table III), perhaps in part because of the perception that house staff have few options in the matter. It is noteworthy that these opinions were expressed at a time when the ethical issue of physician refusal to treat AIDS patients was prominent in the popular press.

The data from this study suggest that early in training students may have misperceptions of the risk of acquiring HIV infection which are not corrected by simply transmitting the facts. These misperceptions may influence subsequent career choice and site of graduate training if they persist into the clinical years of medical school training. It is possible that subsequent experience with the care of AIDS patients during the third and fourth years of medical school may correct both the perceptions of risk and projected behaviors expressed by second-year medical students.

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Geographic and demographic features of the AIDS epidemic in New York City

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ABSTRACT. Extensive surveillance and epidemiologic investigations of acquired immunodeficiency syndrome (AIDS) have enabled the New York City (NYC) Department of Health (DOH) to plan and focus prevention and education efforts. While the DOH manages a number of support and counseling services, many such programs are administered by local community groups. In order to provide appropriate services and keep their communities properly informed, agencies require epidemiologic information specific to their geographic areas. Using surveillance and vital record data, this report analyzes basic epidemiologic features of AIDS in NYC, stressing racial/ethnic and geographic patterns. Borough-specific incidence rates indicate that Manhattan has been affected most severely, with a rate more than 2.5 times that of the borough with the next highest rate, the Bronx. Manhattan is the only borough in which whites have the highest incidence of AIDS; rates in blacks and Hispanics are higher in every other borough and citywide. The disproportionate racial/ethnic impact of AIDS is also evident in mortality rates. Compared to whites, blacks and Hispanics, particularly women, are at a considerably increased risk of dying of AIDS. Mortality rates in small geographic units are also presented in order to provide a more detailed picture of AIDS in NYC.

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Since the first reports of acquired immunodeficiency syndrome (AIDS) in New York City in 1981, the principal

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Data for this analysis were provided by the AIDS Surveillance Unit and the Bureau of Health Statistics and Analysis of the New York City Department of Health.

TABLE I. Cumulative AIDS Incidence Rates per 10,000 Population By Borough of Residence and Race/Ethnicity, 15-64-Year-Olds Only, New York City, 1981-1986

	Race/Ethnicity			
Borough	White	Black	Hispanic	Total*
Manhattan	49.7 (2,684)†	48.9 (972)	34.3 (799)	43.5 (4,455)
Bronx	6.1 (151)	19.7 (458)	24.6 (626)	16.7 (1,235)
Brooklyn	6.2 (441)	18.7 (854)	15.5 (383)	11.7 (1,678)
Queens	4.3 (341)	16.7 (382)	12.0 (217)	7.5 (940)
Staten Island	3.7 (77)	14.1 (22)	15.7 (19)	4.9 (118)
Total	14.9 (3,694)	23.7 (2,688)	21.0 (2,044)	18.0 (8,426)

* Not included are 53 cases of other or unknown race/ethnicity.
† Rate (cases).

risks and routes of transmission of this disease have been well established.[1] Extensive surveillance and epidemiologic investigation have enabled the New York City (NYC) Department of Health (DOH) to focus its prevention and education efforts.[2] While the DOH supports a wide variety of AIDS services and itself provides an information hotline, counseling service, and anonymous human immunodeficiency virus (HIV) testing clinics, many support programs active in NYC are run by local community groups. In order to provide appropriate services and keep their communities properly informed, agencies require epidemiologic information specific to their own treatment areas.

METHODS

In this study, two sources of data, the AIDS surveillance registry and NYC death certificates, were used to analyze geographic and demographic patterns of AIDS in NYC. Borough-specific incidence trends were assessed using the surveillance registry; mortality rates in small geographic units (health districts) were obtained from death certificates. Together these data provide a comprehensive picture of AIDS epidemiology in NYC.

Surveillance data, collected since 1981, are the major source of demographic and patient risk information on cases of AIDS in adults (patients aged 13 years and older) in NYC. The case definition used for surveillance (through August 1987) is that established by the Centers for Disease Control,[3] and includes only those persons with severe (pathologically confirmed) opportunistic infections attributable to HIV infection. The surveillance system is fully described in other reports.[2,4] This analysis includes cases diagnosed from 1981 through 1986 and reported through April 1987. Mortality data are derived from death certificates maintained by the Bureau of Vital Records in the Department of Health. Data accumulated for this period are nearly complete; 90% of AIDS cases identified through surveillance are reported within six months of diagnosis (NYC Department of Health AIDS Surveillance, unpublished data). Deaths are coded using ICD-9 criteria for underlying cause of death. Deaths in which a history of narcotics use is confirmed by the medical examiner are also noted on certificates and are classified as "narcotics-related." All mortality data presented here include 15-64-year-old residents of NYC only. Incidence and mortality rates are generated using 1980 NYC census data.

RESULTS

Compared to all other boroughs, cumulative incidence rates in Manhattan are the highest in all racial/ethnic groups (Table I). However, this is the only borough in which whites have the greatest incidence of AIDS; rates among blacks and Hispanics exceed those of whites in all other boroughs and in total, citywide incidence.

The distribution of risk groups for AIDS differs considerably by borough of residence (Table II).[5] In Manhattan, cases among nonintravenous-drug-using homosexual or bisexual men account for 74.4% (3,341) of all cases diagnosed in this borough from 1981 through 1986. This proportion has been fairly consistent since 1982, the first complete year of reporting (Table III). The incidence of AIDS among intravenous (IV) drug users, as among homosexual men, has increased steadily since 1981. IV drug users have consistently accounted for about 22% (1,006) of Manhattan cases. Finally, 51 cases of AIDS in women attributed to heterosexual contact with a male IV drug user or a bisexual man have been reported in Manhattan. The rate of such cases has remained at just over 1% of Manhattan cases. Because of the high proportion of cases among homosexual men in this borough, the male-to-female ratio of AIDS cases is high: 16:1 vs 9:1 citywide.

The distribution of cases in the Bronx contrasts greatly with that of Manhattan: A majority of cases in this borough (773, or 62%) are attributable to IV drug use, and only 26% (318) to homosexual contact. These proportions have also been fairly steady since 1982. Cases acquired by male-to-female heterosexual transmission comprise 6% (73) of all cases of AIDS in the Bronx. This is the highest such proportion of all the boroughs, and (in addition to the large proportion of AIDS in the Bronx among women) is reflected in the lowest borough-specific male-to-female case ratio in the city, 4.9:1. Interestingly, 60% of heterosexually acquired disease has occurred among Hispanics, as has a nearly identical proportion of IV-drug-related AIDS cases in this borough. This similarity in the racial distribution of AIDS among male IV drug users and women in whom it was heterosexually acquired is present in all boroughs (Table IV). There are only five cases in NYC of men for whom heterosexual contact with a woman is considered the principal source of HIV infection.

Brooklyn and Queens are the only boroughs to have experienced an increase in the proportion of cases occurring among homosexual men. These accounted for 28% of all Brooklyn cases in 1982 and for 39% in 1986. In this period, the proportion of IV-drug-related cases declined from 52% to 45%. Nearly 15% of the cases reported from Brooklyn occurred among persons of Haitian origin; citywide, 75%

TABLE II. Cumulative AIDS Incidence By Borough and Patient Group, New York City, 1981-1986

	Patient Group				
Borough	Homosexual/ Bisexual	Intravenous Drug User*	Male-to-Female Transmission	Other†	Total
Manhattan	74.4 (3,341)‡	22.4 (1,006)	1.1 (51)	2.1 (95)	53.0 (4,493)§
Bronx	25.7 (318)	62.4 (773)	5.9 (73)	6.0 (74)	14.6 (1,238)
Brooklyn	35.6 (599)	46.7 (786)	3.4 (57)	14.4 (242)	19.9 (1,684)
Queens	46.8 (443)	43.5 (412)	2.9 (27)	6.8 (64)	11.2 (946)
Staten Island	41.5 (49)	50.0 (59)	0.8 (1)	7.6 (9)	1.4 (118)
Total	100.0 (4,750)	100.0 (3,036)	100.0 (209)	100.0 (484)	100.0 (8,479)

* Includes homosexual/bisexual IV drug users.
† Includes blood-product-related cases, persons from countries where risks are unclear, and those with no identified risk.
‡ Row % (cases).
§ Column % (cases).

TABLE III. Percent Distribution of AIDS Cases by Year, Borough, and Major Patient Groups, New York City, 1982–1986

Borough	Patient Group	Year of Diagnosis 1982	1983	1984	1985	1986
Manhattan	Homosexual/bisexual	78.0*	73.1	76.9	74.6	72.2
	IV drug user	20.0	24.6	19.6	22.6	24.1
	Male-female transmission	1.0	0.3	0.9	1.0	1.6
	Total (N)	241	529	875	1290	1567
Bronx	Homosexual/bisexual	20.9	29.1	25.6	26.3	24.1
	IV drug user	62.8	63.3	65.0	62.8	62.1
	Male-female transmission	6.9	3.6	4.4	7.0	6.2
	Total (N)	43	110	226	369	494
Brooklyn	Homosexual/bisexual	27.9	29.3	36.4	34.0	39.6
	IV drug user	52.4	45.7	47.2	49.7	44.2
	Male-female transmission	0	4.9	1.8	3.3	4.2
	Total (N)	61	164	324	453	689
Queens	Homosexual/bisexual	36.8	45.3	49.4	39.6	49.6
	IV drug user	50.0	39.5	41.3	52.0	40.8
	Male-female transmission	2.6	2.3	2.3	2.5	3.4
	Total (N)	38	86	174	275	385
Staten Island	Homosexual/bisexual	25.0	50.0	33.0	44.7	39.1
	IV drug user	75.0	40.0	47.6	44.7	54.3
	Male-female transmission	0	0	0	0	6.5
	Total (N)	4	10	21	38	46

* Column percent.

TABLE IV. Racial/Ethnic Distribution (Percent) of Male Intravenous Drug and Female Heterosexually Acquired AIDS By Borough,* New York City, 1981–1986

	Manhattan		Bronx		Brooklyn		Queens	
	IV Drug Use	Heterosexual	IV Drug Use	Heterosexual	IV Drug Use	Heterosexual	IV Drug Use	Heterosexual
White	19.0	20.4	7.9	8.3	16.4	17.9	25.1	20.0
Black	48.1	40.8	33.9	32.0	50.1	48.2	52.1	64.0
Hispanic	33.7	36.7	58.0	59.7	32.1	33.9	22.4	16.0

* Only two male-to-female cases have been reported from Staten Island.

of all Haitians with AIDS are from Brooklyn. (According to the NYC Department of Planning, 63% of Haitian-born immigrants reside in Brooklyn and 27% live in Queens.) After the Bronx, Brooklyn has the lowest male-to-female case ratio, 6.2:1.

While whites and blacks comprise between 35% and 40% of AIDS cases in Queens, the cumulative incidence rate among blacks (17/10,000) is four times that of whites (4/10,000), while the rate among Hispanics (12/10,000) is three times that of whites. Furthermore, within each racial/ethnic group, the distribution of cases by risk group differs considerably. Among blacks, for example, 61% of cases are IV drug related and 31% occur among homosexual men. These proportions are reversed in whites. Again, the racial group with the greatest proportion of male IV drug users (in this case blacks) also has the largest number of heterosexually acquired cases in women. The male-to-female ratio of cases in this borough is 8.5:1.

Only 118 cases of AIDS were diagnosed in Staten Island between 1981 and 1986. This represents 1.4% of all NYC cases, and a cumulative incidence rate of 4.9 per 10,000 population, the lowest in the city. Reflecting the racial distribution of this borough, 63% (77) of the patients are white and nearly 50% (36) are homosexual men. The male-to-female ratio of AIDS cases in this borough is 9.5:1.

Mortality Rates. The following refers only to 15–64-year-old white, black, and Hispanic residents of NYC who died in 1986 and whose underlying cause of death is recorded as AIDS on the death certificate. AIDS mortality and proportional mortality rates in 1986 by age, sex, and race/ethnicity are presented in Table V. From a previous link of death certificates and the AIDS surveillance registry, three ICD-9 codes were found to be the most sensitive indicators of AIDS deaths.[6] These codes are 279.1 (deficiency of cell-mediated immunity); 136.3 (pneumocystosis); and 173.9 (malignant neoplasm of the skin, site unspecified). There were 2,441 AIDS-related deaths among NYC residents 15–64 years of age during 1986, yielding a crude mortality rate of 52.0 per 100,000 population.* Racial/ethnic data were missing or unknown in 169 cases; eight deaths occurred among Asians.

In all racial groups, mortality rates among 25–44-year-old males far exceeded the levels in the younger (15–24 years) and older (45–64 years) age groups. Mortality rates, reflecting incidence rates, were highest in blacks and Hispanics.

Compared to whites, blacks in the 25–44-year age group had a 2.2 times greater risk, and Hispanics a 1.5 times greater risk, of dying of AIDS. While the AIDS mortality rate among men 25–44 years of age was lowest among whites, AIDS did account for the highest proportion of deaths (35.2%) in this group. This indicates that black and Hispanic males in this age group are at greater risk of dying of other, non-AIDS-related causes.[7]

As in males, AIDS mortality rates in females are highest in 25–44-year-olds of all racial/ethnic groups. Furthermore, the rate is again highest in blacks and Hispanics. Compared to white women, the relative risk of dying of AIDS was elevated in black women (5.1) and Hispanic women (3.7). This disproportionate racial/ethnic elevation in the risk of death due to AIDS was originally reported in 1984.[6]

Health District Rates. AIDS mortality among males within each health center district, and the proportion associated with nar-

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* Using ICD-9 code 279.1 only, the Bureau of Health Statistics and Analysis reported 2,407 AIDS deaths among New York City residents aged 15 to 64 in 1986. This represents a crude mortality rate of 51.3 per 100,000 population. These rates do not reflect, of course, the degree of underaccounting of AIDS deaths which occurs due to misclassification of underlying cause of death.

TABLE V. AIDS Mortality Rates (per 100,000) and Proportional Mortality* By Sex, Age, and Race/Ethnicity, New York City, 1986

	Race/Ethnicity†					
	White		Black		Hispanic	
	Rate (No.)	PM‡	Rate (No.)	PM	Rate (No.)	PM
Males						
15-24 years	7.7 (20)	10.4	14.5 (22)	6.2	13.6 (18)	7.8
25-44 years	108.5 (553)	35.2	236.3 (500)	25.4	167.0 (421)	30.4
45-64 years	49.91 (212)	5.4	104.6 (136)	5.2	81.5 (77)	6.6
Total	65.8 (785)	13.6	113.6 (658)	13.2	107.8 (516)	18.6
Females						
15-24 years	1.1 (3)	4.4	6.3 (11)	10.0	4.0 (6)	8.4
25-44 years	9.3 (48)	9.0	47.6 (134)	14.8	34.1 (80)	19.8
45-64 years	0.8 (4)	0.2	7.7 (14)	0.8	4.0 (5)	0.8
Total	4.3 (55)	1.8	24.9 (159)	5.4	18.0 (91)	8.2

* The proportion of all deaths in the specified age, race, and sex group that was attributed to AIDS.
† 169 AIDS deaths occurred in other or unknown racial/ethnic groups and there were eight deaths among Asians.
‡ PM, proportional mortality (%).

TABLE VI. Age- and Race-Adjusted* AIDS Mortality Rates per 100,000, Males 15-64 Years Only, By Health Center District of Residence, New York City, 1986

Borough	District	Number of Deaths	Adjusted Rate	% Narcotics Related
Manhattan	Central Harlem	73	76.1	64.4
	East Harlem	59	157.6	45.8
	Kips Bay	73	150.4	2.8
	Lower East Side	156	205.7	23.0
	Lower West Side	349	299.3	6.4
	Riverside	131	171.1	9.2
	Washington Heights	55	89.1	31.0
	Total	896		
Bronx	Fordham	48	77.2	43.8
	Morrisania	54	295.7	64.8
	Mott Haven	46	92.8	67.4
	Pelham	41	67.2	39.0
	Tremont	56	74.5	49.6
	Westchester	69	81.8	49.2
	Total	314		
Brooklyn	Williamsburg	37	128.4	67.6
	Bay Ridge	14	22.9	28.6
	Bedford	66	95.9	51.6
	Brownsville	64	69.8	59.4
	Bushwick	46	89.7	76.0
	Flatbush	74	49.5	24.4
	Fort Greene	56	117.0	44.6
	Gravesend	26	65.8	46.2
	Red Hook	39	104.7	25.6
	Sunset Park	25	79.1	40.0
	Total	447		
Queens	Astoria	35	79.1	28.6
	Corona	52	64.5	27.0
	Flushing	23	39.6	13.0
	Jamaica East	78	46.9	53.8
	Jamaica West	38	42.8	34.2
	Maspeth-Forest Hills	18	18.7	5.0
	Total	244		
Staten Island		30	38.9	33.4
Total New York City		1,932		32.6

* Direct method using 1980 New York City census data.

cotics use, are shown in Table VI. A map of the number of deaths attributed to AIDS by zip code of residence appears in Figure 1. Age- and race-adjusted mortality rates were calculated to control for the different race and age compositions in the districts. Because of the relatively small number of deaths among females, and the degree of stratification necessary for the adjustment, we were unable to calculate adjusted rates for females.

Four of the five highest adjusted rates are in Manhattan. Morrisania, in the South Bronx, has the second highest adjusted rate after the Lower West Side. In Manhattan, three areas with high AIDS mortality rates are readily visible from the map. Most prominent, in the southern portion of the island, are five zip codes within the health districts of the Lower East and West Sides. The Upper West Side (Riverside) and East Harlem, with respective mortality rates of 171 and 157 per 100,000 population, have also been affected severely.

The health districts of Morrisania and Mott Haven, which constitute the South Bronx, have the highest adjusted mortality rates in this borough, and the largest proportion of deaths (66%) that were narcotics related. Three contiguous health districts along the western portion of Brooklyn—Williamsburg, Fort Greene, and Red Hook—have the highest adjusted mortality rates in this borough. Districts with the largest number of deaths—Flatbush, Bedford, and Brownsville—are in central Brooklyn. Although Astoria and Corona, in the western portion of Queens, have the highest adjusted AIDS mortality rates in this borough, East Jamaica had the greatest number of deaths and the highest proportion of deaths (54%) attributable to IV drug use.

DISCUSSION

This study briefly describes the demographic impact of AIDS in NYC, stressing racial/ethnic and geographic features of the epidemic. A descriptive analysis can be useful to community groups and organizations providing education and support services to those already infected and to those at increased risk of infection. Incidence and mortality rates enable one to compare the impact of AIDS among health districts, and to draw a detailed picture of AIDS epidemiology within a specific neighborhood.

At least three factors influence the distribution of AIDS: varied racial/ethnic, age, and sex compositions within the city; different degrees and types of risk-associated behavior practiced therein; and different underlying seroprevalence levels of the virus. As is clear from the figures presented here, the epidemiology of AIDS is quite different across the five boroughs of NYC and within the boroughs themselves.

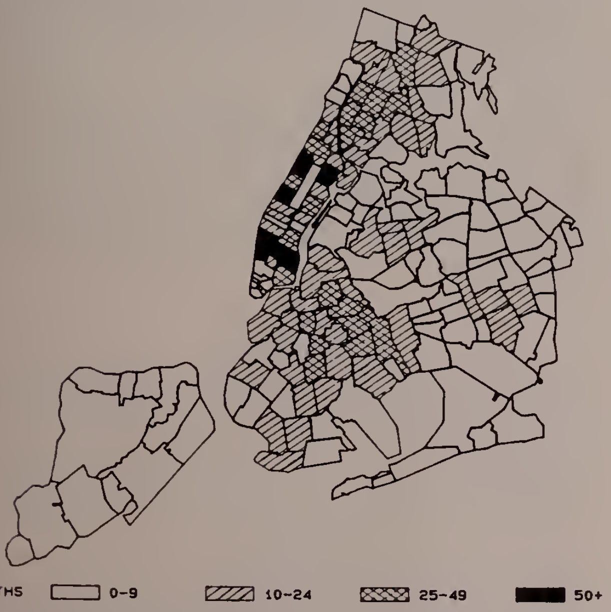

FIGURE 1. Geographic distribution of AIDS mortality in New York City, 1986, by zip code of residence.

In the six-year period analyzed, Manhattan has been affected most severely. The cumulative incidence of AIDS in this borough is more than 2.5 times that of the next highest, the Bronx. Furthermore, transmission patterns in these boroughs differ dramatically. The early and large impact among homosexual men in Manhattan—accounting for 75% of all cases in that borough—has persisted through 1986. Similarly, IV drug use has consistently accounted for 22% of Manhattan cases. These proportions are nearly reversed in the Bronx.

The high seroprevalence of HIV and incidence and prevalence of AIDS among homosexual men in specific areas of Manhattan indicates the continuing need for health care, psychological support services, and education for those affected, as well as for preventive education for new entrants to this population. The reason for the proportional increase in cases among homosexual men in Brooklyn and Queens is unclear, but suggests a growing need for specific services in these boroughs, particularly if individuals are not being reached by agencies located in Manhattan.

The need to reach the Hispanic and black population is especially clear from the figures (Table I) which indicate that incidence rates in these groups exceed those among whites in all boroughs except Manhattan. Moreover, compared to whites, Hispanics and blacks, particularly women, are at increased risk of dying of AIDS.[6] In these racial/ethnic groups AIDS is also disproportionately transmitted by the sharing of intravenous drugs, and IV-drug-related AIDS accounts for the largest proportion of cases in the Bronx, Brooklyn, and Staten Island. Narcotics-related AIDS mortality is especially high in Morrisania and Mott Haven in the South Bronx, and in Williamsburg, Brownsville, and Bushwick in Brooklyn (Table VI). The need for greatly expanded services for intravenous drug users has been widely emphasized.[5,8]

Intravenous drug use is also the principal source of direct infection in women, and of indirect infection through heterosexual contact with male drug users.[9] Because of the geographic and racial/ethnic patterns of IV drug use, black and Hispanic women constitute the large majority of victims of heterosexually acquired AIDS in all boroughs. Furthermore, children born to black and Hispanic women constitute more than 90% of the pediatric AIDS cases in New York City.[10] Clearly, services for current IV drug users and potential future users should be sensitive to these demographic features and trends.[8,9]

City and community services for persons with AIDS and those at increased risk for AIDS have expanded considerably in NYC since the recognition of this epidemic. The current analysis suggests the important role epidemiologic surveillance can play in providing a detailed demographic picture of AIDS, and how this information can assist in monitoring the impact and need for support and preventive services.

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HIV infection among young adults in the New York City area*

Prevalence and incidence estimates based on antibody screening among civilian applicants for military service

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ABSTRACT. Since October 1985, the Department of Defense has screened civilian applicants for military service for antibody to the human immunodeficiency virus (HIV). Among young adult applicants from 15 New York and New Jersey counties in the New York City region, the overall seroprevalence rate was 7.1 per 1,000 (314/44,139). Seroprevalence rates were higher among men than women (male-female ratio, 1.4:1) and higher among black/non-Hispanic applicants than applicants from other racial/ethnic groups, and they increased almost linearly with age from the late teens through the twenties. Male-specific prevalence rates exceeded 0.5% in nine of the 15 counties, while female-specific prevalences exceeded 0.5% in seven. In Manhattan, the Bronx, and Brooklyn, there were geographic foci associated with significantly increased seroprevalences. Despite potential biases, applicant screening data provide unique insights into the current state and the dynamics of the HIV infection epidemic among young adults. Differences of infection risk based on demographic and geographic factors provide empirical bases for allocating prevention, intervention, and clinical services in the region.

(NY State J Med 1988; 88:232-235)

Although it has been approximately six years since end-stage human immunodeficiency virus (HIV) infection was first reported in the New York area, the HIV infection epidemic is at least twice as old in the region. Because of the long and variable period from the time of infection to clinical presentation of end-stage disease, and because the infection epidemic has spread beyond its initial urban epicenters and behaviorally defined risk groups, acquired immunodeficiency syndrome (AIDS) case reports do not reflect the current state of the HIV infection epidemic. Serial prevalence surveillances, based on broad geographic and demographic sampling, are required to assess current infection prevalences and the dynamics of the infection epidemic.

Since October 1985, the Department of Defense has screened more than 1.2 million civilian applicants for military service for antibody to HIV. In this report, data from the applicant screening program are summarized to estimate the concentration and the rate of spread of the virus among young adults living in the New York City metropolitan region.

METHODS

Screening Program and Algorithm. The applicant screening program and the algorithm used to determine antibody positivity have been previously described.[1,2] Briefly, blood is drawn from each applicant at the time of the routine preinduction medical examination. Sera are sent to a single contracting laboratory for analysis. All sera are initially tested by enzyme-linked immunosorbent assay (ELISA). Specimens that are initially ELISA positive are retested in duplicate. Specimens that are repeatedly ELISA positive are further analyzed using the Western blot method. Specimens with either a gp41 band or multiple other HIV-specific bands are considered antibody positive. Applicants who submit specimens that are repeatedly ELISA and Western

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blot positive are notified and asked to submit a second specimen for repeat Western blot analysis. An applicant is therefore considered antibody positive when sera are repeatedly ELISA positive on a single specimen and repeatedly Western blot positive on duplicate specimens. For this analysis, applicants who declined to submit a second specimen were considered antibody positive after a single specimen was repeatedly positive on ELISA and Western blot screening.

New York Area Applicant Population. During the first 21 months of screening, there were 44,139 applicants for military service from the New York metropolitan region—Bronx, Kings, Nassau, New York, Queens, Richmond, Suffolk, and Westchester counties in New York; Bergen, Essex, Hudson, Middlesex, Monmouth, Passaic, and Union counties in New Jersey. Applicants were young adults (44% younger than 21 years; 81% younger than 26 years); predominantly male (86%); and represented the following racial/ethnic groups: black, non-Hispanic (40%), white, non-Hispanic (40%), Hispanic (10%), and other (10%).

RESULTS

Prevalence Estimates

Overall and Sex-Specific Prevalences. Among all applicants, the seroprevalence rate was 7.1/1,000 (314/44,139). The prevalence was 7.4/1,000 (280/37,838) among males and 5.4/1,000 (34.6/6,301) among females. The overall male-to-female prevalence ratio was 1.4:1. Figure 1 shows sex-specific prevalences and male-to-female prevalence ratios during each of the first seven calendar quarters of routine applicant screening.

Race/Ethnicity. Of six subgroups defined by race/ethnicity (black/non-Hispanic, white/non-Hispanic, Hispanic/other) and gender, black males had the highest overall seroprevalence rate (12/1,000; 162/14,008). Black females had the next highest prevalence (7.5/1,000; 28/3,732). Prevalence rates among white males and white females were relatively low: 3.7/1,000 (60/16,225) and 1.8/1,000 (3/1,626), respectively. Prevalence rates among Hispanic/other applicants were intermediate: males, 6.7/1,000 (58/8,605) and females, 3.2/1,000 (3/943). The male to female prevalence ratio was lower among black applicants than among white or Hispanic/other applicants (Fig 2).

Age. Seroprevalence rates consistently increased with age from the late teens through the twenties. Age-prevalence trends were similar among males and females, and male-to-female prevalence ratios were less than 2:1 in each age group (Fig 3). Seroprevalence rates were low among applicants younger than 21 years, regardless of race/ethnicity or gender. However, throughout the twenties, prevalence increased sharply among black applicants, more gradually among white applicants; and at an intermediate rate among Hispanics/others (Fig 4).

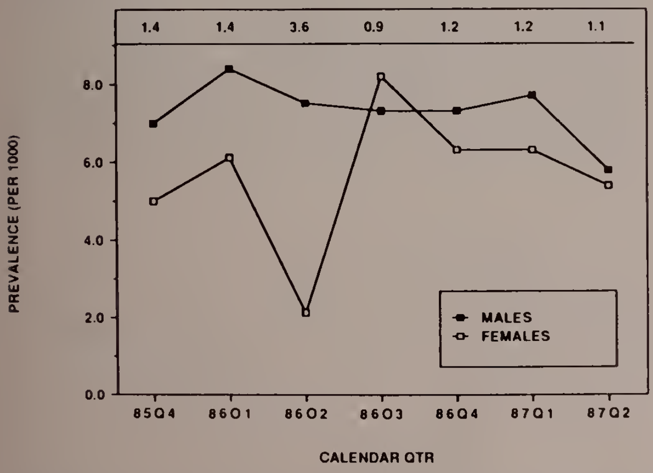

FIGURE 1. Overall seroprevalence rates among male and female applicants from the New York City metropolitan area, October 1985–June 1987, by calendar quarters. Male-to-female prevalence ratios are also indicated.

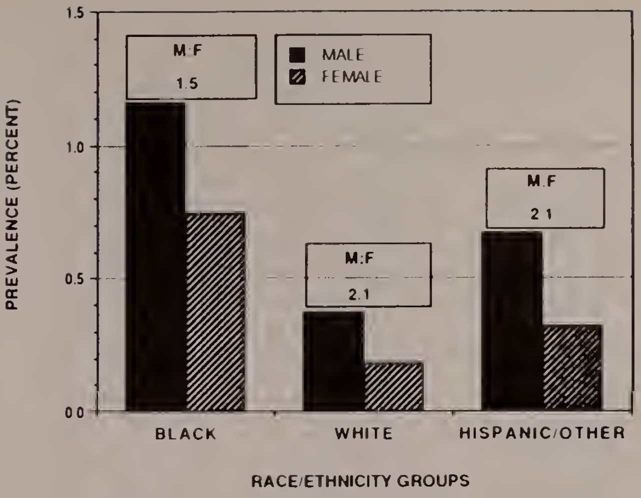

FIGURE 2. Overall seroprevalence rate among New York City metropolitan area applicants for military service, October 1985–June 1987; by sex and race/ethnicity defined subgroups.

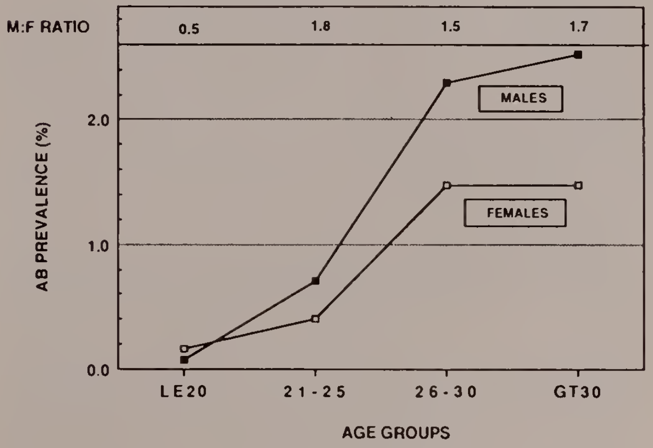

FIGURE 3. Relationship of seroprevalence with age, for male and female military applicants from the New York City-New Jersey metropolitan area. Male-to-female prevalence ratios are also indicated.

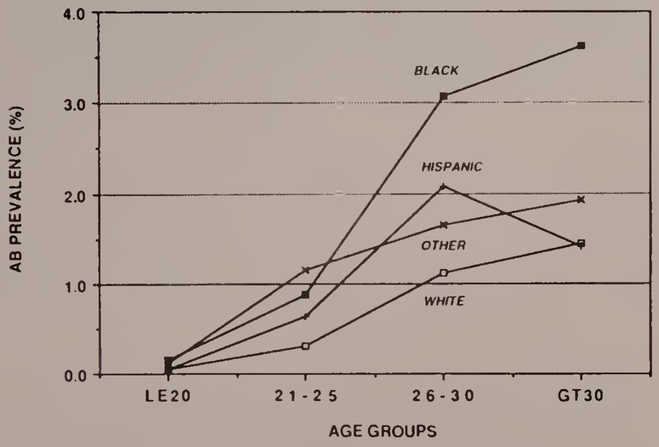

FIGURE 4. Relationship of seroprevalence with age, for race/ethnicity defined subgroups from the New York City area.

TABLE I. Results of HIV Antibody Screening Among Male and Female Applicants for Military Service from 15 New York and New Jersey Counties in the New York City Metropolitan Region, October 1985–June 1987

	Males (per 1,000)	Females (per 1,000)
Bronx	52/4,124 (12.6)	6/ 790 (7.6)
Kings	66/6,091 (10.8)	7/1,198 (5.8)
Nassau	13/2,854 (4.6)	4/ 419 (9.5)
New York	47/2,733 (17.2)	5/ 445 (11.2)
Queens	22/4,606 (4.8)	4/ 784 (5.1)
Richmond	0/ 730 (0.0)	0/ 102 (0.0)
Suffolk	4/3,882 (1.0)	0/ 517 (0.0)
Westchester	5/1,641 (3.0)	0/ 277 (0.0)
Bergen	3/1,508 (2.0)	0/ 152 (0.0)
Essex	18/2,457 (7.3)	6/ 583 (10.3)
Hudson	16/1,507 (10.6)	0/ 223 (0.0)
Middlesex	8/1,423 (5.6)	0/ 160 (0.0)
Monmouth	10/1,581 (6.3)	0/ 229 (0.0)
Passaic	7/1,260 (5.6)	2/ 204 (9.8)
Union	9/1,441 (6.2)	0/ 218 (0.0)

Geographic Distribution (by County). Table I shows county-specific prevalence rates among male and female applicants. In Manhattan (New York County), prevalence rates exceeded 1% among both males and females. In the Bronx and Brooklyn (Kings County), prevalence rates exceeded 1% among males and 0.5% among females. In Essex and Passaic counties in New Jersey, prevalence rates exceeded 0.5% among both male and female applicants. Overall, male-specific prevalence rates exceeded 0.5% in nine of the 15 counties, and female-specific prevalences exceeded 0.5% in seven of the 15 counties.

Location (by Zip Codes): The New York City Health Department recently reported zip codes in Manhattan, Bronx, and Brooklyn that were associated with increased AIDS incidence during the period 1981–1984.[3] We assumed that areas defined by these zip codes had been affected by the HIV infection epidemic relatively longer than other areas of the city. The prevalence rate among applicants from AIDS-associated zip codes significantly exceeded the prevalence rate among applicants from other zip codes in the same high prevalence counties (1.52% [118/7,768] versus 0.85% [65/7,613]; $\chi^2 = 14.47$; one degree of freedom; $p < 0.001$).

Infection Rate Estimates

Prevalence-Age Regression Method: Since antibody to HIV is a persistent marker of HIV infection, the seroprevalence rate in a sample of a population provides an estimate of the cumulative HIV infection incidence in that population. Since seroprevalence rates among applicants increase almost linearly with age in the applicant age range, the regression slope approximates the average increase in cumulative infection incidence with each additional year of age. Therefore, the slopes of prevalence-age regression lines provide estimates of annual HIV infection rates among the young adults in the region who are represented by military applicants.

Figure 5 shows age-specific prevalences for black, Hispanic/other, and white male applicants (for birth year defined groups, approximately aged 18 to 25 years). For each race/ethnicity group, regression lines were fitted to two sets of data collected during two equivalent periods exactly one year apart: October 1985–August 1986 and October 1986–August 1987. The slope of the regression line estimates the average number of new HIV infections per 1,000 persons per year (an incidence rate).

Within each racial/ethnic group, the slopes of age-prevalence regression lines from the two sampling periods were comparable. For black males, the prevalence rates by age regression slopes were 2.65/1,000/year and 2.84/1,000/year. In addition, among black males, birth-year-specific prevalence rates clearly increased between the 1985/86 and the 1986/87 sampling periods (demonstrated by the increase on the prevalence scale of the 1986/87 regression line compared with the 1985/86 line; Fig 5). For Hispanic/other males, the regression slopes were 1.53/1,000/year and 1.68/1,000/year. For white males, the regression slopes were 0.97/1,000/year and 0.78/1,000/year.

Estimated incidence of infection rates based on prevalence by age regression differed markedly among different racial/ethnic groups. Using the estimated rate among white males as the referent, infection rates were approximately 1.8 times higher among Hispanic/other males and 3.1 times higher among black males.

DISCUSSION

Applicants for military service are not randomly selected from the young adults in the New York area, so interpretations of data require consideration of the potential effects of selection bias. Young adults apply for military service subject to institutional and personal criteria. In general, selection factors that are constant over the period of screening and consistent for all age groups (eg, eligibility criteria for military enlistment) define a subgroup of the general population. Parameter estimates based on screening this subgroup provide biased estimates of general population parameters, depending on the strength of the associations of the selection factors and HIV infection risk. In addition, selection factors that change over time or that differ by age (eg, self-selection criteria) additionally bias estimates of time-dependent parameters such as incidence rates and temporal/age trends, even within the subpopulation of military applicants.

Since applicants for military service are self-selected, the consistency of selection criteria is difficult to assess. However, it is presumed that young adults are relatively unlikely to apply for military service if they know of the

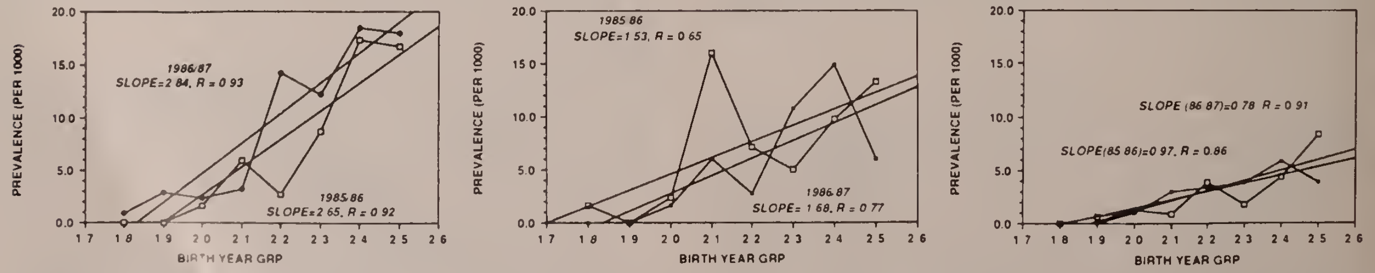

FIGURE 5. Relationships of seroprevalences and age (defined by birth year) are shown for data collected during two screening periods a year apart in time: October 1985–August 1986 and October 1986–August 1987. Slopes and regression coefficients (R) are indicated for prevalence by age regressions for each of the sampling periods. Slopes estimate the average number of new infections per 1,000 persons per year of increasing age. Slopes are consistent within, but significantly vary among, race/ethnicity defined groups from the New York City metropolitan area. Left, black males; center, Hispanic and other males; right, white males.

Department of Defense's applicant screening program and either know or suspect that they are antibody positive.

During the two-year period of applicant screening, awareness of the Department of Defense screening program has almost surely increased among young adults living in urban epicenters of the AIDS epidemic. In addition, millions of young adults have been screened for antibody to HIV through blood donor, alternate test site, and other screening programs. Undoubtedly, many potential military applicants in the New York area were screened through such programs over the past two years. Finally, educational and mass media programs are now directed at broad segments of communities. Consequently, over the past two years, many young adults in the New York area have acquired the information necessary to accurately assess their own infection risk.

Therefore, it seems likely that self-exclusion of infected and high risk young adults has increased over the period of military applicant screening. If so, prevalence and incidence rate estimates that are based on applicant screening data will underestimate actual rates in the New York City region.

While prevalence by age regression is a simple method for estimating annual infection rates, it has at least one significant limitation. The method estimates the infection rate based on the lifelong exposure of applicants to HIV infection risk. Since the dynamics of the epidemic have evolved throughout its course, the regression method will underestimate the current infection rate if the current rate is higher than the average of the annual rates during the preceding years of the epidemic—a situation that seems likely.

In summary, potential biases based on selection factors and the changing dynamics of the epidemic probably result in estimates of prevalence and incidence rates that are lower than the actual population values.

Without question, the epidemiology of the HIV infection epidemic has changed in the period since patients now affected with end-stage HIV disease were infected with the virus. In spite of their limitations, military applicant screening data provide unique insights into the current state, as well as the recent dynamics, of the HIV infection epidemic in the New York area.

The infection epidemic has spread widely throughout the region. Yet there appear to be geographically circumscribed foci of particularly high and low infection transmission. For example, within Brooklyn, the Bronx, and Manhattan, zip codes associated with increased AIDS incidence in 1981–1984 (and therefore with HIV infection transmission for at least a decade) still define geographic areas of particularly high infection prevalence among young adults. In contrast, there are geographic areas where young adults represented by military applicants remain relatively unaffected by the epidemic (eg, Staten Island [Richmond County], New York). Identification of geographic areas with particularly high and low infection transmission rates provides opportunities to target prevention, intervention, and clinical services, and to further define sociodemographic and behavioral determinants of the current infection epidemic.

In contrast to the extreme male predominance among reported AIDS patients in the country, ratios of seroprevalence rates among male and female applicants were generally less than 2:1. While gender specific selection factors may result in underestimates of actual male-to-female infection ratios, these and other data[4] document the significant impact of the infection epidemic on young females in the region. The current involvement of young females of child bearing age in the infection epidemic has important implications regarding the futures of both the adult and the pediatric AIDS epidemics in the region.

Finally, seroprevalence rates were relatively low among teenagers, regardless of their gender or race/ethnicity, and they increased almost linearly from the late teens through the twenties, most strikingly among members of racial/ethnic minorities. These findings define opportunities and responsibilities for aggressive intervention efforts. HIV-related knowledge, attitudes, and behavioral skills that are critical during periods of greatest infection risk must be acquired by young men and women during the relatively lower risk, middle teen years, particularly in demographic subgroups in geographic areas with the highest rates of HIV infection incidence.

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The epidemiology of AIDS in New Jersey*

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ABSTRACT. The epidemiology of acquired immunodeficiency syndrome in New Jersey is presented for the period 1982-1986. Additional data are also presented for 1987. A total of 1,728 nonincarcerated adult AIDS cases were diagnosed in the period 1982-1986. The largest number of cases occurred in the counties that contain sizable urban populations. The proportion of cases among females is very high in New Jersey, with most of them occurring in those areas of the state near New York City. Intravenous drug abuse is the risk factor most directly associated with AIDS in the high incidence metropolitan areas of New Jersey.

(NY State J Med 1988; 88:236-239)

New Jersey has the fifth largest number of cases of acquired immunodeficiency syndrome (AIDS) in the nation, following New York, California, Florida, and Texas. As of December 31, 1987, 3,257 cases of AIDS had been reported to the New Jersey State Department of Health. When AIDS case rates are considered, New Jersey ranks third (as of November 2, 1987), with a case rate of 346 per million population, compared to a case rate of 693 for New York and 372 for California.[1] New Jersey is unique in that the majority of AIDS patients in the state have a history of intravenous drug abuse. Probably related to this is the fact that New Jersey has the highest percentage of female AIDS cases in the nation, a large number of children with AIDS, and a large number of AIDS patients who are incarcerated.

New Jersey has always somewhat gravitated around its neighboring two large cities, New York and Philadelphia. Even the AIDS epidemic shows different patterns around New York and Philadelphia, perhaps to some extent reflecting the demographics of the AIDS epidemic in these cities. Some of these differences will be reflected in the data shown here.

METHODS

A number of years ago, the New Jersey State Department of Health administratively divided the state's 21 counties into four districts, three with five counties each and the fourth with six counties. While these administrative districts are no longer used, they provide a means of dividing the state for the purposes of analysis. Such a division gives a picture of the geographic differences of AIDS in New Jersey and eliminates the analytical problems that arise because of the relatively small number of cases in some of the counties.

As shown in Figure 1, the metropolitan district consisted of the counties immediately surrounding New York City; the northern district contained the area in northwestern New Jersey that used to be largely rural, but now is rapidly becoming suburban; the southern district consisted of all of the south of New Jersey, including rural, suburban and urban areas, much of it in the Philadelphia metropolitan area; and the central district was the region in the center of the state, with areas that can be considered in either the New York or the Philadelphia orbit. Both the central and southern districts contained the New Jersey shore.

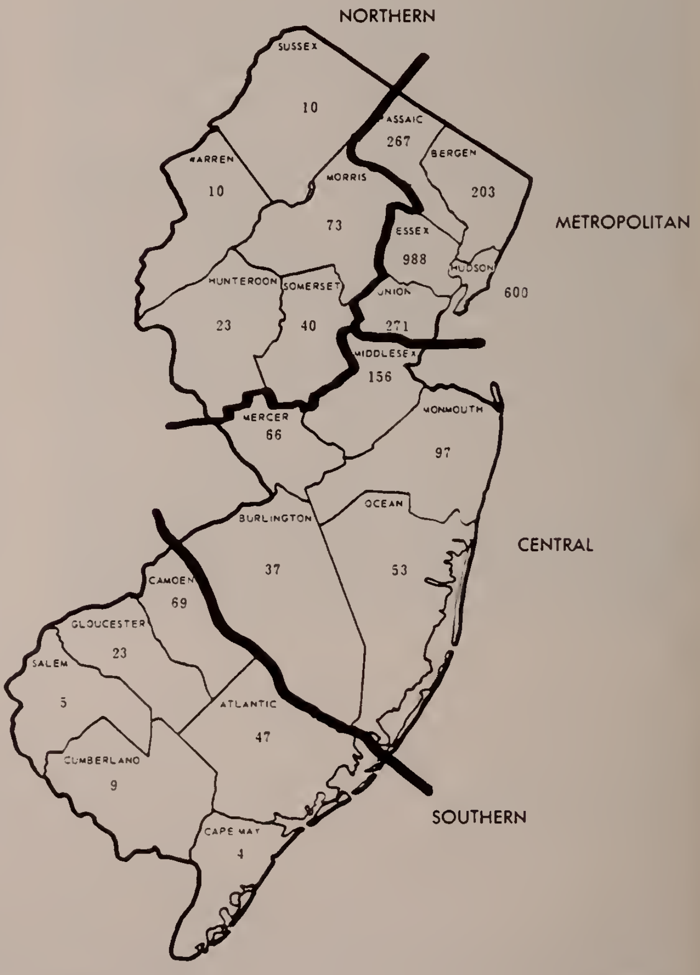

FIGURE 1. AIDS cases in the State of New Jersey as of December 31, 1987.

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TABLE I. Cumulative AIDS Cases by County, New Jersey, Through December 31, 1987

County	Population	No. of Cases	Rate per 1,000,000
Atlantic	194,119	47	242
Bergen	845,385	203	240
Burlington	362,542	37	102
Camden	471,650	69	146
Cape May	82,266	4	49
Cumberland	132,866	9	68
Essex	851,304	988	1,161
Gloucester	199,917	23	115
Hudson	566,972	600	1,077
Hunterdon	87,361	23	263
Mercer	307,863	66	214
Middlesex	595,893	156	262
Monmouth	503,173	97	193
Morris	407,630	73	179
Ocean	346,038	53	153
Passaic	447,585	267	597
Salem	64,676	5	77
Somerset	203,129	40	197
Sussex	116,119	10	86
Union	504,094	271	538
Warren	84,429	10	118
Total	7,365,011	3,257	442

TABLE III. AIDS Cases by Year and Gender, New Jersey, 1982–1986

	Male		Female	
Year	No.	%	No.	%
1982	55	84.6	10	15.4
1983	146	88.0	20	12.0
1984	283	85.5	48	14.5
1985	456	82.8	95	17.2
1986	608	82.1	133	21.9

Most of the analyses presented in this paper are for the period 1982 through 1986. These data would include virtually all of the reported AIDS cases diagnosed during that period. Presenting the data for this period eliminates the problem of the small numbers of cases in the years prior to 1982, which do not give meaningful time trend data, and, since all of the cases diagnosed in 1987 would not have been reported by the time of preparation of this paper, 1987 cannot be included in time trend data. In addition, the case definition of AIDS changed during 1987, so that the case data for this year are not comparable to previous years, at least until the older data are updated to the new case definition. However, where appropriate, data are also presented which include all cases reported through 1987.

RESULTS

The largest cumulative numbers of AIDS cases reported through 1987 were reported from the counties near New York City (Fig 1). The numbers in each county do not include the people with AIDS who were incarcerated at the time of the case report, many of whom were from the counties with large numbers of AIDS cases. Epidemiologically, it is more meaningful to look at incidence rates (Table I). Essex and Hudson counties, which contain the cities of Newark and Jersey City, respectively, each had a cumulative incidence rate of over 1,000 per million population, while Passaic and Union counties, which contain the cities of Paterson and Elizabeth, respectively, each had a cumulative incidence rate of over 500 per million. There was not much difference in incidence among the 17 other New Jersey counties, although a few of the counties had relatively low rates.

Of the 1,728 nonincarcerated adults with AIDS diagnosed in the period 1982–1986, 77.0% came from the metropolitan district (Table II). Overall, 16.5% of the adults with AIDS were female. There was a marked difference in the percentage of female cases in the various districts, with the largest percentage in the metropolitan district, the lowest percentages in the northern and southern districts, with the central district being intermediate, a trend generally present in the cumulative case rates. The cumulative case rates in males were essentially the same in the northern, central and southern districts, which is similar to the lack of difference in case rates among the counties noted above, except for four counties in the metropolitan district. There was an increase in the number and percentage of female cases in the later years (Table III). It should be noted that in the cumulative case data through 1987, the percentage of female patients is up to 20%, which is further evidence that the percentage of female AIDS cases in New Jersey is continuing to rise.

The northern and southern districts tended to follow the national pattern of risk group distribution of AIDS cases, with approximately three-quarters of the cases occurring in homosexual or bisexual men (Table IV). On the other hand, the metropolitan district showed slightly more cases among intravenous drug abusers compared to homosexual-bisexual men, with the central district showing an intermediate pattern between the two types of risk group distribution. The overwhelmingly larger number of AIDS cases in the metropolitan district, compared to the other three districts, resulted in a high percentage of AIDS cases in intravenous drug abusers in the state as a whole. A similar analysis by district is not presented for adult females because of the very small number of female AIDS cases in the northern and southern districts.

Tables V and VI present the risk group percentages by year for adult males and females respectively. Among adult males, there was very little change in the percentage of cases in the different risk groups over the years, the exception being a marked decline in the percentage reporting both homosexual activity and intravenous drug abuse (Table V). Among adult females, there was a marked increase in the percentage of cases reporting heterosexual contact with a person at high risk for HIV infection. Some of this increase came about because of a drop in the cases classified as undetermined, so there is some question as to whether or not all of the increase in heterosexual contact cases represented a real change.

Looking at AIDS case data only gives information about HIV infection that largely occurred several years ago. More up-to-date information on the current status of HIV infection is provided by serosurveys for HIV infection performed in various populations, al-

TABLE II. AIDS Cases by District and Gender, New Jersey, 1982–1986

	Number of Cases			Percent by Gender			Rate per 1,000,000 Population		
District	Male	Female	Total	Male	Female	Total	Male	Female	Total
Metropolitan	1,075	255	1,330	80.8	19.2	100.0	708	151	415
Northern	77	8	85	90.6	9.4	100.0	175	17	95
Central	182	29	211	86.3	13.7	100.0	177	27	100
Southern	93	9	102	91.2	8.8	100.0	170	15	110
Incarcerated population	121	5	126	96.0	4.0	100.0	—	—	—
Total	1,548	306	1,854	83.5	16.5	100.0	438	80	252

TABLE IV. AIDS Cases (%) by Risk Group and District, Adult Males, New Jersey, 1982–1986

Risk Group	Metropolitan District	Northern District	Central District	Southern District	Incarcerated Population
Homosexual/bisexual	40.0	80.5	49.5	67.7	1.7
Intravenous drug user, heterosexual	43.5	11.7	30.8	14.0	88.4
Intravenous drug user, homosexual	7.5	0	7.7	8.6	7.4
Hemophiliac	0.7	2.6	4.4	4.3	0
Heterosexual contact	3.2	0	1.6	0	0.8
Transfusion	1.3	3.9	2.2	2.2	0
Undetermined	3.0	1.3	3.8	3.2	1.75
Total number of cases	1,075	77	182	93	121

though one-time serosurveys do not give information about the incidence of HIV infection. The New Jersey State Department of Health is currently involved in doing or planning a variety of HIV serosurveys. One of these surveys is nearing completion, a survey of specimens submitted to the State Health Department's laboratory for syphilis testing from sexually transmitted disease clinics throughout New Jersey. As might be anticipated, preliminary results are showing high HIV prevalence in specimens from those counties where there is a high incidence of AIDS cases, with lower seroprevalence rates from the remainder of the counties.

DISCUSSION

The New Jersey AIDS cases show different patterns of occurrence even within the confines of a physically small state. The largest numbers of AIDS cases are occurring in the counties that both contain sizable urban areas and are near New York City. These counties also have the highest cumulative AIDS incidence rates, with much lower case rates in most of the remaining counties in the state. The percentage of female cases is very high in New Jersey, a percentage that appears to be growing. Again, most of the female AIDS cases are also occurring in the portion of New Jersey near New York City.

In most of New Jersey, the risk group distribution of AIDS cases among men is similar to the national distribution, with about three-quarters of the cases occurring in homosexual or bisexual men. However, in the area of the state with by far the largest number of cases, the male cases are almost equally divided between homosexual-bisexual men and intravenous drug abusers. Among women, most of the cases are occurring in intravenous drug abusers, making intravenous drug abuse the single most important AIDS risk behavior in New Jersey. In addition, a growing percentage of the AIDS cases in women in New Jersey is related to heterosexual contact with a person at high risk for HIV infection. In most such cases, the risk behavior of the contact is intravenous drug abuse.

Thus, the key to much of the AIDS problem in New Jersey is intravenous drug abuse. Intravenous drug abuse is the risk behavior most directly associated with AIDS in the high incidence metropolitan area of New Jersey. It is responsible for the very high incidence of AIDS in women in the state, both directly in drug-abusing women, and indirectly in women who are sexual partners of drug-abusing men, and is also responsible for the high incidence of AIDS in children born to HIV-infected women. It is also almost the only behavior responsible for the very large number of incarcerated persons with AIDS in the state.

Clearly, the surveillance data on the AIDS cases point to prevention of the spread of HIV through prevention of intravenous drug abuse as the primary means to slow the spread of the AIDS epidemic in New Jersey. This is necessary to diminish the already present epidemic of AIDS in the cities of the metropolitan area of the state, to do something about the increasing numbers of cases in women and children, and perhaps to prevent the areas of the state not currently as badly affected by the epidemic from soon resembling the metropolitan district. The group we need to

TABLE V. AIDS Cases (%) by Risk Group and Year, Adult Males, New Jersey, 1982–1986

Risk Group	1982	1983	1984	1985	1986
Homosexual/bisexual	36.4	37.0	40.3	43.6	43.2
Intravenous drug user, heterosexual	38.2	43.8	39.9	43.6	42.5
Intravenous drug user, homosexual	16.4	11.0	10.2	4.8	5.9
Hemophiliac	0	0.7	1.4	2.2	1.2
Heterosexual contact	7.2	1.4	2.8	2.0	2.7
Transfusion	0	1.4	0.7	1.5	2.0
Undetermined	1.8	4.8	4.6	2.2	2.5
Total number of cases	55	146	283	456	608

TABLE VI. AIDS Cases (%) by Risk Group and Year, Adult Females, New Jersey, 1982–1986

Risk Group	1982	1983	1984	1985	1986
Intravenous drug user	90.0	65.0	62.5	63.2	60.2
Heterosexual contact	10.0	20.0	16.7	26.3	32.3
Transfusion	0	5.0	8.3	3.2	4.5
Undetermined	0	10.0	12.5	7.4	3.0
Total number of cases	10	20	48	95	133

reach most are those who have not yet become intravenous drug abusers, particularly people in their teenage years. Until such time as there is an effective vaccine or chemoprophylaxis, we must learn effective methods of preventing people from becoming intravenous drug abusers.

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Effectiveness of distribution of information on AIDS

A national study of six media in Australia

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ABSTRACT. Differential use of media for providing AIDS information and attitudes toward AIDS were assessed in a geographically stratified proportional sample of 2,601 adults in all states and territories in Australia. Data indicated that exposure to media information, regardless of type, was associated with lower levels of fear of homosexual persons and less fear of death, as well as lower levels of social conservatism. Those who obtained AIDS information from health care workers were found to have fewer unrealistic concerns. Individuals who were more homosexual in expressed sexual identity and those with other at-risk behaviors tended to get information more frequently from friends and pamphlets or posters, and those in higher occupational levels and those with high levels of personal and social concerns about AIDS tended to get their information from friends and health care workers. Electronic media were not utilized more frequently by those with at-risk behaviors. These results suggest that greater emphasis should be placed on more informal sources of information for those most at risk of HIV infection, and that the public media convey little advantage in providing information to such target groups.

(NY State J Med 1988; 88:239–241)

The spread, or potential for spread, of acquired immunodeficiency syndrome (AIDS) has instigated major media campaigns in an attempt to modify behaviors that place individuals at risk of infection with the human immunodeficiency virus (HIV), the causative agent of AIDS. Such risk behaviors include unprotected sexual intercourse, both heterosexual and homosexual, and sharing of needles for the administration of intravenous drugs. Media campaigns have been the most common form of attempting to reach the general population,[1] while pamphlets and brochures have commonly been utilized to motivate change among homosexual and bisexual men.[2]

There is little if any published research that looks at the attitudes toward AIDS held by those who utilize various media, and whether there is a differential use of particular media by those most at risk of HIV infection. While Temoshok, Sweet, and Zich[3] found that the level of knowledge about AIDS was negatively correlated with fear of AIDS and anti-homosexual attitudes in a three-city sample, their data were based on a small sample of convenience, and its generalizability is uncertain.

METHODS

We assessed both the attitudes toward AIDS, and the differential use of media by those whose behaviors potentially put them at risk of HIV infection, as part of a national study on AIDS knowledge, attitudes, and risk behaviors in all states and territories of Australia. We noted in a related publication utilizing these data that a higher level of knowledge about AIDS was significantly related to the use of pamphlets or posters as a media source and that in some cases obtaining information from public media lead to a *lower* level of accurate knowledge about AIDS.[4]

Three research questions were asked in order to determine which media were likely to be the most effective in reaching those individuals most in need of information about AIDS: First, do attitudes toward AIDS differ according to the media from which individuals report having received their information about AIDS? Second, what demographic variables differentiate those who report receiving their information about AIDS from the various media? And third, do those whose behavior places them at greater risk of HIV infection receive their information about AIDS from particular media in preference to others?

The study was carried out on a geographically stratified, proportional sample of 2,601 individuals aged 16 years or over in all states and territories of Australia (population, 16 million). The sampling frame was provided by the Australian Bureau of Statistics on the basis of the most recent (1981) census data and was carried out in October–November 1986. Experienced market research interviewers employed by a national market research company visited the selected dwellings and carried out a face-to-face interview on knowledge of and attitudes toward AIDS. Selected census tracts were randomly generated from the state and urban-rural stratification requirements. Every third residence in the selected tracts was visited from a random start point. Respondents in each dwelling were selected by asking for the individual whose next birthday was nearest the date of the visit. Only individuals over 16 years of age were eligible to participate.

The questions and scales measuring attitudes toward AIDS have been described elsewhere.[5] Four additional questions were

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scored on a six-point Likert scale (the first two questions were defined at the poles by the terms "not at all concerned" and "extremely concerned"): for example, "How concerned are you about AIDS as a social issue in Australia (personally)?" A further two questions—"Do you personally know any homosexual people (intravenous drug users)?"—were scored "yes, unsure, no." Demographic data included age, state or territory, occupation, place of residence (city, town, rural), position on the Kinsey scale of sexual identity,[6] and marital status. Media sources of AIDS information (scored "yes," "no") were television, radio, newspapers and magazines, posters and pamphlets, friends, and health professionals.

At the conclusion of the 15-minute interview, an anonymous questionnaire with a reply-paid envelope was left with the respondent. The questionnaire requested yes/no responses for the following items (separate items for "ever" and "in the past 12 months"): homosexual contact, heterosexual contact, use of intravenous drugs, contact with prostitutes, and blood transfusion from 1980 to 1985. The surveyors made one return visit to interview respondents who were not at home at the time of the initial call.

The anonymous risk behavior questionnaire was matched by code number with the data from the face-to-face interview. Data analysis consisted of computing t-tests (two-tailed) and χ^2 tests (for interval/ratio and ordinal data, respectively) between those who responded "yes" and "no" to the six sources of information, with significance set at the 0.05 level.

RESULTS

Risk behavior questionnaires were returned by 60.2% of the interview subjects. Twenty-one individuals had not heard of AIDS, and their face-to-face interviews were terminated after the demographic data were collected. Results of the sampling appear in Table I. In comparison with the census data, the sample appeared to be weighted toward females, and to slightly underrepresent those younger than 24 years of age and slightly overrepresent those aged 25 to 44 years. Unmarried females also appeared to be slightly underrepresented. The marginal additional weighting given to South Australia is a result of the study's having been piloted in that state.

Comparison between the face-to-face interview results of those who did return the risk behavior forms and those who did not revealed only three significant differences at $p < 0.05$ (mean ± standard error given). Those who returned the forms were older (age 43.3 ± 0.05 vs 40.7 ± 0.05; $t = 3.9$, $p < 0.01$); had lower personal concern about AIDS on a six-point scale with the poles labelled "not at all concerned" and "extremely concerned" (3.1 ± 0.05 vs 3.4 ± 0.05; $t = 3.8, p < 0.01$); and were more likely to come from a lower occupational classification (classed on the Congalton[7] seven-point status ranking list of occupations in Australia (1 = highest status); $\chi^2 = 52.3$, degrees of freedom = 6, $p < 0.01$).

Results of the data analysis are summarized in Table II. The data reveal a number of differences among those who obtained their knowledge of AIDS from the six media sources (the "other" source was, without exception, the workplace). Relative importance of sources for information on AIDS was TV, 93.8%; newspapers and magazines, 89.7%; radio, 73.7%; friends, 38.3%; pamphlets and posters, 29.6%; health workers, 19.9%; and work, 9.3%.

The data in Table II illustrate that exposure to media information, regardless of type, was associated with lower levels of fear of homosexual persons and less fear of death, as well as lower levels of social conservatism. Those who obtained information from health care workers had a lower level of unrealistic concerns about AIDS, and those with who indicated higher levels of pity for people with AIDS tended to have received their information from radio and newspapers and magazines more frequently.

Individuals who were more homosexual in expressed sexual identity tended to get their information significantly more often from sources that are likely to include the homosexual subculture (friends and pamphlets/posters), and those in higher occupational classes tended to get their information from personal sources (friends and health care workers). Those with higher levels of personal and social concerns about AIDS also tended to get information from personal sources (friends and health care workers). For those engaged in behaviors that may be associated with increased risk of HIV infection, personal sources and specific informational sources such as pamphlets and posters were more frequently used for information. The electronic media (TV and radio) were not utilized more frequently by those engaging in risk-related behaviors than by those who were not.

TABLE I. Sample Characteristics by Comparison with the General Australian Population (%)

Variable	Census	Sample
State		
New South Wales	34.8	35.7
Victoria	26.2	24.8
South Australia	8.7	12.1
Queensland	16.1	14.7
West Australia	8.9	7.8
Australian Capital Territory	1.6	1.8
Northern Territory	1.0	0.8
Tasmania	2.8	2.3
Age (yr)		
15–19	10.9	6.6
20–24	11.4	9.0
25–29	10.9	12.3
30–34	10.7	13.7
35–39	10.0	10.6
40–44	7.8	8.7
45–49	6.7	6.7
50–54	6.3	6.3
55–59	6.3	5.8
60–64	5.8	7.0
65–69	4.6	5.2
≥70	8.6	8.3
Sex		
Male	49.8	40.6
Female	50.2	59.4
Marital Status		
Male		
Single	31.4	30.4
Married	62.7	62.0
Separated, widowed, divorced	5.9	7.6
Female		
Single	23.2	17.2
Married	61.7	64.7
Separated, widowed, divorced	15.1	18.1

DISCUSSION

It is difficult to see how the differences between those who did return the risk questionnaires and those who did not might systematically bias the data. Kinsey, Pomeroy and Martin[6] have noted, however, that sexual contact occurs consistently earlier in lower socioeconomic strata. Thus, the data on those engaging in risk-related behaviors must be interpreted cautiously, as the prevalence of risk-related behavior is estimated from an incomplete sample.

Nevertheless, these data provide some useful information on the importance of various sources of information about AIDS. First, those who obtain information from a number of media sources appear to have more positive and less conservative attitudes toward AIDS, although socially conservative individuals appear to use television more frequently. Second, those with a history of engaging in risk-related behaviors generally tend to seek information from more specific sources such as pamphlets and posters

TABLE II. Characteristics Differentiating Those Utilizing Various Media Sources for Information about AIDS

Variable	TV	Radio	Newspapers	Media Source Pamphlets/ Posters	Friends	Health Care Workers
Attitudes						
Fear of homosexuals	−*	−		−	−	−
Fear of death		−		−	−	−
Lack of unreal concerns						+
Pity for people with AIDS		+*	+			
Fear of unknown	−					
Social conservatism	+	−		−	−	−
Demographics						
Kinsey scale				+[a]	+[a]	
Occupational class					+[b]	+[b]
Marital status			−[c]	+[d]	+[d]	−[c]
Sex (more males)	−	+				
Personal concern about AIDS			+	+	+	+
Social concern about AIDS					+	+
Know more homosexuals		+	+	+	+	+
Know more intravenous drug users	−		+	+	+	+
Risk behaviors						
Transfusion 1980–1985		−		−		+
Intravenous drug use						
Male homosexual sex				+	+	+
Male heterosexual sex						
Female homosexual sex	−					
Female heterosexual sex	+			+	+	+
Male prostitute contact						

* The notation "+" indicates that those scoring higher on the variable reported use of that particular source or medium significantly more often. The notation "−" indicates that those scoring lower on the variable reported use of that particular source or medium significantly more often.

[a] Those more homosexual higher than those more heterosexual.

[b] Those in classes 1–3 higher.

[c] Those separated lower than those married or single.

[d] Those single higher than those married or separated.

on AIDS, and from friends and health care workers. This suggests that these less public and more informal sources should be emphasized to provide information to those most at risk, and that the public media convey little advantage in reaching such target groups, though they do serve to alert the general public to the issue. Third, such personal sources as health care practitioners (as well as friends) are more commonly utilized by those in the higher occupational levels. These data are consistent with the finding of Sherr[1] that information in the public media is unlikely to alter significantly attitudes or knowledge in the desired direction, even after major public information campaigns about AIDS. Sherr[1] also reported that 57.2% of high risk and 65.2% of low risk individuals would turn to doctors or clinics for information on AIDS. However, the lack of significant differences among the various public media in the present study does not imply lower rates of use of these media; rather, their use appears to be equally common across all population groups. Nevertheless, Sherr[1] found that fewer than half of the respondents in his sample had read AIDS advertisements in the UK national campaign, and that there was no significant increase in knowledge following reading of such advertisements.

These data from a large, stratified sample of adults provide useful information on the sources for information on AIDS. They suggest that the public media (radio and TV) are significantly associated with attitudes toward AIDS, but that the more intimate and personal sources of information are selectively sought by those whose behaviors may place them at risk of HIV infection. Information campaigns should concentrate on educating health care workers and providing pamphlets and posters on AIDS for those who do not wish to raise questions personally. The public media (TV, radio, newspapers, and magazines) may be of more use in directing individuals toward sources of information than in actually conveying such information.

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The epidemiology of HIV in New York State*

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This examination of the epidemiology of acquired immunodeficiency syndrome (AIDS) by the New York State Department of Health (NYSDOH) in 1987 takes into account three factors. First, New York State has the largest number of reported AIDS cases of any state. As of October 28, 1987, there were 12,790 cases reported among New York State residents.[1] However, the prevalence of AIDS is not uniform throughout New York State communities and displays marked variation by age, sex, race, and geography. Further, the dominance of risk factors and the phase of the epidemic vary between regions within the state. New York State, excluding New York City, has approximately 10% of the total cases. However, this proportion still accounts for 1,181 cases, with an additional 432 cases occurring among prisoners housed in New York State Department of Correction facilities. If New York City cases were excluded, New York State would have the sixth highest number of cases among the 50 states.

The second critical factor in any consideration of AIDS epidemiology in New York State is that most of the information describing the epidemic has relied on reported AIDS cases. Reporting of AIDS cases may not in fact reflect the true number of cases, as described in a recent report by New York City Department of Health staff members on deaths of intravenous (IV) drug users.[2] Further, reported cases of AIDS represent only one part of the spectrum of disorders related to human immunodeficiency virus (HIV) infection. The lag time between HIV infection and the appearance of reportable cases, compounded by the largely unknown natural history of this infection, limits the value of relying on reports of disease to understand the distribution and extent of the epidemic. However, reliance on case reports alone will soon no longer be necessary. The capability now exists to determine the extent of HIV infection in the New York State population, particularly in a number of subgroups.

Another critical issue in regard to AIDS epidemiology in New York State is the validity of projections that forecast the future course of the epidemic. At NYSDOH, projections have been developed that rely on the past experience with AIDS as described by surveillance. This projection forecasts 46,000 cumulative cases in 1991.[3] The problem with this type of projection is that it is based on a static mathematical model reflecting past experience. It does not take into account the current or future dynamics of the epidemic, such as changes in the size of the pool of susceptible individuals or in the frequency or types of contacts between individuals.

ACQUIRED IMMUNODEFICIENCY SYNDROME IN NEW YORK STATE

At the beginning of the epidemic in 1982, the majority of AIDS cases in the United States were reported from New York State. By 1987, the proportion of national AIDS cases reported by New York State had declined to 30% (Fig 1), and this rate is projected to decrease to 17% by 1991. The rate of increase for the United States as a whole is now greater than that for New York State.

Figure 2 shows the number of reported cases of AIDS in adults in New York State and in the United States by month of diagnosis. Approximately 370 cases of AIDS are reported each month in New York State. Some 325 cases per month are reported by New York City and 45 are reported by upstate New York (Fig 3).[1] The ratio of cases reported by upstate New York in relation to New York City has remained constant (Fig 4). The increasing number of upstate counties with confirmed AIDS cases demonstrates the spread of this epidemic. By the end of 1986,

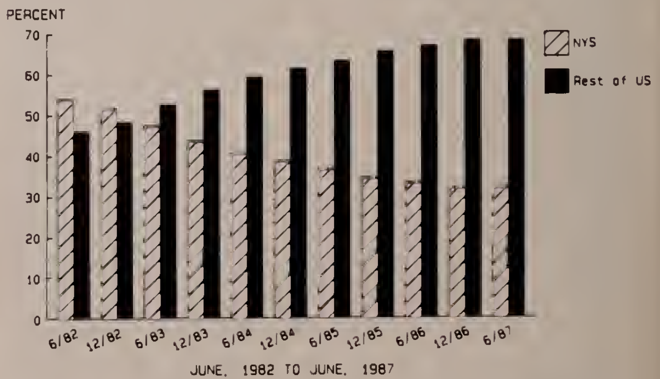

FIGURE 1. Percent of cumulative AIDS cases in the United States reported from New York State at six-month intervals, June 1982 through June 1987.[1]

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* This article is based on a presentation at the Second Montefiore Symposium on AIDS, "AIDS in the Tri-State Area: A Regional Portrait of the Epidemic," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held November 5, 1987, at the New York Academy of Medicine.

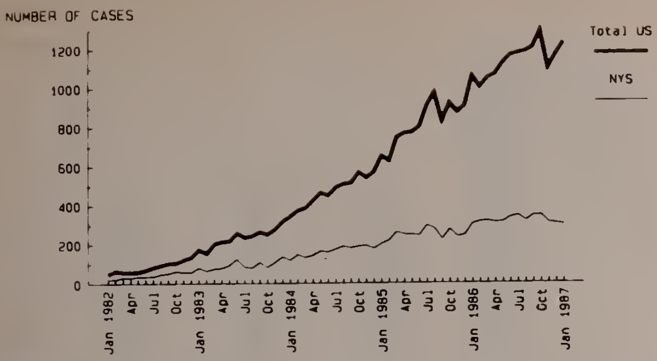

FIGURE 2. Reported cases of AIDS in adults among New York State residents and total in United States by month of diagnosis, January 1982 to January 1987.[1]

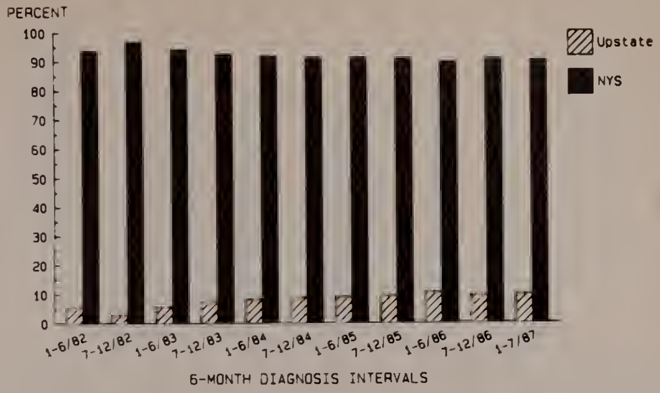

FIGURE 4. Percent of reported New York State adult AIDS cases from upstate general population, by six-month intervals of diagnosis, as of July 17, 1987.[1]

52 out of 57 upstate New York counties had reported AIDS cases (Fig 5). In New York City, the largest number of reported cases as of June 1987 was in Manhattan (5,914), followed by Brooklyn (2,269) and the Bronx (1,634).[4]

Adult AIDS cases in the 57 upstate counties were grouped into four regions on the basis of the degree of urbanization, physical proximity to New York City, and cumulative burden of AIDS to date. Region 1 (New York City vicinity) includes Nassau, Suffolk, and Westchester counties. Together they account for 61% of the AIDS morbidity outside of New York City. They are highly urbanized areas and closest to the New York epicenter. Region 2 (Mid-Hudson Valley) includes the counties of Dutchess, Orange, Putnam, Rockland, Sullivan, and Ulster. Further away from New York City and less urbanized, these counties account for 11.5% of cumulative AIDS morbidity upstate. Region 3 (urban upstate) includes the counties of Albany, Schenectady, Onondaga, Erie, and Monroe. These highly urbanized counties include the cities of Albany, Schenectady, Syracuse, Rochester, and Buffalo. They are physically distant from New York City, yet the factors that contribute to the spread of AIDS in New York City may also be operating in these communities. Region 3 accounts for 18% of the upstate AIDS morbidity. Region 4 (upstate rural) includes the remaining 43 counties outside of New York City. These counties are as far away from New York City as those of region 3, but only one has reported more than ten cases of AIDS. Nine have reported no cases. Region 4 is composed mainly of small towns and rural communities, and accounts for 9.5% of the upstate morbidity.

Age, Sex, Race, and Risk Factors

The percentage distribution of adult AIDS cases is shown in Figure 6 by age group and by place of residence at time of diagnosis. In New York City, New York State, and the United States, the age group 20–39 years accounts for the majority of cases.[3,4] For both New York City and upstate, about 10% of the reported cases are among women (Fig 7). This is approximately twice the proportion observed in the United States as a whole and can be related to the prominence of IV drug use as a risk factor in New York State.

Distribution of adult AIDS cases by race and place of residence at time of diagnosis is shown in Figure 8. In New York City approximately 25% of the AIDS patients are Hispanic and 30% are black. Thus, the majority of the reported AIDS cases occurred in minority groups. Conversely, whites account for 67% and 68% of the cases reported from upstate New York and from the United States, respectively.

Reported risk groups for New York State AIDS cases are shown in Figure 9. Of these risk groups, homosexual or bisexual behaviors can be related to approximately 50% of the cases, and IV drug use is a factor in approximately 35% of the cases.

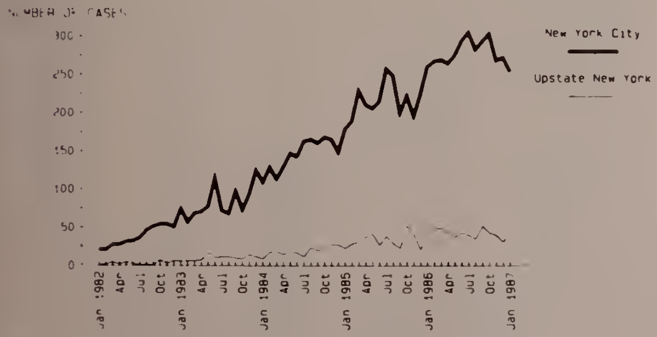

FIGURE 3. Reported cases of AIDS in adults among residents of New York City and upstate New York, by month of diagnosis, January 1982 to January 1987.[1]

YEAR	CUMULATIVE NUMBER OF COUNTIES	CUMULATIVE NUMBER OF CASES
<1982	3	6
1982	11	46
1983	20	171
1984	29	402
1985	45	799
1986	52	1301

FIGURE 5. Upstate counties with confirmed AIDS cases (inmate or general population) by year of diagnosis.[1]

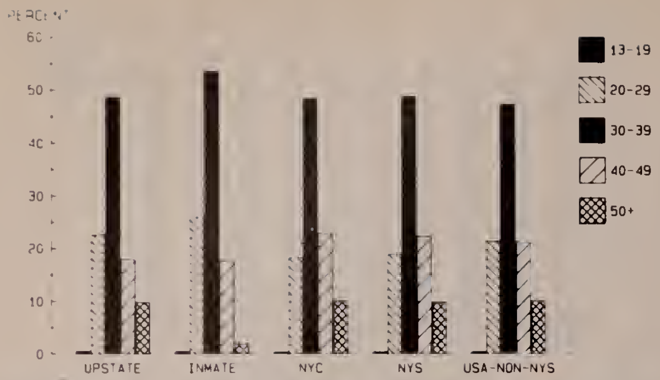

FIGURE 6. Percentage distribution of adult AIDS cases by age group and place of residence at time of diagnosis, as of July 17, 1987.[1]

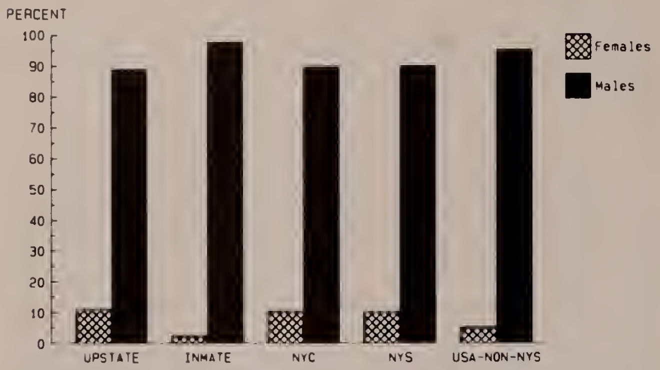

FIGURE 7. Percentage of adult AIDS cases by sex and place of residence at time of diagnosis, as of July 17, 1987.[1]

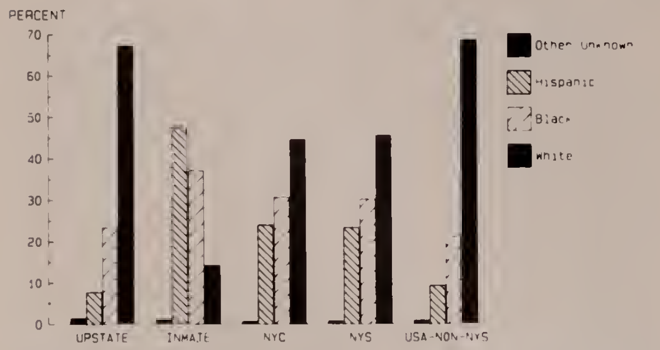

FIGURE 8. Percentage distribution of adult AIDS cases by race and place of residence at time of diagnosis, as of July 17, 1987.[1]

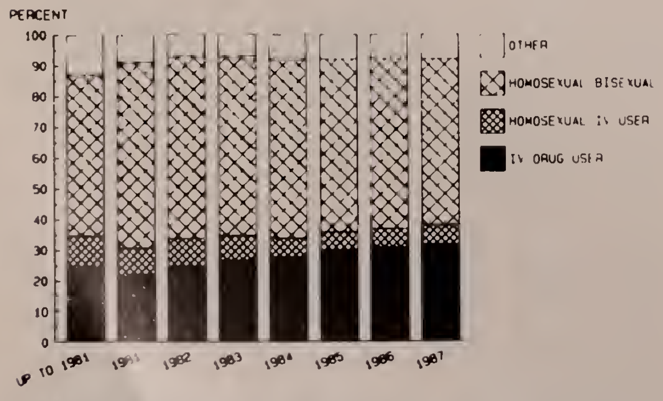

FIGURE 9. New York State AIDS risk groups, percent distribution by year, as of July 17, 1987.[1]

The distribution of adult AIDS cases by risk group varies by place of residence (Fig 10). The major difference between both upstate and New York City and the rest of the United States is the proportion of IV drug users. This proportion is approximately 35% for New York City and upstate and only 15% for the rest of the United States.

Of interest is that the upstate proportion for IV drug use as a risk factor is similar to that of New York City. Examining the IV drug use risk factor by the four regions described earlier, however, a different pattern emerges. In Region 1 (New York City vicinity) and Region 2 (Mid-Hudson Valley), the proportion of IV drug abuse as a risk factor is similar to that of New York City. However, for Region 3 (upstate urban), designated as urban and including a number of upstate cities, the proportion of drug abusers is somewhat lower (28%), and in Region 4 (upstate rural) only 15% have a drug abuse risk factor.

Risk and race distribution among New York State men with AIDS is shown in Figure 11. Among blacks and Hispanics, IV drug abuse is the leading risk factor (50% and 55%, respectively). However, among whites, IV drug abuse is reported among 15% of the cases.

A dissimilar pattern is found among women (Fig 12). IV drug use is the most common risk factor for black, Hispanic, and white women.

PROJECTIONS

In 1991, incident cases per year are projected to be approximately 8,100 for New York City and 9,600 for New York State as a whole (Fig 13).

The adult AIDS cases diagnosed each month between January 1984 and December 1986 (adjusted for reporting

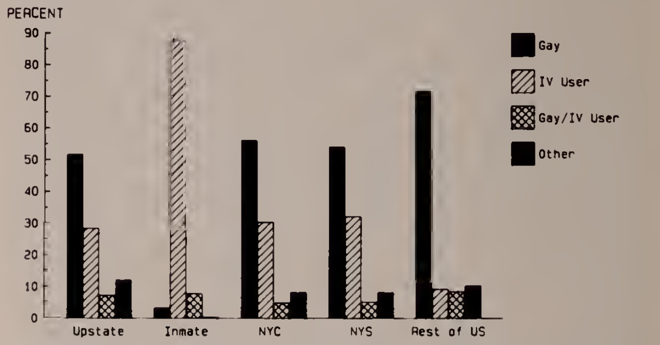

FIGURE 10. Percentage distribution of adult AIDS cases by risk group and place of residence at time of diagnosis, as of July 17, 1987.[1]

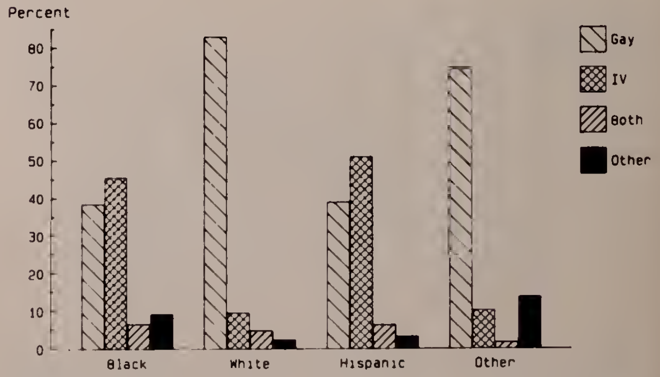

FIGURE 11. New York State men with AIDS by risk group and race/ethnicity.[1]

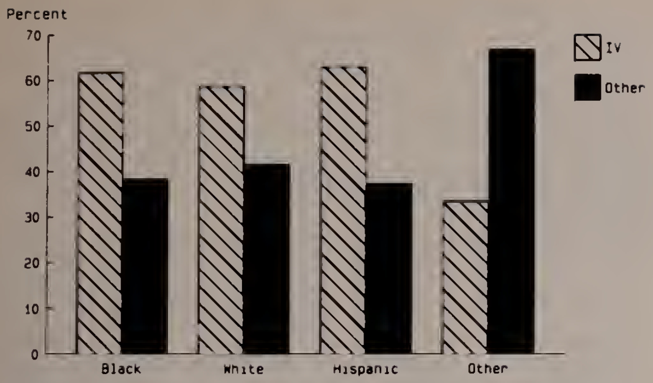

FIGURE 12. New York State women with AIDS by risk group and race/ethnicity.[1]

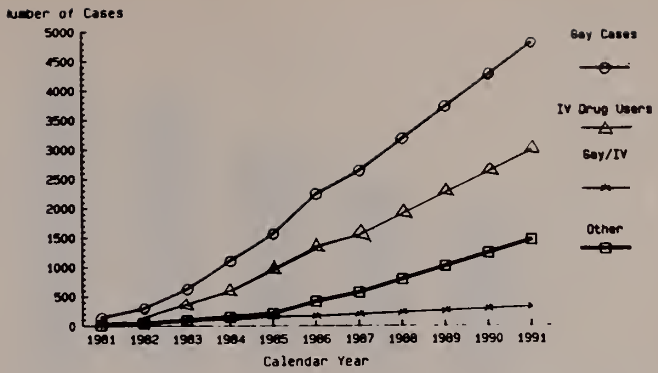

FIGURE 14. New York State AIDS cases by risk group. Cases for 1987 to 1991 are projected.

lag) were divided into five risk group categories: homosexual and bisexual males, IV drug abusers, a group reporting both homosexual and IV drug use behaviors, women of childbearing age, and a group reporting other risk factors. For each category a linear model was extrapolated forward to predict the monthly incidence of AIDS cases through 1991 (Fig 14). These predictions are simply linear extrapolations and do not take into account changes in the dynamics of the epidemic or the susceptibility of various groups. Projections of AIDS cases among women in New York State indicated a 100% increase by 1991 (Fig 15).

A revised case definition for surveillance of acquired immunodeficiency syndrome was instituted by the Centers for Disease Control in the fall of 1987.[5] The major proposed changes applied to patients with laboratory evidence of HIV infection. This new definition includes HIV encephalopathy, HIV wasting syndrome, and a broad array of specific indicator diseases. The inclusion of AIDS patients whose indicator diseases are diagnosed presumptively and the elimination of exclusions due to other causes of immunodeficiency were also important revisions to the surveillance case definition.

The change in definition is expected to increase the number of reportable cases by 13% (Fig 16). Projected prevalent and incident cases taking into account the new definition are shown in Figure 17.

NEW YORK STATE DEPARTMENT OF HEALTH SEROPREVALENCE STUDY

A seroprevalence study comprising a group of studies is being implemented to better determine the HIV antibody prevalence in the population of New York State. This initiative is necessary because of the lack of information about HIV infection in our communities. Previous estimates of the extent of HIV infection have been speculative and not useful for planning or preventive purposes.

Seroprevalence is determined in a number of groups by utilizing blood specimens collected for other purposes. Groups undergoing testing include newborns; clients of family planning clinics, sexually transmitted disease and tuberculosis clinics, and drug treatment programs; and prison entrants. Hospital and emergency room admissions and other categories may be considered in the future for this type of study.

In the newborn study, mandatory blood specimens for detection of hereditary disorders are obtained from all newborn infants prior to discharge or within 14 days of birth and submitted to the NYSDOH Wadsworth Center for Laboratories and Research in Albany, NY. Since maternal antibodies cross the placenta, serologic testing of newborns reflects the HIV antibody status of the mother and not necessarily infection of the infant.

Testing of all specimens is completely blind. The absence of identifying information will protect the anonymity of the individual. Efforts are made to provide the women with information about HIV and to assure the availability of testing and counseling where this is elected.

As of February 5, 1988, 40,259 blood specimens of new-

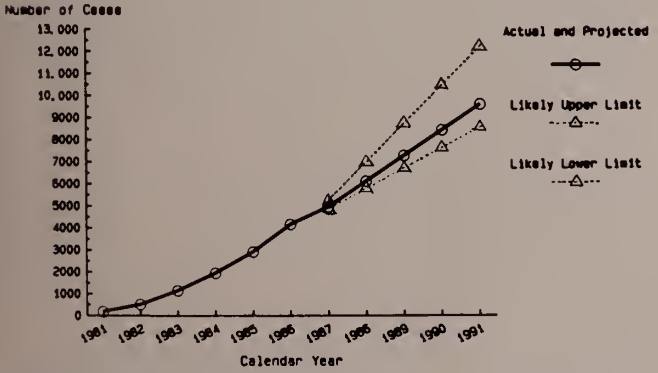

FIGURE 13. New York State AIDS cases with upper and lower limits. Cases for 1987 to 1991 are projected.

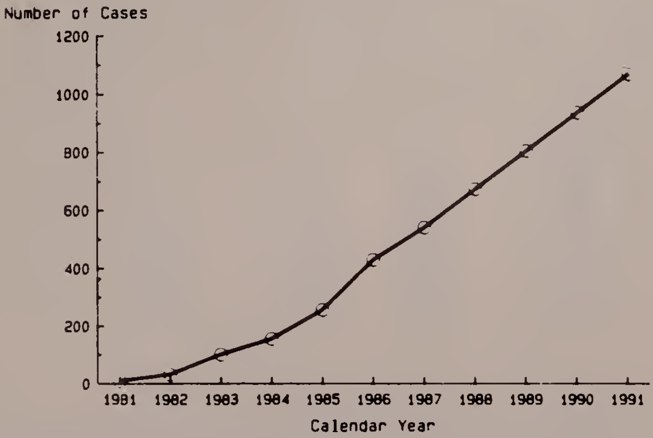

FIGURE 15. New York State AIDS cases in women aged 15 to 44. Cases for 1987 to 1991 are projected.

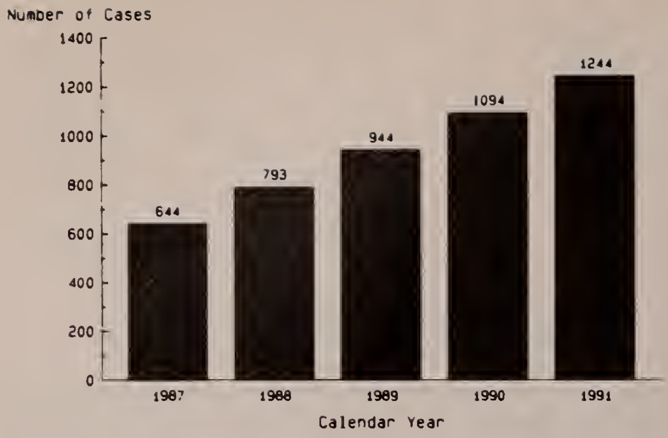

FIGURE 16. Potential added New York State AIDS cases from 1987 to 1991. New case definition increases incident cases by 13%. Mortality experience will remain constant.

borns had been submitted and analyzed for HIV serologic status. The overall seroprevalence rate was 0.84 percent. These seroprevalence rates were 293/18,718 births (1.57%) for New York City and 36/20,809 births (0.18%) for upstate New York. Substantial rates of infection in New York City are linked to areas where intravenous drug abuse is predominant.

Information gained from this type of study can be utilized to develop programs for preventive recommendations and activities. These include educational programs and expanded counseling and testing. A similar seroprevalence testing effort has been initiated by the Centers for Disease Control. A "family of studies" is being implemented in 30 cities across the country, including 20 high prevalence areas and ten low prevalence areas. New York State is the site of two of these studies, with New York City designated as a high prevalence and Rochester as a low prevalence area.

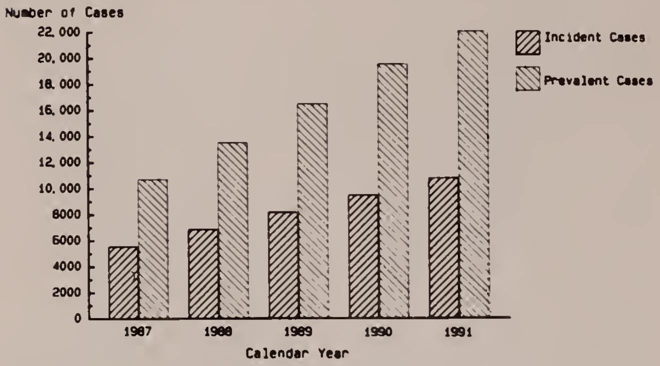

FIGURE 17. New definition projected prevalent and incident New York State AIDS cases, 1987 to 1991.

Seroprevalence studies in New York State have already increased our knowledge of the extent of infection with human immunodeficiency virus. Measurements of seroprevalence over time will lead to a better understanding of the future course of this epidemic. Relationships can now be elucidated between cases of reported AIDS and HIV seroprevalence, especially for particular groups such as infants and young children.

A blind methodology utilizing available blood specimens in the New York State seroprevalence study cannot fully access a cross section of the general population and, therefore, actual prevalence in the entire population cannot be determined. However, an estimate of the prevalence of HIV infection, heretofore unavailable, is now available for subpopulations including women at the time of childbirth. The proportion of this group that is infected has profound implications for the community at large.

These serosurveys supplement but do not supplant case report information for AIDS. In many cases, case information obtained will complement the knowledge obtained from serosurveys, but we will also undoubtedly learn in the near future from an epidemiology based on population and community rather than derived from cases of illness.

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The impact of AIDS on New York's not-for-profit hospitals*

KENNETH E. RASKE

Surgeon General Dr C. Everett Koop has been quoted as saying, "How many are infected? That's our whole problem—we don't know that number."[1] The numbers we do know tell the story of the ever-worsening problem of acquired immunodeficiency syndrome (AIDS) in New York City. As of October 19, 1987, the US Centers for Disease Control (CDC) reported 11,166 diagnosed cases of AIDS in New York City, placing the city at the top of the AIDS census.[2] Approximately 30% of AIDS cases nationwide have been reported from New York.

The AIDS patient profile in New York City is unique. Whereas other cities with a large AIDS population see more uniformity in the risk factors relating to the incidence of AIDS, risk factors in New York City's caseload are diverse, including intravenous drug use and transmission of AIDS through both heterosexual and homosexual activity and from mothers to babies at birth.

THE AIDS CENSUS

The Greater New York Hospital Association's (GNYHA) recent survey of not-for-profit voluntary and public hospitals, conducted in June 1987 with the Health and Hospitals Corporation (HHC), showed 1,266 AIDS and AIDS-related complex (ARC) inpatients in New York City (Fig 1). That is a full 18% over the number reported in GNYHA's first survey of AIDS and ARC inpatients in March 1987, when a total of 1,071 were counted. The results of GNYHA's latest survey revealed an average daily census of 1,335 patients with AIDS or suspected AIDS in New York City during the week of October 18, 1987. According to these figures, the number of hospitalized patients with AIDS or suspected AIDS increased overall by 24.6% between March and October 1987. However, the census increase from June to October (5.5%) was considerably less than the March to June increase.

This new demand on health care resources comes at a time when New York's hospitals are stretched to capacity. A spot check of occupancy rates of not-for-profit voluntary and municipal hospitals in October 1987 showed 34 of 46 voluntary hospitals citywide at or above 90% occupancy, as were all 11 municipal hospitals. Seventeen of the voluntary hospitals were at or above 100% occupancy. Throughout the voluntary hospital system, 379 patients were awaiting admission in emergency rooms.

This bottleneck in the system is created by the overcrowding problem, as the capacity of New York's hospital system is pushed to its natural limit. As hospital occupancy increases and demand for beds outpaces supply, patients are forced to wait in emergency rooms for admission, making it more and more difficult for emergency rooms to accommodate additional emergency cases. Indeed, GNYHA's same spot survey showed that of 37 hospitals, 22 had either asked for and been refused or were on diversion status from 911.

While the increasing numbers of AIDS and ARC patients may be an easy explanation of the occupancy problem of hospitals in New York, it is likely not the complete explanation. The New York State Department of Health is currently undertaking an analysis of additional factors that may be affecting occupancy, including changes in the number of alternate level of care days, patterns of elective surgery, and average lengths of stay. GNYHA will be cooperating with Department of Health officials on this project and will be contributing data for analysis.

PLANNING AIDS TREATMENT FOR THE FUTURE

A major part of the challenge of coping with the AIDS crisis is literally predicting the future, and then planning

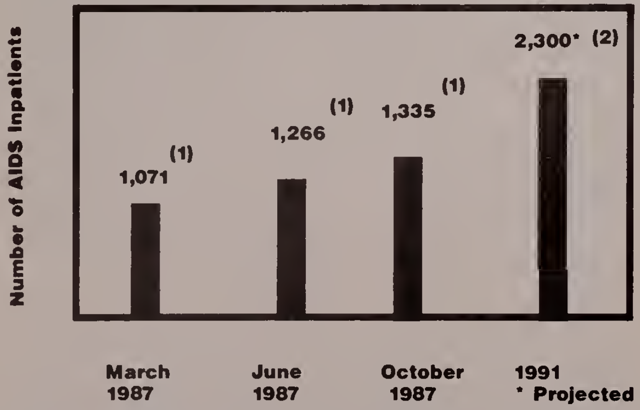

FIGURE 1. Inpatients in New York City with AIDS and AIDS-related complex (Sources: (1) GNYHA and Health and Hospitals Corporation; (2) New York State Department of Health.)

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for it. That is no easy task, as is seen from the differing numbers of AIDS cases predicted by city and state officials and other sources. According to recently released figures presented in a New York State Department of Health draft report, by 1991 New York City will have an average daily inpatient census of more than 2,300 patients with AIDS or AIDS-related illnesses.[3] The mid-point projection of these figures for 1987–1988, at 1,296, is close to GNYHA's June 1987 survey figure of 1,266 AIDS inpatients.

These new estimates reflect a substantial upward revision from previous Department of Health estimates. Since these figures corroborate GNYHA's actual count of AIDS inpatients, they may be more reliable than other predictions. But some would argue that even those figures may be too low. However, the appropriate question is not who may eventually be right and who may be wrong in estimating future numbers of AIDS patients, but rather finding a way to provide current information and to update estimates accordingly. Projection equations, therefore, should be adjusted periodically, perhaps quarterly, to reflect the most recent experience. In the final analysis, we need to be alerted to the need for a resource response to any changes in demand.

COSTS OF TREATING AIDS INPATIENTS

As the number of AIDS and ARC patients in New York City increases, so do the demands on the health care system, from nursing and social work to laundry, housekeeping, and dietary facilities. In April 1986, GNYHA commissioned a study of the differential cost increase in treating AIDS patients over the average per diem cost.[4] The following routine cost centers at the two hospital study sites were significantly affected by the AIDS caseload: general nursing service, central services, social work, housekeeping, laundry and linen services, and dietary services. The study found that the cost of treating AIDS patients was approximately 22% greater than the cost of treating an average medical/surgical patient. (The reported total health bills for AIDS patients from time of diagnosis to time of death ranged from a low of approximately $42,000 to a high of $150,000.[5])

Hospitals, it was found, assign more nursing resources to the care of AIDS patients, who require up to 28% more nursing hours per patient day than average medical/surgical patients. In addition, AIDS patients incur four times as many bed linen changes as the average medical/surgical patient. Every AIDS patient receives social work services, while cases with social work referrals, in general, are a small proportion (approximately 17.5%) of the total patient population. Hospitals follow more extensive room cleaning procedures for isolation patients, and many AIDS patients require such precautions. And as the incidence of AIDS increases, the overall costs of treating potentially infectious waste will clearly rise.

Based on this study of the routine costs of treating AIDS patients, and a state-generated analysis of SPARCS data, in which a 19.6% difference for ancillary components including laboratory, radiology, blood bank, pharmacy, and other services was identified, the New York State Department of Health agreed to adjust hospital reimbursement rates for AIDS patients. The new rates, applied to inpatient days from 1984 to 1987, provide for a 22.5% increase in the routine portion of the inpatient rate and a 19.5% increase in the ancillary component. This rate adjustment, designed to account for the costs of treating AIDS patients, is applicable to the current reimbursement methodology entitled the New York Prospective Hospital Reimbursement Methodology.

The state has also developed an initiative to designate some hospitals as AIDS Centers, a concept which GNYHA supports because of its potential to increase access and quality services and to enhance coordination of the continuum of care—all appropriate goals for the treatment of AIDS patients and all patients with chronic and terminal illnesses. AIDS Centers receive a prospective per diem reimbursement rate for treating AIDS patients with Medicaid and Blue Cross based on budgeted costs approved by the state. It will be important to monitor and evaluate the appropriateness and adequacy of this payment approach to allow each center to meet the comprehensive care requirements of AIDS patients and to comply with state standards. The performance of both the AIDS Centers and other hospitals that care for AIDS patients should also be carefully examined in terms of organizational structure, cost, and quality of care.

As we move into the case payment system in 1988, a distinct series of diagnosis related groups (DRGs) to recognize the illnesses of AIDS patients will be utilized to reflect greater accuracy in the weights assigned to AIDS cases, and therefore the resource intensity and payment on behalf of AIDS patients. While it is the providers' responsibility to deliver quality care, it is GNYHA's belief that the state has a public responsibility to continue to recognize the financial implications of delivering this care and to reimburse hospitals accordingly.

STAFFING DEMANDS: THE BURNOUT FACTOR

Use of hospital resources is not only measured in dollars, however. In addition to the medical interventions and treatments and support services required to manage the disease, hospital staff members who have contact with AIDS patients are frequently called on to provide emotional support to patients and their friends and relatives, to assist in daily living tasks, to identify appropriate community resources, and to assist in preparations for death. Unfortunately, few staff members are adequately prepared to deal with the full range of needs of AIDS patients. And this shortcoming has become more and more apparent as the number of AIDS patients grows.

Individuals who choose careers in health professions seek to cure sickness, save lives, and improve the overall quality of life. Modern medicine permits the overwhelming majority of patients to recover fully from episodes of illness and to return to productive lives. Occasionally, however, this outcome is not achieved. Such is the case with AIDS patients. Furthermore, the unique needs of these patients create problems for patients and health care workers alike. The result may be the psychological burnout of the professional and support staff. One must remember, however, that there are tens of thousands of health care workers who come face to face with AIDS patients every day and who provide them with compassionate, quality care. Their daily heroics should not be dwarfed by the isolated cases we hear so much of in news reports of health care professionals who may be reluctant to treat AIDS patients.

GNYHA is keenly aware of the demands on hospital staff and the additional stress involved in caring for patients with AIDS. In a major project, Greater New York Hospital Foundation, Inc, a subsidiary of GNYHA, in cooperation with representatives of the academic community in New York City, is developing an educational program aimed at health care personnel working primarily in hospitals and long term care facilities. It is being designed with major funding support from the W.K. Kellogg Foundation and supplemental funding from the United Hospital Fund of New York and the Health Services Improvement Fund. The pilot sessions to help health care personnel cope with the treatment of AIDS and other chronic illnesses took place in April 1988.[6]

It is still to be determined how the prevalence of AIDS affects recruitment and retention of interns and residents, a question of great concern for this city's major teaching hospitals. GNYHA is currently drafting a grant proposal and will soon be seeking funding to explore this issue.

UNIVERSAL PRECAUTIONS

Clearly, AIDS patients draw more heavily on existing resources and practices in the health care system than do patients who do not have AIDS. In addition, management of the disease is necessitating major changes in daily hospital operations. In August 1987, the CDC published revised recommendations for the prevention of transmission of the AIDS virus in the health care setting. In establishing "universal blood and body fluid precautions," this document emphasized the need for health care workers to consider all patients as potentially infected with the AIDS virus and to adhere rigorously to infection control precautions for minimizing the risk of exposure to blood and body fluids. These precautionary procedures are especially important in emergency care settings where the risk of exposure to blood is increased and the infection status of the patient is usually unknown.[7] GNYHA has asked the CDC for clarification of its universal precautions guidelines regarding the possible waste disposal implications of these procedures.

These new procedures have practical consequences. It is no surprise, for example, that GNYHA's group purchasing arm reports a 30–40% increase in orders for gloves from 1986 to 1987. Some back orders for the latex gloves preferred by hospital personnel have taken as long as two to three months to fill.

CONCLUSION

AIDS has left no component of the health care delivery system untouched. However, facing us today is not merely an assessment of a present problem and a measurement of current resources. We must confront the long term future of treating patients with AIDS and AIDS-related diseases. There are no quick, temporary solutions to the AIDS problem and, barring a medical miracle, AIDS will be a continuing health care challenge at least until the end of the century. All involved parties, including the New York State Legislature, the New York State Department of Health, the New York City Department of Health, GNYHA, Health and Hospitals Corporation, Health Systems Agency of New York City, and others must work closely together to develop and implement effective solutions.

Most importantly, bed needs in New York City must be reassessed to reflect the demands of AIDS cases and to build a reserve capacity of 10–15% into the acute care system to respond to other emergencies. Assessing the current situation, it appears that by 1991 a minimum of two to three new or refurbished hospital facilities must be added to the stock of hospitals in New York City. Public policymakers must begin examining ways to get new capital on line at the appropriate times. In the meantime, we must also find new ways to channel inpatient care into alternate delivery systems.

When an AIDS patient no longer requires the services of an acute care institution, a major stumbling block to timely and humane discharge has been the lack of appropriate community or long term care placement options. Placement options for all AIDS patients, especially the very difficult to place intravenous drug users and babies who have AIDS, need to be established. Organized home care services, hospice services, and beds in long term care facilities need to be encouraged and increased, and options for day care services and support services for children of AIDS patients need to be explored.

In addition, improvements to the present system of delivering outpatient services to AIDS patients should be examined. Since 1981, however, reimbursement for clinic services provided to Medicaid patients in New York State has been capped at an all-inclusive rate of $60 per visit. Such reimbursement constraints do not encourage creativity and innovation. AIDS Centers do receive special pricing for providing outpatient services to AIDS patients, however, the majority of hospitals caring for AIDS patients today are not designated as AIDS Centers. Reimbursement rates, therefore, need to address the resource requirements and costs for treating a significant segment of the AIDS population. We must continue to study and evaluate outpatient costs of treating AIDS patients, and consequent adjustment to the clinic rate cap relating to AIDS should be recommended.

In many instances, however, neither long-term placement options nor outpatient services will be able to provide the care AIDS patients require. In the final analysis, there may prove to be no substitute for acute inpatient care in the complete treatment of AIDS patients. How the health care community responds to the AIDS epidemic in the coming months and years will determine not only the future treatment of AIDS but the future of the health care system as well. If we are to continue to respond meaningfully to the needs of patients with AIDS and AIDS-related illnesses for quality health care, responsible and effective planning must begin immediately.

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AIDS in Connecticut*

JAMES L. HADLER, MD, MPH; JULIA WANG MILLER, PhD; MARGE EICHLER, RN, MPH

In order to understand the epidemiology of acquired immunodeficiency syndrome (AIDS) in Connecticut, it is important to review the demographic features that underlie the manner in which the data have been analyzed.

Connecticut is by far the smallest of the three states in the tri-state (New York, New Jersey, Connecticut) area. With a population of 3.2 million, it is the 25th largest state in the US.[1] Geopolitically, it is divided into eight counties and 169 towns. Unlike in New York and New Jersey, the counties have little political significance. There are no county governments or county health departments. Rather, the relevant political and health organization is at the town level. Correspondingly, for practical epidemiologic purposes, most data analysis is done at the town level. For a disease such as AIDS, in which morbidity is measured as cumulative incidence per million population, this can pose difficulties, as the average town population is only 20,000.

The five largest towns have between 100,000 and 150,000 residents. When their metropolitan areas are taken into consideration, their populations expand to between 200,000 and 700,000. The population is most dense in the southwest corner close to New York City: nearly half the population lives in Fairfield and New Haven counties, in the urban-suburban corridor leading out of New York City (Fig 1, areas labelled 1). The rest of the state is predominantly suburban and rural with the one exception of the City of Hartford.

In terms of proximity to New York City, Connecticut can be divided up by county into four regions with decreasing commuting and public transportation potential to reach the city. These are shown in Figure 1.

Racially, Connecticut's population is mainly white: 87% white, 7% black, and 4% Hispanic, making it the state with the lowest proportion of minorities in the tri-state area.[1 (pp 220-221,225)] Most of the minority population lives in seven towns in the southwest corner of the state (Fig 1); these towns comprise 24% of the total state population, but house 70% of all black and Hispanic persons.

Finally, in spite of Connecticut's ranking as one of the wealthiest states in the US, and in spite of the small size of its urban areas, at least three of these towns have neighborhoods in which socioeconomic conditions are nearly as poor as any to be found in New York or New Jersey. These towns are Bridgeport, New Haven, and Hartford.

EPIDEMIOLOGY OF AIDS IN CONNECTICUT

Although Connecticut is small and lacks large urban centers, the epidemiology of AIDS in Connecticut is more similar to than different from the epidemiology of AIDS in New York or New Jersey.

Magnitude of the AIDS Problem. The magnitude of the AIDS problem in Connecticut is relatively large. As of October 15, 1987, 515 cases of AIDS had been reported for a cumulative incidence ratio (CIR) of 156 cases per million population. Nationally, Connecticut ranked 14th among states in number of cases reported, and ninth in per capita incidence (expressed as CIR), behind New York (first, CIR of 683) and New Jersey (third, CIR of 347) but ahead of New York State minus New York City (CIR of 129).

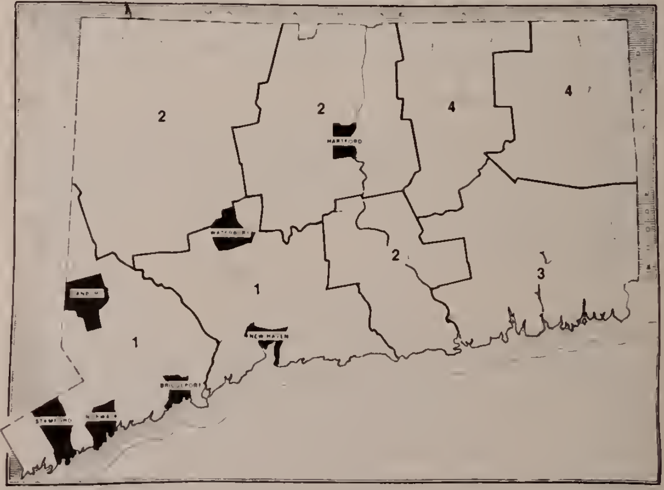

FIGURE 1. Map of Connecticut by region, counties, and major cities. Region 1, Fairchild and New Haven counties; Region 2, Litchfield, Hartford, and Middlesex counties; Region 3, New London County; Region 4, Tolland and Windham counties. Regions are numbered in decreasing proximity to New York City. Towns outlined in black include Stamford, Norwalk, Bridgeport, New Haven, Danbury, Waterbury, and Hartford.

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* This article is based on a presentation at the Second Montefiore Symposium on AIDS, "AIDS in the Tri-State Area: A Regional Portrait of the Epidemic," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held November 5, 1987, at the New York Academy of Medicine.

In spite of their small size, urban-metropolitan areas of Connecticut also have a relatively significant AIDS problem. Until mid-1987, the Centers for Disease Control routinely reported information ranking metropolitan areas (expressed as Standard Metropolitan Statistical Areas (SMSAs)) with ten or more AIDS cases by CIR. As of June 19, 1987, three Connecticut SMSAs ranked in the top 30 nationally, along with two from New York and five from New Jersey. These included New York City (CIR, 1,083), Jersey City (CIR, 790), Newark (CIR, 458), Patterson (CIR, 413), New Haven (CIR, 307), Poughkeepsie (CIR, 298), Trenton (CIR, 266), Norwalk (CIR, 213), Stamford (CIR, 201), and New Brunswick (CIR, 198). More focally, the City of New Haven had a CIR of 848 at that time. Thus, Connecticut has geographic foci of AIDS which would be of major concern anywhere.

Finally, projections based on Connecticut data have not yet been made. However, Connecticut has contributed a steady 1.2% to the national total of reported AIDS cases. Borrowing from the projections made by the US Public Health Service at Coolfont, West Virginia, in 1986 and assuming that the Connecticut contribution will remain stable, more than 800 new cases are expected to be reported in 1991. On the other hand, this assumption may not hold up. As the next section demonstrates, the dynamics of AIDS in Connecticut are somewhat different than those nationally.

AIDS by Transmission Category. Table I shows the relative contribution to AIDS by underlying risk behavior category and trends over time. As with New York and New Jersey, Connecticut has a much higher proportion of AIDS cases comprising intravenous (IV) drug users than is seen nationally (33% vs 16%). In addition, the relative percentage of all cases attributable to IV drug use has been increasing over time, so that IV drug use is becoming the leading AIDS risk behavior in Connecticut. The trend toward an increasing contribution to AIDS incidence from intravenous drug users has not been observed nationally.

In parallel to the increase of AIDS in IV drug users, the percentage of AIDS cases attributable to heterosexual contact has also increased (Table I). Nearly all of these cases are the sexual partners of IV drug users.

AIDS by Sex. Connecticut also has a much higher proportion of AIDS cases among women than is seen nationally (16% vs 7%). Overall, Connecticut ranks third nationally in sex- and race-specific incidence in women, behind only New York and New Jersey (Table II). Thus, women in the tri-state area have the highest AIDS risk of women anywhere in the country. The risk to women is largely related to IV drug use. More than 80% of the women with AIDS in Connecticut are either intravenous drug users (56%) or their sexual partners (25%).

Pediatric AIDS. The theme for pediatric AIDS is similar to that for AIDS in women: Connecticut has a disproportionately high representation, and most of the cases are directly or indirectly IV drug related. As of mid-October 1987, 3.1% of Connecticut's AIDS cases were in children, compared to 1.4% nationally. Eighty percent (12/15) were directly or indirectly related to IV drug use.

AIDS in Minorities. Minorities in Connecticut have been at particularly high risk of AIDS. Although only 11% of the population is black or Hispanic, nearly 50% of all reported AIDS cases have occurred in these two groups. Furthermore, blacks and Hispanics in Connecticut have a higher relative risk of AIDS than do their counterparts nationally. Compared to whites, blacks in Connecticut have been 8.6 times more likely to have developed AIDS; Hispanics have been 7.1 times more likely. Nationally, the comparable figures are 3.0 for each. This high relative risk has been corroborated in seroprevalence studies in Connecticut. Black and Hispanic men and women attending sexually transmitted disease (STD) clinics and counseling and testing sites, entering methadone clinics, and taking the human immunodeficiency virus (HIV) antibody test as part of military recruitment all have more than four times the probability of being seropositive than their white counterparts. This disproportionate representation among minorities is of major importance in devising and targeting prevention efforts in Connecticut.

Among minorities, intravenous drug use and sexual transmission from drug users appear to be the largest sources of HIV transmission. Altogether, 76% of AIDS cases in minorities have been drug related: 61% in drug users, 9% in heterosexual contacts of drug users, and another 6% in their offspring. Although only 21% of minority AIDS cases have been in homosexual/bisexual men, 22% of all cases in homosexual/bisexual men have been minorities, a figure that is double the minority population representation. Thus, prevention efforts targeted to minorities need to be directed at all types of risk behavior, including drug use, sexual transmission, and perinatal transmission.

AIDS by Place. AIDS in Connecticut is not equally distributed geographically. Residents of urban areas have been at considerably greater risk than residents of non-

TABLE I. Transmission Categories for AIDS Trends Over Time, Connecticut, October 30,1987

Transmission Category	1980–1983	1984–1985	1986–1987	All Years	*P*-value Trend
Homosexual/bisexual*	51%	46%	40%	43%	.08
Intravenous drug user*	20%	27%	39%	33%	.002
Heterosexual†	0%	3%	6%	4%	.05
Total cases	41	173	273	487	

* Excluding those with both homosexual/bisexual and intravenous drug use exposure.

† Excluding persons whose heterosexual risk was birth in a country where heterosexual transmission is believed to be the primary mode of transmission.

TABLE II. Cumulative AIDS Incidence Ratios* in Women By State† and Race, July 29, 1987

State	White	Race Black	Hispanic
New York	4.5	69.9	64.7
New Jersey	4.0	76.0	22.6
Connecticut	2.4	50.8	20.1
Massachusetts	1.9	30.7	7.9
California	1.7	(6.2)	(1.8)
Florida	1.5	51.0	(2.6)

* Cases per million population, 1980 census.

† Highest ranking states nationally by incidence in white women. Numbers in parentheses indicate less than top six ranking.

TABLE III. AIDS Incidence in Connecticut By Proximity to New York City and Year of Diagnosis, October 15, 1987

Region*	1980–83	1984–85	1986–87	CIR[†]	RR[‡]
1	78%	67%	66%	220	3.8
2	22%	27%	28%	129	2.2
3	—	3%	4%	71	1.2
4	—	4%	2%	58	—
Total cases	41	171	303	—	—

* Regions defined by county (Fig 1).
[†] Cumulative incidence ratio: no. AIDS cases/population in millions.
[‡] Risk ratio of the region compared to Region 4.

urban areas. Sixty percent of AIDS cases have occurred in residents of seven towns comprising only 24% of the population (Bridgeport, Danbury, Hartford, New Haven, Norwalk, Stamford, Waterbury). Residents of these seven towns have been 4.6 times more likely to develop AIDS than nonresidents, based on comparison of CIRs. When analyzed by race-ethnicity, whites in the seven towns have had 2.7 times the risk of their nonurban counterparts, and minority groups have had 2.1 times the risk.

New York City AIDS Connection. Connecticut's geographic proximity to New York City and the ease with which the city can be reached by public transportation make it likely that much of Connecticut's AIDS problem, at least initially, represented spread from the New York "epicenter." At least four types of epidemiologic information suggest that this is what did indeed occur.

First, all initial AIDS cases in Connecticut occurred in the southwest region in the urban-surburban corridor leading out of New York City. No cases were seen east of the Connecticut River until 1984 (Table III, Regions 3 and 4). Second, as part of surveillance during 1983–1984, randomly selected incident AIDS patients, including both gay men and intravenous drug users, were interviewed as to where their risk-taking behavior had taken place. All interviewed persons had been to New York City for either shooting gallery use or sexual activity in bathhouses between 1980 and the time their disease was diagnosed.

In addition, the incidence of AIDS increases the closer one gets to New York City, at least at the county level (Table III). Finally, in the one statewide seroprevalence study, entrants to methadone clinics closer to New York have been more likely to have antibodies to HIV than those further from New York. This has been true regardless of race-ethnicity (Figure 2).

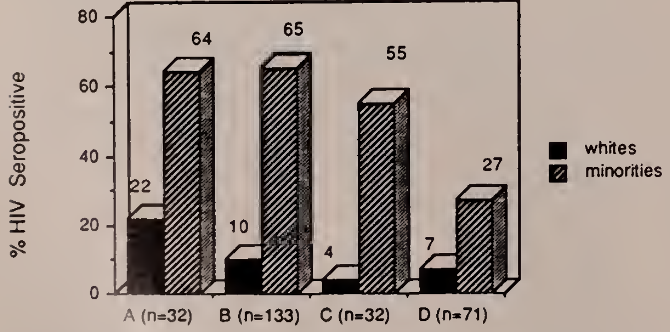

FIGURE 2. HIV seropositivity in methadone clinic entrants by proximity to New York City, for Connecticut, March 1986–March 1987. (Location A is nearest New York City, location D is furthest.)

TABLE IV. Sources of HIV Antibody Seroprevalence, Connecticut, October 30, 1987

Source	Group Tested	Number Tested	Percent Seropositive*	RR[†] in Minorities
Anonymous counseling and testing sites (3/86–6/87)	Total	2,200	6.9	3.5
	Intravenous drug abusers	306	18.3	—
	Homosexual men	699	10.6	—
	Heterosexuals	938	1.9	—
Methadone maintenance clinics (3/86–3/87)	Intravenous drug abusers	268	24.0	5.0
Sexually transmitted disease clinic (3/87–10/87)[‡]	Clients	159	3.8	
Military (10/85–9/87)	Recruits	11,287	0.14	8.3
Red Cross, Connecticut chapter (1/87–9/87)	Donors	152,124	0.005	—

* Confirmed seropositive by Western blot.
[†] Relative risk = probability of seropositivity for blacks and Hispanics divided by probability for whites only.
[‡] Six out of 90 minority group members vs 6 out of 67 whites were seropositive.

Although these data suggest that at least some of the AIDS cases in Connecticut, especially early on, have been attributable to exposure to HIV in New York City, it is clear that there is indigenous transmission as well. Many of the seropositive intravenous drug users have not been to shooting galleries outside of Connecticut, there is an increasingly wide geographic distribution of cases within the state (Table III), and nearly all of the cases involving heterosexual contacts and children appear to have been acquired in the state.

Seroprevalence Studies. As of October 1987 there were five ongoing sources of information on HIV antibody seroprevalence in Connecticut. These are shown in Table IV. As noted previously, race-ethnicity has been the best predictor of seropositivity in each of these, thus far.

There will be an expansion of surveillance for seroprevalence in 1988. Additional multiyear seroprevalence studies that have started or will begin shortly include seroprevalence in tuberculosis cases statewide, in newborns statewide using the filter paper disc method, and in additional selected sexually transmitted disease clinics, family planning clinics, and prenatal clinics. Particular emphasis will be placed on the newborn study. It is hoped that this will become a major component of Connecticut's surveillance to monitor the extent of ongoing transmission of HIV to women and children.

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Current issues concerning AIDS in New York City*

Stephen C. Joseph, MD, MPH

This is a critical period in the acquired immunodeficiency syndrome (AIDS) epidemic. Today's public policy decisions will determine the state of the epidemic, and its impact on New York City, five, seven, and even ten years hence.

As of October 1987, 12,000 people had been diagnosed with AIDS in New York City—comprising 28% of the national total—with more than 6,500 deaths. More than 300 new cases of AIDS are reported every month. AIDS is the current leading cause of death in the city among men aged 25–44 years and women aged 25–34 years.[1]

Currently, upwards of 400,000 New Yorkers are estimated to be infected with the human immunodeficiency virus (HIV), including some 250,000 homosexual and bisexual men and 120,000 intravenous (IV) drug abusers.

AIDS Among Intravenous Drug Abusers

Dr Rand Stoneburner, Director of AIDS Research for the New York City Department of Health, presented a paper at the recent American Public Health Association meeting that received a good deal of media attention.[2] It is important to understand the relevance of Dr Stoneburner's findings. For more than three years, we have reported what we think is a compelling hypothesis that the number of HIV-related deaths is substantially undercounted in the IV drug abuser community because of shortcomings in the Centers for Disease Control (CDC) case definition. We now estimate that the number of AIDS-related deaths among IV drug abusers in New York City is more than 150% higher than previously thought.[2]

A study of all drug-related deaths in the city from 1982 through 1986 found that an additional 2,500 people died of AIDS-related conditions than we had previously counted in our surveillance. These deaths are not included because they did not meet the strict federal definition of AIDS. Investigation of death records revealed, over time, this evidence of excess mortality among IV drug abusers. When we sampled charts of people who died of nonspecific pneumonia, endocarditis, and tuberculosis, many charts exhibited evidence of HIV infection or symptoms compatible with AIDS, such as oral thrush.[2]

If the HIV mortality rate is readjusted to account for these excess deaths, AIDS-related deaths involving IV drug abusers would account for 53% of all AIDS-related deaths in New York City since the epidemic began, while deaths among homosexual men would account for 38%. Previous figures showed that homosexual men accounted for 55% of all AIDS-related deaths, and intravenous drug abusers for 31%[2] (Fig 1). These figures represent a compelling hypothesis that needs validation.

Besides increasing the number of drug abusers who are so sick with HIV-related illness that they are dying, the new data are disturbing because recent evidence suggests that people at the end stage of illness have a return of viremia, making them more infectious and a greater danger to the community than in the middle stage of the illness. The actual rates of transmission from addicts here may be underestimated.

This is particularly worrisome because IV drug abusers have been the main conduit of HIV infection to women and children (Fig 2). Of the more than 1,200 cases of AIDS in women in New York City, 80% have been IV drug abusers or their sex partners. Most of the 231 children with AIDS were infected through their mothers, 80% of whom were IV drug abusers or their sex partners (Fig 3).

Epidemiologic investigation and early results of serologic surveys have shown that HIV infection in heterosexuals is primarily linked to exposure to IV drug users or bisexual men. There is little current evidence for secondary or tertiary spread among heterosexuals. The large pool of infected people in New York City does increase the

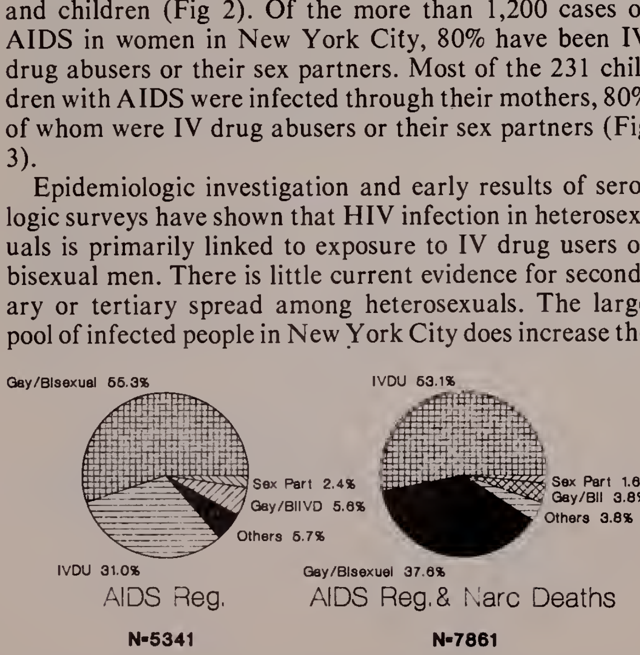

FIGURE 1. Postulated impact of narcotic deaths on total AIDS mortality by risk group, New York City, 1982–1986.

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* This article is based on a presentation at the Second Montefiore Symposium on AIDS, "AIDS in the Tri-State Area: A Regional Portrait of the Epidemic," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held November 5, 1987, at the New York Academy of Medicine.

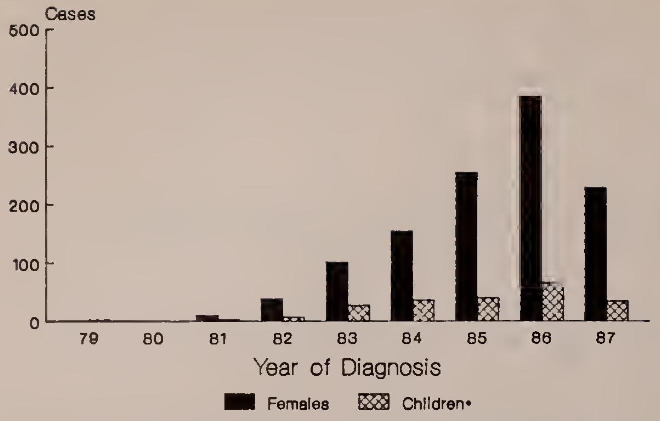

FIGURE 2. AIDS incidence among women and children in New York City, August 1987 (preliminary data). Transfusion-associated cases are excluded.

probability of HIV spread through heterosexual sex. At particular risk are sex partners of IV drug users, who are mostly minority women.

A Department of Health study is now under way to determine the rate of HIV infection among women at high risk, primarily IV drug users and sex partners of men at high risk. Early results indicate that 40% of these women are infected. Early results from Health Department studies estimate that 1.5% of all women in New York City who become pregnant may be infected with HIV.

Three-quarters of the children with AIDS in New York City have died, most by the age of three years. An estimated 600 HIV-infected children are expected to be born this year. Among teenagers aged 13 to 19 years, 35 AIDS cases have been reported. Ominously, young women represent 40% of these cases, compared to 11% of all AIDS cases. Half the adolescent women with AIDS in New York City were IV drug users or their sex partners.

With IV drug use concentrated in the city's poorest neighborhoods, the AIDS epidemic is hitting minorities the hardest. Thirty-one percent of the city's AIDS cases are among blacks; 23% are among Hispanics. Eighty-six percent of male IV drug users with AIDS and 90% of mothers of children with AIDS are black or Hispanic (Fig 4).

AIDS PROJECTIONS

The best current estimates project that by the end of 1991, over 43,000 people will have developed full-blown AIDS in New York City; 32,000 will have died (Fig 5). More AIDS cases will have been diagnosed here in 1991 alone than have shown up from 1981 through 1986 (Fig 6).

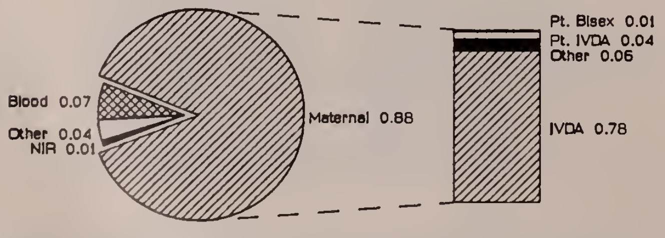

FIGURE 3. New York City pediatric AIDS cases by risk groups, March 1987.

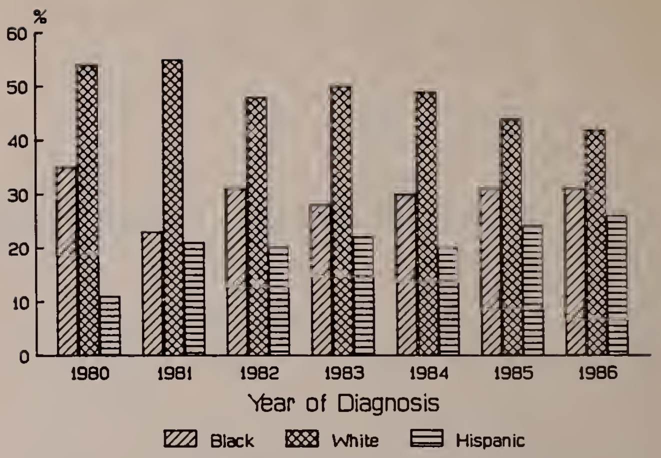

FIGURE 4. AIDS incidence in New York City by race/ethnicity.

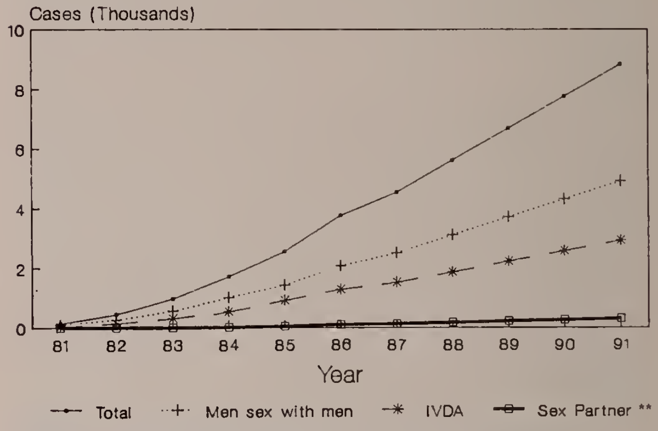

FIGURE 5. AIDS incidence in New York City, 1981–1991.

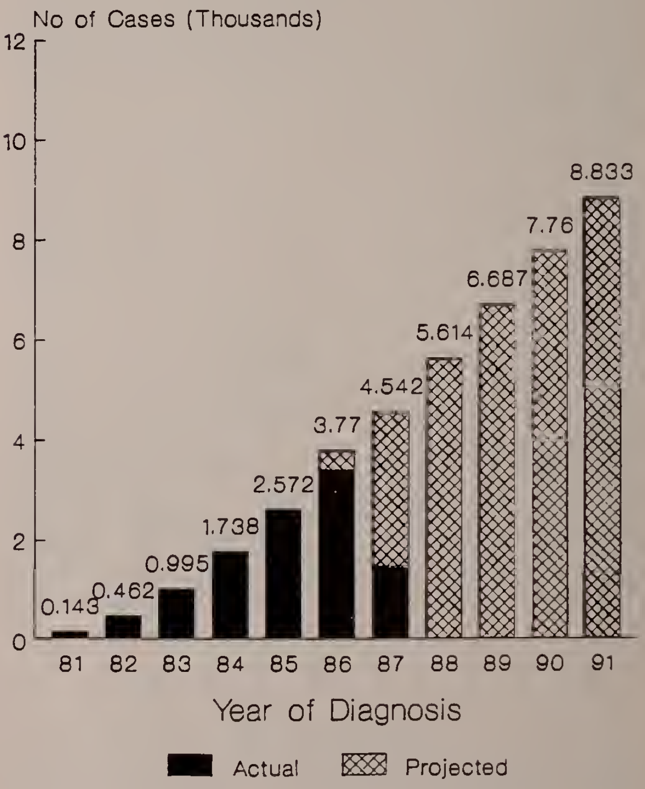

FIGURE 6. AIDS incidence in the total population (August 17, 1987).

Among men, close to 37,000 cumulative AIDS cases are projected for 1991, compared with 6,000 for women (Fig 7). By 1991, women will have accounted for 13% of the total cases, compared with 11% presently (Fig 8). Homosexual men will represent 24,000 cases, 56% of the total (Fig 9); 14,500 cumulative cases are projected among IV drug users, 34% of the total (Fig 10). Almost 2,800 cumulative cases are projected among young people aged 13 to 24 years (Fig 11).

These projections assume that the rates of the last six months will remain constant over the next four years. They reflect only cases already counted, using the current standard case definition. They do not include the newly reported figures that reflect the undercounting of mortality among addicts. These would greatly inflate the current number of persons ill with HIV infection and the deaths from HIV-related illness, as well as increase the projections for the future by as much as 150% for drug abusers.

These estimates challenge us to understand the patterns of infection, control the spread of the virus, protect people from AIDS-related discrimination, and provide medical care and social service support to those sick from the disease. Long term resource requirements for clinical care and social, mental health, housing, hospice, and respite services are major concerns. If a vaccine or effective treatment were developed, resource demands would still intensify as the number of people with AIDS continues to rise.

HOSPITAL SERVICES

Hospital services in particular will be strained by the increase in AIDS incidence. On any day, more than 1,200 people with AIDS or AIDS-related illnesses occupy city hospital beds, including 420 in municipal hospitals. The annual cost of treating AIDS patients here is projected to reach $1 billion a year by 1991.

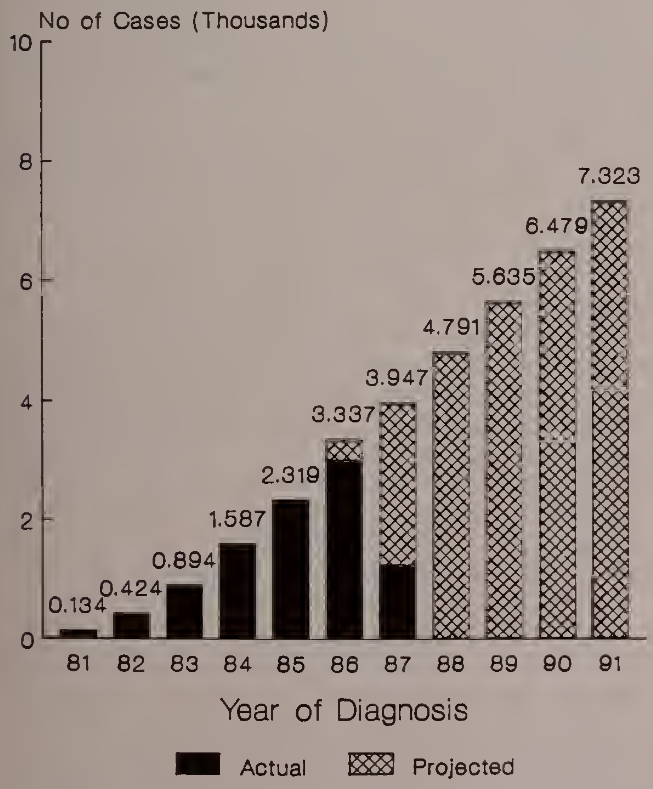

FIGURE 7. AIDS incidence in the male population (August 17, 1987).

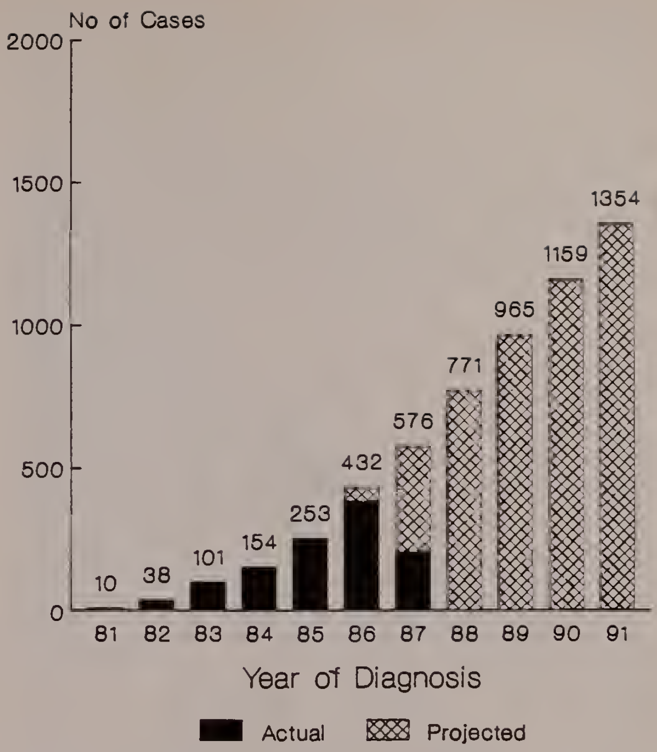

FIGURE 8. AIDS incidence in the female population (August 17, 1987).

The HIV-related patient census in the Health and Hospitals Corporation (HHC) is expected to rise at least 20%

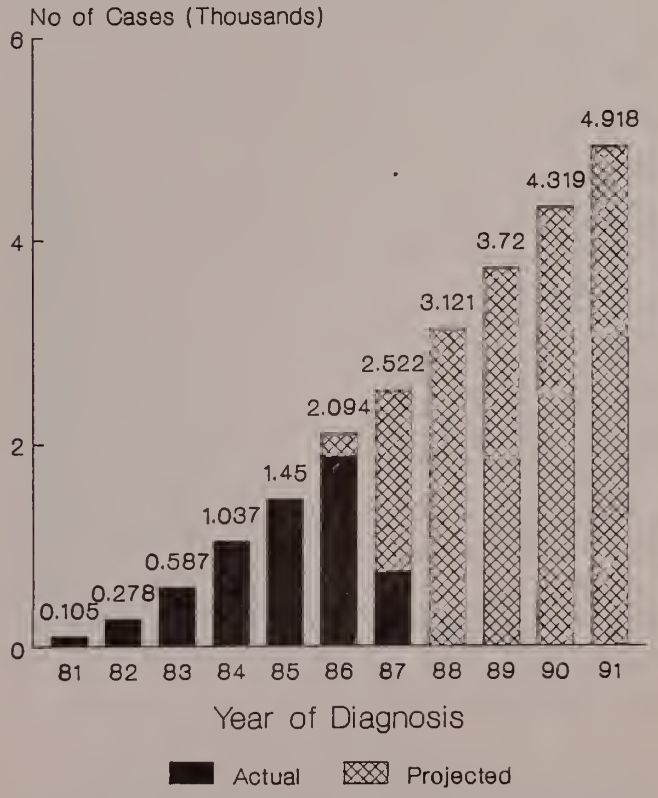

FIGURE 9. AIDS incidence in the male homosexual, non-intravenous drug abuser population (August 17, 1987).

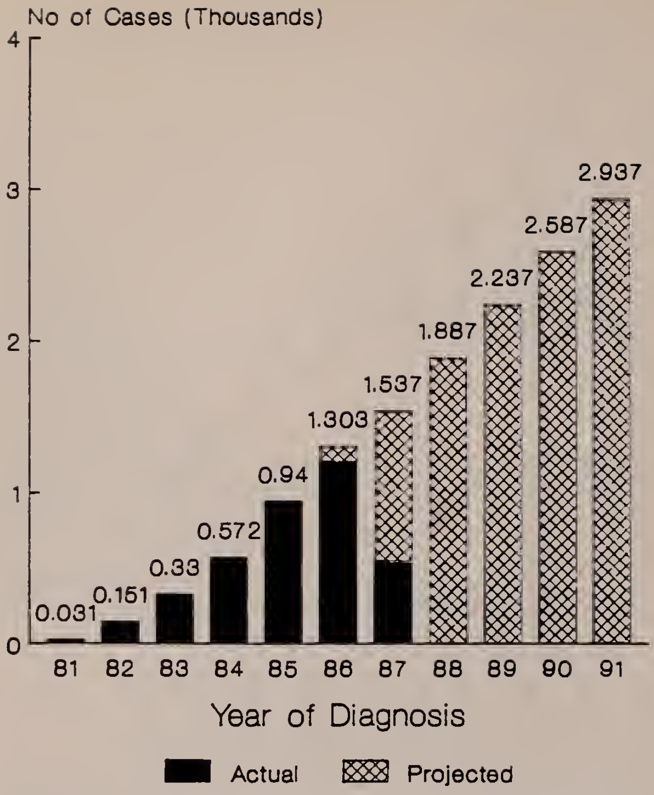

FIGURE 10. AIDS incidence in the intravenous drug abuser population (August 17, 1987).

annually, including a large share of substance abusers with poorer health status. Already, 75% of AIDS patients at HHC hospitals contracted the virus from IV drug use, including direct drug use, sexual contact with a drug user, or being born to an intravenous drug abuser or sex partner. With inadequate federal and state reimbursement, the burden of funding for unreimbursed care for AIDS patients will continue to fall on the city, and especially its public hospitals.

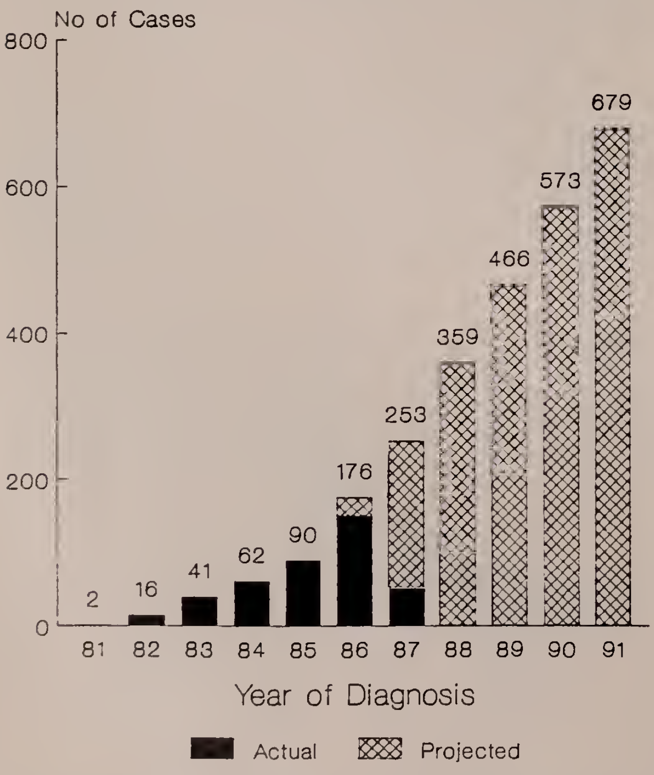

FIGURE 11. AIDS incidence among persons aged 13–24 years (August 17, 1987).

For the increasing numbers of patients with AIDS, HHC provides AIDS-related clinical assessment, complete inpatient and outpatient medical care, and extended care services. The Human Resources Administration provides emergency shelter and assists in housing matters and AIDS-related problems with family and children's services. The Department of Mental Health is developing outpatient clinics and day-treatment services for persons with AIDS, and for families and friends affected by AIDS. The AIDS Discrimination Unit of the City Human Rights Commission is handling an increasing number of complaints of AIDS-related discrimination.

PUBLIC HEALTH EDUCATION

A pattern of discrimination against people known to be or even suspected of being infected with HIV has emerged in employment, education, housing, and health care and financial services. To eliminate this, AIDS education efforts must be increased, and legislation supported that protects against discrimination and unauthorized or inappropriate disclosures of confidential health records.

New York City has mounted an all-out effort to stop the spread of HIV infection. This effort includes several elements.

First, the New York City Department of Health is increasing public health education risk reduction efforts. In fiscal 1988, projected spending for the department's AIDS prevention activities will be $8.4 million. This includes $7.1 million for a range of public health education programs that direct education to the general public and target special programs to people engaged in high-risk sex and drug behavior.

Recently, the department launched the second in a series of frank multimedia advertising campaigns, this one directed at intravenous drug users. The first series promoted the use of condoms among heterosexuals to prevent the spread of AIDS. A third campaign will soon promote abstinence among adolescents, and a fourth will reinforce the prevention message to homosexual men.

The department is increasing its outreach program to people practicing high-risk behaviors. Public health educators take the message of prevention into neighborhoods where IV drug use is high to reach drug users and their sex partners. This complements the state's activities in directly providing AIDS education to intravenous drug users in treatment programs, shooting galleries, and on the streets. The outreach program is expanding from nine to 15 communities and works with many citywide and local groups to reach those at risk through drug abuse.

Half of the roughly 100,000 people who move through the city correctional system each year are current or former IV drug users, and 50–60% of these are estimated to be HIV-positive. A Health and Correction Departments program educates city jail inmates about AIDS and distributes condoms to those at highest risk of infection to slow transmission in and out of jail. Prison Health Services offers inmates risk-reduction counseling and voluntary, confidential HIV testing.

To reach people before they take part in unsafe sex, public health educators distribute educational literature and condoms at sex clubs, massage parlors, and other places where patrons seek multiple sex encounters or unsafe sex.

RISK-REDUCTION COUNSELING

A critical element of prevention efforts is an aggressive, extensive program to increase voluntary, confidential risk-reduction counseling and HIV antibody testing. The New York City Department of Health's intensive voluntary, confidential counseling and HIV antibody testing helps to increase people's awareness of their risk and encourages them to change their behavior to reduce the chance of becoming infected or infecting others.

Testing is available at free, anonymous test sites, including the department's three Anonymous Counseling and Testing Sites (to expand to five in fiscal 1988), through any licensed physician, and at the department's sexually transmitted disease clinics. In fiscal 1987, the department's Bureau of Laboratories performed a total of 108,635 tests, approximately 13 times as many as in fiscal 1986. In fiscal 1988, the department will be able to test more than 450 specimens a day because of increased funding; currently, an average of 300 samples are tested daily.

The department's contact notification program actively helps HIV-infected people to notify their contacts. This service will soon be offered to the AIDS/ARC patients of private physicians.

The department's counseling and testing policy recommends that physicians routinely discuss AIDS infection and risk-avoiding behavior with their patients, and offer counseling and testing to patients at risk. Thus far, this has not occurred to the extent mandated by the epidemic; fewer than 3,000 of the 25,000 city physicians have sent blood specimens of at least one of their patients to the department's laboratory for testing. Physicians must assume a greater role in helping to contain the spread of HIV infection.

REFUSAL TO TREAT AIDS PATIENTS

A small number of physicians and other health care workers have refused to treat patients known or thought to be infected with the AIDS virus. This threatens the assumptions on which our health care system is based and cannot be tolerated. Comprehensive education programs must destigmatize AIDS among health workers. The department has been working with health providers to ensure that they understand the CDC precautions.

CONCLUSIONS

The latest statistics on AIDS and drug abuse-related deaths must signal a renewed effort to break the AIDS-IV drug connection. The problems of narcotic addiction make it difficult to apply traditional public health measures to control the spread of infection in this population.

Critical to the prevention effort must be linking AIDS public health education programs to drug treatment programs. Currently, there are only 225 inpatient drug detoxification beds in the city, and only 30,000 methadone maintenance slots. Waiting periods for admission range from weeks to months. There are currently only 3,800 beds for those needing long-term residential treatment after detoxification.

I have advocated expanded drug treatment programs to prevent AIDS since the beginning of my tenure as Commissioner of Health. Although we have seen modest increases from the state government, and the promise of innovative federal programs, the effort to prevent the spread of AIDS through drug treatment programs is currently insufficient.

According to the New York State Department of Substance Abuse Services, 8% of seronegative persons who are IV drug abusers seroconvert each year. We need to approach drug treatment interventions in HIV prevention with a greater urgency. We continue to advocate a needle exchange program with the New York State Commissioner of Health; while we must explore this option, the most important step would be the rapid and large-scale expansion of capacity in substance abuse treatment facilities.

The New York City Department of Health is also expanding its efforts to investigate the nature of HIV and its transmission, as well as to assess how well risk-reduction messages change behavior. It is attempting to eliminate the false dichotomy between civil liberties and public health. Opposition to mandatory antibody testing is based on sound public health principles. With no effective treatment available, mandatory testing would inefficiently use resources and would drive people away from the public health system.

Finally, the department is increasing coordinated planning and funding at the federal, state, and local levels. An interagency task force coordinates the city's AIDS programs and services. Spending for treatment, testing, counseling, education, and other programs in fiscal 1988 will be over $385 million, of which $98 million is city funds. This is up from $250 million, $75 million of which was city tax levy, in fiscal 1987. In 1989, we project continuing increases in city funding for AIDS programs, as well as a rise in AIDS funding for the city from federal sources.

Even this will not be enough to keep up with the epidemic. We have been fortunate in this city to have the vigorous support of the mayor for our policy and program initiatives. Analogous leadership has not come at the federal level. A significant federal commitment is long overdue. We need nothing less than a bold and comprehensive national prevention strategy. It must inhibit the spread of the virus among heterosexuals, and reduce the toll among homosexual men and intravenous drug abusers and their sex partners and children.

Implementing a national prevention strategy is already within our reach. The strategy should include three major elements:

- A massive national public health education program consisting of an "outer shell" of information to the general public and an "inner shell" of targeted education for people practicing high-risk behavior.
- Rapid expansion of voluntary, confidential counseling and HIV antibody testing into every public and private clinical facility, including physicians' offices, hospital outpatient departments, sexually transmit-

ted disease clinics, family planning and abortion clinics, and anonymous test sites.

- Major efforts to curtail AIDS transmission via the intravenous drug user. Efforts must range from interdiction at the international level, to law enforcement at all levels, to more education programs, increased and liberalized methadone maintenance, and rapid and massive detoxification programs, plus availability of clean needle exchange.

A national prevention strategy demands a "moon-shot" approach to federal resource commitment, guided by aggressive, articulate, and visible leadership from the highest levels of the federal administration. With the exception of Surgeon General Koop, that leadership has so far been lacking.

Without a vigorous, comprehensive national prevention program within the next 18 to 24 months, we will fall behind in the epidemic among heterosexuals, as we have with homosexual men and intravenous drug users. Seizing the opportunity now would save large numbers of lives and substantially reduce the enormous burdens and costs of the epidemic into the mid-1990s.

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The impact of AIDS on the health care system in New Jersey*

STEVEN R. YOUNG, MSPH

The goal of the New Jersey State Department of Health (NJSDH) with regard to an acquired immunodeficiency syndrome (AIDS) health care delivery system is to "develop an integrated network of AIDS health services that will provide for acute care and cost-effective, post-hospitalization, community-based care for AIDS/ARC patients, families and significant others." This is a lofty goal which will require much more effort before it is reached.

The issues surrounding this goal can be viewed from three broad perspectives: the cost of that care to New Jersey's system of health care and service delivery; the direct impact on persons and institutions who have a role and legal responsibility in providing treatment and care; and the broad spectrum of health and social service needs of persons with AIDS (PWAs).

In addition, the financial burden of AIDS has caused widespread concern. Despite this, data on expenditures for services are surprisingly scarce. In New Jersey, several programs are presently studying this issue. The Robert Wood Johnson Foundation–AIDS Health Services Program for Newark and Jersey City will be studying cost-effectiveness and appropriateness of community-based services, as will New Jersey's "Home and Community-based Services Model Waiver for Persons with AIDS or ARC," administered by the Department of Human Services. Several institution-based studies are taking place in an effort to document costs of AIDS acute care and to develop an AIDS-specific diagnosis related group (DRG) rate for reimbursement. Service programs, such as a residential AIDS drug treatment program and a pediatric AIDS residential/respite/foster care facility, both funded by the NJSDH, will be subject to cost-effectiveness studies. Most recently, the Health Research and Education Trust, an affiliate of the New Jersey Hospital Association, approached the NJSDH to assist in the formulation of a study proposal focused on utilization and cost of services for PWAs.

The purpose of this paper is to describe the interrelationship between financing care, the delivery of needed services, and education for providers and the public alike, not only for prevention, but to sensitize people to the important issues so that they will be more supportive of the services that need to be operationalized. The one benefit of our experience with an ever-increasing caseload is that more and more persons are being personally touched by the disease and have become committed to the effort.

OVERVIEW OF AIDS IN NEW JERSEY AND ITS IMPACT ON THE PROVISION OF CARE

In New Jersey, a majority of human immunodeficiency virus (HIV) infection is intravenous (IV) drug related. Nearly 41% of the state's 2,500 reported AIDS cases have occurred among male intravenous drug users, and nearly 10% have been among female IV drug users. Another 7%

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* This article is based on a presentation at the Second Montefiore Symposium on AIDS, "AIDS in the Tri-State Area: A Regional Portrait of the Epidemic," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held November 5, 1987, at the New York Academy of Medicine.

of New Jersey's AIDS cases have resulted from heterosexual transmission, and nearly 3% more have involved children with an infected parent. Drug use is implicated in a majority of these cases as well, bringing the total percentage of drug-related AIDS cases in the state to over 60% (Fig 1). New Jersey's proportion of AIDS cases in each of these drug-related categories (male IV drug user, female IV drug user, heterosexual transmission, child with parent at risk) is roughly double that found in the United States as a whole. In New Jersey's largest city, Newark, more than 70% of the AIDS cases are among IV drug users, exclusive of sexual partners or children.

The delivery of services to people with AIDS or AIDS-related complex (ARC) in New Jersey is complicated by three factors. The first is discrimination against people with AIDS that results from ignorance and fear on the part of the public and providers alike. Among its consequences are eviction from housing, loss of employment, refusal of ambulance services, and denial of medical services.

Poverty is a second factor that stands between many people with AIDS and the services they need. In New Jersey, poverty is especially severe in inner-city minority populations. The infant mortality rate in Newark, for example, which can be taken as an indicator of the general state of nutrition and health, is about 18/1,000, or nearly twice the New Jersey average.[1] These figures suggest that this population in general lacks a wide range of services, including timely and appropriate health care; shelter and food; social and community support, both emotional and material; and help in conducting daily activities. If these basic human needs (the last generally applying only to the young, the old, and the infirm) frequently go unmet in Newark's non-HIV-infected population, they are certain to pose even more serious problems for those who are infected. The latter category includes a disproportionate number of blacks. Overall, Newark's population is 56% black, 19% Hispanic, and 23% white, but its reported AIDS cases are 80% black, 12% Hispanic, and 7% white.[2]

A third factor that complicates service delivery to New Jersey's people with AIDS is that many of them are IV drug users. A number of problems arise when AIDS is associated with drug use. It is difficult to gain access to IV drug users, partly because their activity is illegal. Once they have entered the treatment system, there is a need to treat their addictions as well as their HIV-related illnesses. IV drug users frequently live on the streets, and this complicates their placement from acute care hospitals following episodes of severe illness. More than other people with AIDS (many of whom experience dementia), IV drug users suffer from impaired judgment as a result of their drug use. The deficits of community support, good nutrition, and good health that are typical in the inner city are amplified for IV drug users. Indeed, IV drug users typically present themselves at acute care facilities relatively late in the course of their HIV infections, resulting in medical complications more severe and lifespans shorter than those of other people with AIDS. And like most of the unemployed, IV drug users typically lack private insurance or personal resources to cover the costs of their health care.

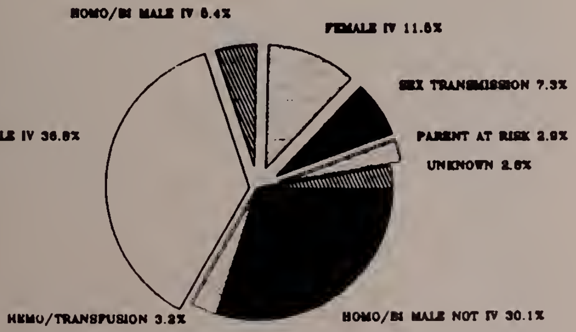

FIGURE 1. New Jersey AIDS cases by risk factor, as of December 1, 1987.

Similar problems are experienced by children born with AIDS or ARC. Many have unstable home situations: Their parents may be IV drug users, deceased, poor at parenting, or unable to cope with AIDS. As with IV drug users, this results in longer-than-necessary hospital stays following episodes of acute illness. Because children with AIDS have poorly developed immune systems, they experience different and more frequent illnesses than adults with AIDS/ARC.

Due to the IV drug use-AIDS connection in New Jersey, extreme pressures exist to provide and finance care. Additional specific factors include the presentation of different medical problems among IV drug users, including a higher incidence of *Pneumocystis carinii* pneumonia, which requires more intense hospital care; and the inability to appropriately discharge IV drug users due to lack of homes and community services to support them. This increases length of stay, which is reported to be anywhere from 12 days to 26 days in New Jersey hospitals with a range of three days to over one year, and acute care costs, since much of that care is inappropriate. Outpatient services need to be carefully structured to provide effective service for those that do not have a community placement. Another factor is the growing number of pediatric AIDS cases, which also require more care than adult cases.

In the absence of a cure or vaccine for AIDS, every effort must be made to prevent the spread of HIV infection. In New Jersey, this is best accomplished through the prevention and control of drug abuse. In the current fiscal year, funding for drug treatment in New Jersey has increased 33% to support increased treatment slots and enhanced reimbursement for those slots. Of course, more money for more drug abuse treatment is a simplistic approach that does not take into consideration how to increase slots, how to get more people into treatment via outreach, and the development of new treatment approaches. The Treatment and Community Support Unit, Division of Narcotic and Drug Abuse Control, has been among the innovators in AIDS prevention among IV drug users. NJSDH initiatives conceptualized and developed by this group include:

- the use of ex-addict street educators to take AIDS prevention messages to current addicts;
- the use of AIDS coordinators to give risk reduction messages to IV drug users in treatment, serve as a community education resource, provide training for other drug treatment center staff, and provide advocacy for patients;

- a coupon program to entitle addicts not recently in treatment to a free initial drug detoxification and an AIDS prevention class; and
- mobile outreach vans to go to areas frequented by IV drug users and give basic health examinations, prevention information, and referrals.

In light of the AIDS epidemic, new strategies for treatment that are more attractive to addicts need to be developed. In fact, drug treatment centers may need to become primary care centers with a full complement of AIDS professionals.

FINANCIAL IMPACT ON HOSPITALS AND HEALTH CARE PROVIDERS

The clinical course of AIDS is characterized by a progressive decline in immune system competence and repeated episodes of severe opportunistic infections which lead to hospitalization for diagnosis and therapy. The episodic nature of the disease necessitates a full spectrum of health and social support services. A large share of the rapidly escalating costs for such services in New Jersey will fall on Medicaid and uncompensated care. Approximately 50% of patients in northern New Jersey hospitals are enrolled in the State Medicaid Program. Through New Jersey's hospital rate setting and uncompensated care system, paying hospital patients and insurers (private and public) have had to bear financial responsibility for the state's uninsured PWAs.

New Jersey's system operates on the basis of prospective hospital payments that are determined by a patient's admitting diagnosis. The reimbursement rates that correspond to DRGs are the same for all payers, though they vary from one hospital to another. (No DRGs have yet been established specifically for people with AIDS.) The reimbursement system has one other outstanding feature: No patient is turned away from a New Jersey hospital because of an incapacity to pay for his or her care. To cover the costs of care for those who cannot afford it, New Jersey has instituted a system whereby all payers to hospitals (patients, insurance companies, state, etc) pay a surcharge on each calculated rate to cover the costs of uncompensated care. The uncompensated care system operates only for acute care facilities; it does not cover the costs of nursing facilities or home care, for example.

The cost of AIDS, direct and indirect, has been relatively low to date when compared with the economic costs of all other illnesses. However, the cost of AIDS treatment per case is at the top of the list of major diseases, along with other expensive illnesses such as renal disease and the terminal stages of certain cancers. Estimated lifetime treatment costs have ranged from $29,000 to $147,000. This wide variation is due, in part, to differences in length of hospital stays in different geographic areas of the country. One of the factors holding down treatment costs in New Jersey is that IV drug user patients present late to the health care system and die quickly. This is not a comforting measure of cost control. On the other hand, there is less likelihood that IV drug users with AIDS will be discharged home between acute episodes. In addition, the cost of care for pediatric AIDS cases, of which there are a significant number, is estimated by the Centers for Disease Control (CDC) to be 140% of the cost of care for adults.[3]

The real financial significance of AIDS is that the costs have not been anticipated, since those afflicted are primarily young persons with historically low rates of sickness and death. By adding a layer of unexpected cost, AIDS has confounded risk assessment and prediction for all payers and disrupted established patterns for distributing the cost burdens among them. The AIDS scene is rapidly changing in regard to the demographics of patient groups, medical interventions, treatment models, and development of state policies, making cost estimates "calculated guesses." What is certain, however, is that given the health and social service needs of PWAs, the barriers to service, and the financial implications of what is occurring, high priority must be placed upon developing a continuum of services outside of hospitals for PWAs.

INTERRELATIONSHIPS BETWEEN THE FINANCING OF CARE AND THE DELIVERY OF SERVICES

Acute care for AIDS patients has fallen disproportionately on public and church institutions in inner-city areas of northern New Jersey. This phenomenon has been referred to as the "ghettoization of PWAs." Green[4] has indicated that by 1991, 2% of all medical/surgical beds will be occupied by AIDS patients. In New Jersey, the rate at one institution is currently at 50%. Five hospitals have cared for more than 100 AIDS patients, six hospitals have cared for 50–100 patients, 15 hospitals have cared for 25–50 patients, and most other hospitals have cared for at least one patient. Much of this trend is due to the demographics of the disease; however, to some extent we are aware of the inappropriate referrals of PWAs. Patient referral may sometimes be due to the "inability to provide appropriate care." Inappropriate referrals which essentially deny access to care, and are the result of HIV routine testing without informed consent or suspicion of positivity, are clearly unethical and intolerable.

High caseloads—ones that are rapidly increasing—cause concern about the operational aspects of hospitals. Similar concerns exist in other community-based health care agencies. There are staffing concerns; institutions have recognized the difficulty in recruiting teams of professionals necessary to work with PWAs. Ongoing training and support systems are necessary. The development of multidisciplinary care teams for PWAs is one of the greatest challenges facing the health care system.

Communities in New Jersey have accepted the fact that PWAs are treated in hospitals. But what about the future? As caseloads increase disproportionately in some hospitals, they may lose other patients who have freedom of choice and choose not to be treated in an "AIDS hospital."

The NJSDH has maintained that acute care should be shared throughout institutions as defined by their service area and patient profile. However, as public and church institutions become hospitals of last resort, their financial viability may be threatened. Even with Medicaid and uncompensated care reimbursement, there is potential for calamitous AIDS cost burdens. Clearly, the public role of financing is increasing but inadequate. As the total bill for AIDS increases, there is a need to divide the responsibility of paying among patients, families, private payers, and government health plans. So far, the New Jersey system of reimbursement is keeping hospitals afloat financially.

In addition, there is a broad spectrum of issues related to patient care, employee safety, and provider services that may affect a hospital's liability insurance. In fact, this has already been witnessed in other AIDS care settings such as drug treatment centers and transitional homes for children.

In the long run, however, a positive approach and outlook must be maintained on New Jersey's treatment capability for PWAs. The large number of empty hospital beds in New Jersey makes it unlikely that the AIDS epidemic will completely overload the state's hospital capacity, except possibly in high impact communities such as Newark, Jersey City, and Paterson. In these cities, AIDS care has contributed to an increasing occupancy rate. Building new hospitals for AIDS may be a necessity in the future. Clearly, hospitals will not default due to the previously described reimbursement mechanism and the fact that state and local leaders realize the political fallout from such occurrences.

Increased availability of alternative levels of care is essential. In the short term this will increase costs due to startup expenses, but in the long term will be a more cost-effective approach. In fact, hospitals may choose to provide alternative levels of care themselves. Conversion of unused acute care beds, identification of "swing" beds, or use of decertified beds may be viable options. Politically and clinically, linking a continuum of care with acute care makes sense.

Hospitals in New Jersey have filed formal requests for higher reimbursement for their AIDS caseloads and a $600 increase for AIDS-related DRG admissions has been adopted. Hospitals will be strained but will not go out of business due to AIDS. What is ultimately needed is a network of providers to share responsibility for a continuum of health services that are adequately funded via existing reimbursement systems to relieve some of the burden felt on the acute care side.

NEW JERSEY PLANS

Since most of the burden of financing medical care for persons with AIDS who lack sufficient coverage now falls on the state, we will continue to develop innovative strategies in providing appropriate care. Methods used include the following:

- seed money for start-up demonstration programs;
- providing financial support for AIDS staff through contracts;
- provision of training and consultation to local agencies' staff in order that they may assume AIDS-related functions;
- creation of enhanced reimbursement rates.

The major barriers in developing a continuum of care fall into two broad categories:

- lack of political/community support;
- lack of adequate financing mechanisms.

The broad range of services needed indicates the complexity of caring for persons afflicted with this disease. In some instances, completely new types of resources must be created and questions arise as to how they can be licensed and reimbursed for operational costs. A description of New Jersey's programmatic efforts is presented in *AIDS in New Jersey: A Report from the Department of Health*.[5] The report illustrates the wide range of services and their potential funding source. Services include case management, outpatient ambulatory care, medical day care, dental care, home health care, skilled nursing, personal care assistance, therapy, hospice, pediatric residential/respite/foster care, residential AIDS drug treatment, adult residential housing, long term care, volunteer buddy support, early intervention (children), transportation, counseling/mental health, legal counsel, pastoral care, and drug abuse home treatment. Many services are not reimbursed, and some are reimbursed at an inadequate level. Reimbursement sources include federal, state, third party Medicaid, and private foundation monies.

In New Jersey, an uncompensated care pool has been used to share the financial risk among acute care hospitals and third party payers. Our State Medicaid Program instituted a "Home and Community-based Services Model Waiver for Persons with AIDS or ARC," which provides for reimbursement of cost-effective services that meet some of the needs of our patient population: case management, skilled nursing at home, personal care assistance, medical day care, drug abuse treatments at home, and foster care for children. The program, along with a system of in-hospital case management, is attempting to reduce the need for hospitalization, thus reducing costs and enhancing the quality of life for AIDS patients. It is currently too early to provide a demonstration of success. On the negative side, there are many instances where there are no options for case management and referral of clients—institutionalized hospice, long term care, housing, or subacute unit.

There are many programmatic areas where further attention is needed. The NJSDH proposes in the near future to:

- coordinate a planned effort to educate, train, and desensitize primary care physicians, nurses, aides, social workers;
- study the costs of care, the medical procedures that contribute to those costs, and the methods of payment used by people with AIDS;
- implement a statewide mass media/public education campaign not only for purposes of prevention, but to elicit support for the necessary complement of health services;
- conduct feasibility studies to determine providers/sites of alternative levels of care. Such a study has taken place for the City of Newark, in conjunction with both the city and the New Jersey Health Care Facilities Financing Authority, and conducted by a private, independent consulting firm;
- conduct a periodic census of hospitals to determine the demand for AIDS-related care and identify inappropriate referral patterns.

SUMMARY

The New Jersey State Department of Health will continue to facilitate the creation of health care sites outside of hospitals for people with AIDS. This will relieve some of the burden carried by acute care inner-city institutions and provide more appropriate and humane care. In addition, we are urging hospitals to work with us in performing prospective studies to determine reliable cost estimates.

Important questions remain unanswered. How will cost burdens be shared among individuals, private hospitals, HMOs, local/state/federal governments, and insurance companies and their investors? In Chicago, the AIDS Medical Resource Center is doing some innovative work in cost determinations based on patients' needs and negotiating for a prepaid lifetime reimbursement with third party payers. Services are provided via a network of providers who are contractually linked and who share the financial risk should their costs exceed the negotiated fee. Will access to care be equitable? Will we have appropriately trained health care workers for many settings? Will the shortage of nurses affect our ability to provide care? Will insurance companies require HIV testing for new policyholders, thereby resulting in an increase in patients without coverage, or will such testing be prohibited, with insurance companies raising their charges for all so as to cover risk? Some people will then not be able to afford a health insurance policy.

These issues deserve consideration from a variety of health professionals, especially in regard to their role and responsibility in addressing them.

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FROM THE LIBRARY

THE HEALTH OF THE NATION

The health of the nation seems rather a large topic, yet not too large for hopeful and practical consideration, nor yet so large as would be a kindred topic, the health of all nations, which is likewise one for serious and hopeful consideration. Indeed, these two subjects have a direct bearing one upon another, and one cannot be adequately considered without considering the other.

It is a somewhat trite idea, but one whose significance is of great present import, that the nations of the earth today are more nearly related than ever before in the world's history. "Not only has the 'narrow frith' been practically abolished," says a recent writer, "but the wide ocean is traversed by passenger ships in five days, and by thoughts put into words in a few seconds. All the world has become one neighborhood so far as relates to distances." In no manner has this been more strikingly shown than in the warfare against contagious disease. But a few years ago a violent epidemic of yellow fever in Cuba would excite no more than passing notice, while today the news of two cases in the far-off neighborhood of Santiago is immediately wired throughout the United States and foreign countries. A few cases of bubonic plague in the Orient, which a few years ago would receive no attention, are instantly reported and published throughout the United States, and one case of cholera in a ship in the Mediterranean is likewise immediately telegraphed to the principal cities of the world. International congresses, conferences and conventions are frequent, bringing the nations together as one family in the struggle against these foes of mankind. As in 1892, when a case of cholera appeared in Jersey City, the New York Board of Health took active interest therein, so is the United States Government interested when epidemic disease is reported in England, France, Germany, Turkey, Egypt, or any port in the Orient, for communication therewith is now swift and frequent. And so closely related are we in health matters to our neighbors of Mexico, Central and South America, that periodical international sanitary conventions have been agreed upon by the several republics, and a permanent international sanitary bureau of American republics has been established and is maintained. I need further to refer only to the international congresses of Medicine, of Hygiene and Demography, of Tuberculosis, Leprosy, and other allied subjects, to show how closely the nations are getting together in the efforts to prevent and suppress disease. . . .

WALTER WYMAN, MD
(*NY State J Med* 1905; 5:83–86)

Ethical issues involved in the growing AIDS crisis*

The Council on Ethical and Judicial Affairs of the American Medical Association recognizes the growing AIDS crisis as a crucial health problem involving the physician's ethical responsibility to his patients and to society. The House of Delegates adopted Report YY (A-87) of the Board of Trustees which provides excellent guidance for a responsible public policy. As stated therein, AIDS patients are entitled to competent medical service with compassion and respect for human dignity and to the safeguard of their confidences within the constraints of the law. Those persons who are afflicted with the disease or who are seropositive have the right to be free from discrimination.

A physician may not ethically refuse to treat a patient whose condition is within the physician's current realm of competence solely because the patient is seropositive. The tradition of the American Medical Association, since its organization in 1847, is that: "when an epidemic prevails, a physician must continue his labors without regard to the risk to his own health." (See Principles of Medical Ethics, 1847, 1903, 1912, 1947, 1955). That tradition must be maintained. A person who is afflicted with AIDS needs competent, compassionate treatment. Neither those who have the disease nor those who have been infected with the virus should be subjected to discrimination based on fear or prejudice, least of all by members of the health care community. Physicians should respond to the best of their abilities in cases of emergency where first aid treatment is essential, and physicians should not abandon patients whose care they have undertaken. (See Section 8.10 of *Current Opinions of the Council on Ethical and Judicial Affairs of the American Medical Association, 1986*).

Principle VI of the 1980 Principles of Medical Ethics states that "A physician shall in the provision of appropriate patient care, except in emergencies, be free to choose whom to serve, with whom to associate and the environment in which to provide medical services." The Council has always interpreted this Principle as not supporting illegal or invidious discrimination. (See Section 9.11 of *Current Opinions, 1986*). Thus, it is the view of the Council that Principle VI does not permit categorical discrimination against a patient based solely on his or her seropositivity. A physician who is not able to provide the services required by persons with AIDS should make an appropriate referral to those physicians or facilities that are equipped to provide such services.

At its 1987 Annual Meeting, the House of Delegates adopted Substitute Resolution 18 which asked the Council on Ethical and Judicial Affairs to address "the patient confidentiality and ethical issues raised by known HIV antibody positive patients who refuse to inform their sexual partners or modify their behavior." Physicians have a responsibility to prevent the spread of contagious diseases, as well as an ethical obligation to recognize the rights to privacy and to confidentiality of the AIDS victim. These rights are absolute until they infringe in a material way on the safety of another person or persons. Those who are not infected with the virus are entitled to protection from transmission of the disease. Thus, the societal need for accurate information and public health surveillance must also be respected. As the Board of Trustees stated in Report YY (A-87), "A sound epidemiologic understanding of the potential impact of AIDS on society requires the reporting [on an anonymous or confidential basis to public health authorities] of those who are confirmed as testing positive for the antibody to the AIDS virus."

In those jurisdictions in which the reporting of individuals infected with the AIDS virus to public health authorities is not mandated, a physician who knows that a seropositive patient is endangering a third party faces a dilemma. The physician should attempt to persuade the infected individual to refrain from activities that might result in further transmission of the disease. When rational persuasion fails, authorities should be notified so that they can take appropriate measures to protect third parties. Ordinarily, this action will fulfill the physician's duty to warn third parties; in unusual circumstances when all else fails, a physician may have a common law duty to warn endangered third parties. However, notification of any third party, including public authorities without the consent of the patient may be precluded by statutes in certain states. Therefore, the Council reiterates and strongly endorses Recommendations 16 and 17 of Board Report YY (A-87). They are:

> *Recommendation 16:*
>
> Specific statutes must be drafted which, while protecting to the greatest extent possible the confidentiality of patient information, (a) provide a method for warning unsuspecting sexual partners, (b) protect physicians from liability for failure to warn the unsuspecting third party but, (c) establish clear standards for when a physician should inform the public health authorities, and (d) provide clear guidelines for public health authorities who need to trace the unsuspecting sexual partners of the infected person.
>
> *Recommendation 17:*
>
> Given the risk of infection being transmitted sexually, and given the dire potential consequences of transmission, serious consideration should be given to sanctions, at least in circumstances where an unsuspecting sexual partner subsequently finds out about a partner's infection and brings a complaint to the attention of authorities. Pre-emptive sanctions are not being endorsed by this recommendation.

The civil rights and liberties of those who are infected with the AIDS virus, as well as those who are not, are entitled to protection. The ethical challenge to the medical profession is to maintain a judicious balance in this regard, including the issue of whether physicians who are HIV-infected must inform their patients or whether they may continue in patient care at all. The Council's new opinion on PHYSICIANS AND INFECTIOUS DISEASES is:

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* Reproduced from a report of the American Medical Association's Council on Ethical and Judicial Affairs, Report A (I-87). Chicago, American Medical Association, 1987. The Council of the Medical Society of the State of New York has endorsed this report.

> A physician who knows that he or she has an infectious disease should not engage in any activity that creates a risk of transmission of the disease to others.

In the context of the AIDS crisis, the application of the Council's opinion depends on the activity in which the physician wishes to engage.

The Council on Ethical and Judicial Affairs reiterates and reaffirms the AMA's strong belief that AIDS victims and those who are seropositive should not be treated unfairly or suffer from discrimination. However, in the special context of the provision of medical care, the Council believes that if a risk of transmission of an infectious disease from a physician to a patient exists, disclosure of that risk to patients is not enough; patients are entitled to expect that their physicians will not increase their exposure to the risk of contracting an infectious disease, even minimally. If no risk exists, disclosure of the physician's medical condition to his or her patients will serve no rational purpose; if a risk does exist, the physician should not engage in the activity.The Council recommends that the afflicted physician disclose his or her condition to colleagues who can assist in the individual assessment of whether the physician's medical condition or the proposed activity poses any risk to patients. There may be an occasion when a patient who is fully informed of the physician's condition and the risks that condition presents may choose to continue his or her care with the seropositive physician. Great care must be exercised to assure that true informed consent is obtained.

In summary, the Council on Ethical and Judicial Affairs believes that:

- A physician may not ethically refuse to treat a patient whose condition is within the physician's current realm of competence solely because the patient is seropositive. Persons who are seropositive should not be subjected to discrimination based on fear or prejudice.
- Physicians are dedicated to providing competent medical service with compassion and respect for human dignity.
- Physicians who are unable to provide the services required by AIDS patients should make referrals to those physicians or facilities equipped to provide such services.
- Physicians are ethically obligated to respect the rights of privacy and of confidentiality of AIDS patients and seropositive individuals.
- Where there is no statute that mandates or prohibits the reporting of seropositive individuals to public health authorities and a physician knows that a seropositive individual is endangering a third party, the physician should: (1) attempt to persuade the infected patient to cease endangering the third party; (2) if persuasion fails, notify authorities; and (3) if the authorities take no action, notify the endangered third party.
- A physician who knows that he or she is seropositive should not engage in any activity that creates a risk of transmission of the disease to others.
- A physician who has AIDS or who is seropositive should consult colleagues as to which activities the physician can pursue without creating a risk to patients.

Counseling patients about the prevention of AIDS*

The Food and Drug Administration (FDA) is urging that physicians and other health professionals help educate patients about ways to prevent the spread of human immunodeficiency virus (HIV). It should be stressed to patients that because acquired immunodeficiency syndrome (AIDS) is a sexually transmitted disease (STD), sexual abstinence or a mutually monogamous relationship with an uninfected partner is the best insurance against acquiring the disease (except when one partner is an intravenous drug abuser, in which case mutual monogamy offers no protection).

CONDOMS

For sexually active persons, the only instance when condoms are unnecessary for reduction of infection risk is within a longstanding, mutually monogamous relationship in which neither partner uses IV drugs and neither partner is infected with HIV. This applies to any sexual activity where the exchange of semen and/or blood is possible, including vaginal, anal, and oral sex.

Recent studies reported in the press have indicated that natural membrane condoms are not able to contain—and, therefore, are unable to protect against infection from—the HIV virus. Therefore, FDA allows only latex condoms to be labeled for the prevention of STDs, including AIDS.

Because the diameter of the HIV virus is about 1/25th that of spermatozoa, there was initially some question about whether condoms could contain the virus. Subsequently, however, *in vitro* tests of latex condoms at the University of California at San Francisco[1] and epidemiologic data[2,3] confirmed that the HIV virus does not pass through an intact latex condom.

To maximize protection against STDs, it is important that condoms be used properly. FDA sent a letter to all US condom manufacturers, importers, and repackagers regarding the labeling of condoms for prevention of STDs. The letter stated that only latex condoms could be labeled for the prevention of STDs, including AIDS. FDA does not allow natural membrane condoms to be so labeled because they have a different permeability than latex and may not lend themselves to the same degree of uniformity. However, regardless of the product's labeling and composition, FDA requested that all condoms contain adequate instructions for use to maximize the degree of protection they afford.

The Agency suggested the following as an acceptable labeling statement for latex condoms:

> When used properly, the latex condom may prevent the transmission of many sexually transmitted diseases (STDs) such as syphilis, gonorrhea, chlamydial infections, genital herpes, and AIDS. It cannot eliminate the risk. *For maximum protection, it*

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* This material is reprinted from the *FDA Drug Bulletin*, September 1987. The Council of the Medical Society of the State of New York has approved the content of this publication on the recommendation of the society's Committee on Preventive Medicine.

is important to follow the accompanying instructions. Failure to do so may result in loss of protection. During intimate contact, lesions and various body fluids can transmit STDs. Therefore, the condom should be applied before any such contact.

In its letter to manufacturers, FDA provided the following example as an acceptable set of instructions:

- Use a condom every time you have sexual intercourse or other acts between partners which involve contact with the penis.
- Put the condom on after the penis is erect and prior to intimate contact, because lesions, pre-ejaculate secretions, semen, vaginal secretions, saliva, urine, and feces can contain sexually transmitted disease (STD) organisms.
- Place the condom on the head of the penis and unroll or pull it all the way to the base.
- Leave an empty space at the end of the condom to collect the semen. Remove any air remaining in the tip of the condom by gently pressing the air out towards the base of the penis.
- If a lubricant is desired, use water-based lubricants such as ——. *Do not use oil-based lubricants*, such as those made with petroleum jelly, mineral oil, vegetable oil, or cold cream, as these may damage the condom.
- After ejaculation, carefully withdraw the penis while it is still erect. Hold onto the rim of the condom as you withdraw so that the condom does not slip off.
- Store condoms in a cool, dark, dry place.
- If the rubber material is sticky or brittle, discolored, or obviously damaged, do not use it.
- Do not reuse condoms.

INFORMATIONAL MATERIALS

Government and private agencies have developed a number of informational materials that health professionals may want to have on hand to help answer patients' questions.

Up to 50 free copies of the following materials, produced jointly by the US Public Health Service (PHS) and the American Red Cross, can be obtained from AIDS, Suite 700, 1555 Wilson Blvd, Rosslyn, VA 22209:

- Poster featuring Patti LaBelle with Toll-free AIDS hot line number.
- Leaflets:
  AIDS, Sex and You
  Facts About AIDS and Drug Abuse
  AIDS and Your Job—Are There Risks?
  Gay and Bisexual Men and AIDS
  AIDS and Children—Information for Parents of School Age Children
  AIDS and Children—Information for Teachers and School Officials
  Caring for the AIDS Patient at Home
  If your Test for Antibody to the AIDS Virus is Positive...

In addition, PHS has developed the following materials:

- Surgeon General's Report on AIDS (October 1986) from AIDS, PO Box 14252, Washington, DC 20004 (up to 50 free copies)
- Facts About AIDS from AIDS, Suite 700, 1555 Wilson Blvd, Rosslyn, VA 22209 (up to 50 free copies)

Up to 25 free copies of the following scriptographic booklets are available from the Office of Public Inquiries, CDC, Bldg 1, Room B-63, 1600 Clifton Rd, Atlanta, GA 30333:

- What Everyone Should Know About AIDS (also available in Spanish)
- Why You Should Be Informed About AIDS (for health care workers)
- What Gay and Bisexual Men Should Know About AIDS
- AIDS and Shooting Drugs

The following videotapes may be purchased for $55 each from the National Audiovisual Center, 8700 Edgeworth Dr, Capitol Heights, MD 20743-3701; Attn: Customer Service. They may also be borrowed free from Modern Talking Picture Service, 5000 Park St North, St Petersburg, FL 33709; Attn: Film Scheduling:

- AIDS: Fears and Facts (for the general public)
- What If the Patient Has AIDS? (for health care workers)
- AIDS and Your Job (for policemen, firemen, and other emergency personnel)

Practitioners may want to inform patients of the number of the AIDS toll-free national hot line: (1-800) 342-AIDS, and may want to check with the local health department regarding local hot lines.

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1. Conant M, et al: Condoms prevent transmission of AIDS-associated retrovirus [letter]. *JAMA* 1986; 255:1706.
2. Fischl MA, et al: Evaluation of heterosexual partners, children, and household contacts of adults with AIDS. *JAMA* 1987; 257:640-644.
3. Mann J, et al: Condom use and HIV infection among prostitutes in Zaire [letter]. *N Engl J Med* 1987; 316:345.

Immunizations for children with HIV infections*

Addressing theoretical concerns that vaccination with live, attenuated vaccine viruses may produce serious adverse events in symptomatic HIV-infected children, and that antigenic stimulation might lead to a deterioration of clinical status of HIV-infected children,[1,2] the Immunization Practices Advisory Committee (ACIP) has established recommendations for immunizations of HIV-infected children in the United States.

The complete recommendations were published in the *MMWR* of Sept 26, 1986.[3] The following is a summary of the recommendations for oral polio vaccine (OPV), inactivated polio vaccine (IPV), and vaccines for measles, mumps, and rubella (MMR), bacille Calmette-Guérin (BCG), diphtheria, pertussis, and tetanus (DPT), haemophilus influenzae type B (Hib), influenza, and pneumococcus.

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* This material is reprinted from the *FDA Drug Bulletin*, September 1987. The Council of the Medical Society of the State of New York (MSSNY) has approved the content of this publication on the recommendation of the society's Committee on Preventive Medicine.

Children with Symptomatic HIV Infections

- *OPV, MMR, BCG* and other live vaccines should not be given to children and young adults who are immunosuppressed as a manifestation of HIV infection. These persons should receive IPV and should be excused for medical reasons from regulations requiring measles, rubella, and/or mumps immunization.
- *DPT, IPV, Hib*: The potential benefits of immunization outweigh the theoretical concerns that stimulation of the immune system by immunization with inactivated vaccines might cause deterioration in immune function. Such effects have not been noted thus far among children with AIDS or other immunosuppressed individuals. Immunization with DTP, IPV, and Hib vaccines is recommended, although immunization may be less effective than in immunocompetent children.
- *Flu, pneumococcal vaccines*: As with other conditions producing immunosuppression, annual immunization with inactivated influenza vaccine is recommended for children over 6 months of age, and one-time administration of pneumococcal vaccine is recommended for children over 2 years of age.
- *Immune globulins*: As with other immunosuppressed patients, children with clinical manifestations of HIV infection may be at increased risk of having serious complications of infectious diseases such as measles and varicella and therefore, following significant exposure to these diseases, should receive passive immunization with immune globulin or varicella-zoster immune globulin, respectively.

Children with Previously Diagnosed Asymptomatic HIV Infection

- *MMR*: Pending further data, it is recommended that these children be vaccinated with MMR and followed for possible adverse reactions and for the occurrence of vaccine-preventable diseases, since immunization may be less effective than for other children.
- *OPV*: Available data suggest that OPV can be administered without adverse consequences to HIV-infected children who do not have overt clinical manifestations of immunosuppression. However, because family members may be immunocompromised due to HIV infection, it may be prudent to use IPV routinely to immunize these children.
- *DTP, Hib*: Immunization with DTP and Hib vaccines in accordance with ACIP recommendations is recommended.

Children Residing in the Household of a Patient with AIDS

- *OPV*: Children living with others known to be immunocompromised due to AIDS or other HIV infections should not receive OPV because they would be likely to excrete vaccine viruses that would be communicable to their immunosuppressed family members.
- *MMR*: MMR may be given to such a child because extensive experience has shown that live, attenuated MMR vaccine viruses are not transmitted from vaccinated individuals to others.

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1. ACIP: General recommendations on immunization. *MMWR* 1983; 32:1-8, 13-17.

2. Zagury D, et al: Long-term cultures of HTLV-III-infected T-cells: a model of cytopathology of T-cell depletion in AIDS. *Science* 1986; 231:850-853.

3. ACIP: Immunization of children infected with Human T-Lymphotropic Virus Type III/Lymphadenopathy-Associated Virus. *MMWR* 1986; 35:595-598, 603-606.

Guidelines for effective school health education to prevent the spread of AIDS*

Introduction

Since the first cases of acquired immunodeficiency syndrome (AIDS) were reported in the United States in 1981, the human immunodeficiency virus (HIV) that causes AIDS and other HIV-related diseases has precipitated an epidemic unprecedented in modern history. Because the virus is transmitted almost exclusively by behavior that individuals can modify, educational programs to influence relevant behavior can be effective in preventing the spread of HIV.[1-5]

The guidelines below have been developed to help school personnel and others plan, implement, and evaluate educational efforts to prevent unnecessary morbidity and mortality associated with AIDS and other HIV-related illnesses. The guidelines incorporate principles for AIDS education that were developed by the President's Domestic Policy Council and approved by the President in 1987 (see Appendix I).

The guidelines provide information that should be considered by persons who are responsible for planning and implementing appropriate and effective strategies to teach young people about how to avoid HIV infection. The guidelines should not be construed as rules, but rather as a source of guidance. Although they specifically were developed to help school personnel, personnel from other organizations should consider these guidelines in planning and carrying out effective education about AIDS for youth who do not attend school and who may be at high risk of becoming infected. As they deliberate about the need for and content of AIDS education, educators, parents, and other concerned members of the community should consider the prevalence of behavior that increases the risk of HIV infection among young people in their communities. Information about the nature of the AIDS epidemic, and the extent to which young people engage in behavior that increases the risk of HIV infection, is presented in Appendix II.

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* These guidelines were developed by the United States Public Health Service Centers for Disease Control and are reprinted from *Morbidity and Mortality Weekly Reports* 1988; 37(S-2):1-14. The Council of the Medical Society of the State of New York (MSSNY) has adopted these guidelines as MSSNY policy on the recommendation of the society's Committee on Preventive Medicine.

Information contained in this document was developed by CDC in consultation with individuals appointed to represent the following organizations:

- American Academy of Pediatrics
- American Association of School Administrators
- American Public Health Association
- American School Health Association
- Association for the Advancement of Health Education
- Association of State and Territorial Health Officers
- Council of Chief State School Officers
- National Congress of Parents and Teachers
- National Council of Churches
- National Education Association
- National School Boards Association
- Society of State Directors of Health, Physical Education, Recreation and Dance
- US Department of Education
- US Food and Drug Administration
- US Office of Disease Prevention and Health Promotion

Consultants included a director of health education for a state department of education, a director of curriculum and instruction for a local education department, a health education teacher, a director of school health programs for a local school district, a director of a state health department, a deputy director of a local health department, and an expert in child and adolescent development.

PLANNING AND IMPLEMENTING EFFECTIVE SCHOOL HEALTH EDUCATION ABOUT AIDS

The Nation's public and private schools have the capacity and responsibility to help assure that young people understand the nature of the AIDS epidemic and the specific actions they can take to prevent HIV infection, especially during their adolescence and young adulthood. The specific scope and content of AIDS education in schools should be locally determined and should be consistent with parental and community values.

Because AIDS is a fatal disease and because educating young people about becoming infected through sexual contact can be controversial, school systems should obtain broad community participation to ensure that school health education policies and programs to prevent the spread of AIDS are locally determined and are consistent with community values.

The development of school district policies on AIDS education can be an important first step in developing an AIDS education program. In each community, representatives of the school board, parents, school administrators and faculty, school health services, local medical societies, the local health department, students, minority groups, religious organizations, and other relevant organizations can be involved in developing policies for school health education to prevent the spread of AIDS. The process of policy development can enable these representatives to resolve various perspectives and opinions, to establish a commitment for implementing and maintaining AIDS education programs, and to establish standards for AIDS education program activities and materials. Many communities already have school health councils that include representatives from the aforementioned groups. Such councils facilitate the development of a broad base of community expertise and input, and they enhance the coordination of various activities within the comprehensive school health program.[6]

AIDS education programs should be developed to address the needs and the developmental levels of students and of school-age youth who do not attend school, and to address specific needs of minorities, persons for whom English is not the primary language, and persons with visual or hearing impairments or other learning disabilities. Plans for addressing students' questions or concerns about AIDS at the early elementary grades, as well as for providing effective school health education about AIDS at each grade from late elementary/middle school through junior high/senior high school, including educational materials to be used, should be reviewed by representatives of the school board, appropriate school administrators, teachers, and parents before being implemented.

Education about AIDS may be most appropriate and effective when carried out within a more comprehensive school health education program that establishes a foundation for understanding the relationships between personal behavior and health.[7–9] For example, education about AIDS may be more effective when students at appropriate ages are more knowledgeable about sexually transmitted diseases, drug abuse, and community health. It may also have greater impact when they have opportunities to develop such qualities as decision-making and communication skills, resistance to persuasion, and a sense of self-efficacy and self-esteem. However, education about AIDS should be provided as rapidly as possible, even if it is taught initially as a separate subject.

State departments of education and health should work together to help local departments of education and health throughout the state collaboratively accomplish effective school health education about AIDS. Although all schools in a state should provide effective education about AIDS, priority should be given to areas with the highest reported incidence of AIDS cases.

PREPARATION OF EDUCATION PERSONNEL

A team of representatives including the local school board, parent-teachers associations, school administrators, school physicians, school nurses, teachers, educational support personnel, school counselors, and other relevant school personnel should receive general training about a) the nature of the AIDS epidemic and means of controlling its spread, b) the role of the school in providing education to prevent transmission of HIV, c) methods and materials to accomplish effective programs of school health education about AIDS, and d) school policies for students and staff who may be infected. In addition, a team of school personnel responsible for teaching about AIDS should receive more specific training about AIDS education. All school personnel, especially those who teach about AIDS, periodically should receive continuing education about AIDS to assure that they have the most current information about means of controlling the epidemic, including up-to-date information about the most effective health education interventions available. State and local departments of education and health, as well as colleges of education, should assure that such in-service training is made available to all schools in the state as soon as possible and that continuing in-service and pre-service training is subsequently provided. The local school board should assure that release time is provided to enable school personnel to receive such in-service training.

PROGRAMS TAUGHT BY QUALIFIED TEACHERS

In the elementary grades, students generally have one regular classroom teacher. In these grades, education about AIDS should be provided by the regular classroom teacher because that person ideally should be trained and experienced in child development, age-appropriate teaching methods, child health, and elementary health education methods and materials. In addition, the elementary teacher usually is sensitive to normal variations in child development and aptitudes within a class. In the secondary grades, students generally have a different teacher for each subject. In these grades, the secondary school health education teacher preferably should provide education about AIDS, because a qualified health education teacher will have training and experience in adolescent development, age-appropriate teaching methods, adolescent health, and secondary school health education methods and materials (including methods and materials for teaching about such topics as human sexuality, communicable diseases, and drug abuse). In secondary schools that do not have a qualified health education teacher, faculty with similar training and good rapport with students should be trained specifically to provide effective AIDS education.

PURPOSE OF EFFECTIVE EDUCATION ABOUT AIDS

The principal purpose of education about AIDS is to prevent HIV infection. The content of AIDS education should be developed with the active involvement of parents and should address the broad range of behavior exhibited by young people. Educational programs should assure that young people acquire the knowledge and skills they will need to adopt and maintain types of behavior that virtually eliminate their risk of becoming infected.

School systems should make programs available that will enable and encourage young people who *have not* engaged in sexual intercourse and who *have not* used illicit drugs to continue to—

- Abstain from sexual intercourse until they are ready to establish a mutually monogamous relationship within the context of marriage;
- Refrain from using or injecting illicit drugs.

For young people who *have* engaged in sexual intercourse or who *have* injected illicit drugs, school programs should enable and encourage them to—

- Stop engaging in sexual intercourse until they are ready to establish a mutually monogamous relationship within the context of marriage;
- Stop using or injecting illicit drugs.

Despite all efforts, some young people may remain unwilling to adopt behavior that would virtually eliminate their risk of becoming infected. Therefore, school systems, in consultation with parents and health officials, should provide AIDS education programs that address preventive types of behavior that should be practiced by persons with an increased risk of acquiring HIV infection. These include:

- Avoiding sexual intercourse with anyone who is known to be infected, who is at risk of being infected, or whose HIV infection status is not known;
- Using a latex condom with spermicide if they engage in sexual intercourse;
- Seeking treatment if addicted to illicit drugs;
- Not sharing needles or other injection equipment;
- Seeking HIV counseling and testing if HIV infection is suspected.

State and local education and health agencies should work together to assess the prevalence of these types of risk behavior, and their determinants, over time.

CONTENT

Although information about the biology of the AIDS virus, the signs and symptoms of AIDS, and the social and economic costs of the epidemic might be of interest, such information is not the essential knowledge that students must acquire in order to prevent becoming infected with HIV. Similarly, a single film, lecture, or school assembly about AIDS will not be sufficient to assure that students develop the complex understanding and skills they will need to avoid becoming infected.

Schools should assure that students receive at least the essential information about AIDS, as summarized in sequence in the following pages, for each of three grade-level ranges. The exact grades at which students receive this essential information should be determined locally, in accord with community and parental values, and thus may vary from community to community. Because essential information for students at higher grades requires an understanding of information essential for students at lower grades, secondary school personnel will need to assure that students understand basic concepts before teaching more advanced information. Schools simultaneously should assure that students have opportunities to learn about emotional and social factors that influence types of behavior associated with HIV transmission.

Early Elementary School. Education about AIDS for students in early elementary grades principally should be designed to allay excessive fears of the epidemic and of becoming infected.

> *AIDS is a disease that is causing some adults to get very sick, but it does not commonly affect children.*
>
> *AIDS is very hard to get. You cannot get it just by being near or touching someone who has it.*
>
> *Scientists all over the world are working hard to find a way to stop people from getting AIDS and to cure those who have it.*

Late Elementary/Middle School. Education about AIDS for students in late elementary/middle school grades should be designed with consideration for the following information.

> *Viruses are living organisms too small to be seen by the unaided eye.*
>
> *Viruses can be transmitted from an infected person to an uninfected person through various means.*
>
> *Some viruses cause disease among people.*
>
> *Persons who are infected with some viruses that cause disease may not have any signs or symptoms of disease.*
>
> *AIDS (an abbreviation for acquired immunodeficiency syndrome) is caused by a virus that weakens the ability of infected individuals to fight off disease.*
>
> *People who have AIDS often develop a rare type of severe pneumonia, a cancer called Kaposi's sarcoma, and certain other diseases that healthy people normally do not get.*
>
> *About 1 to 1.5 million of the total population of approximately 240 million Americans currently are infected with the AIDS virus and consequently are capable of infecting others.*
>
> *People who are infected with the AIDS virus live in every state in the United States and in most other countries of the world. Infected people live in cities as well as in suburbs, small towns, and rural areas. Although most infected people are adults, teenagers can also become infected. Females as well as males are infected. People of every race are infected, including whites, blacks, Hispanics, Native Americans, and Asian/Pacific Islanders.*
>
> *The AIDS virus can be transmitted by sexual contact with an infected person; by using needles and other injection equipment that an infected person has used; and from an infected mother to her infant before or during birth.*
>
> *A small number of doctors, nurses, and other medical personnel have been infected when they were directly exposed to infected blood.*

It sometimes takes several years after becoming infected with the AIDS virus before symptoms of the disease appear. Thus, people who are infected with the virus can infect other people—even though the people who transmit the infection do not feel or look sick.

Most infected people who develop symptoms of AIDS only live about 2 years after their symptoms are diagnosed.

The AIDS virus cannot be caught by touching someone who is infected, by being in the same room with an infected person, or by donating blood.

Junior High/Senior High School. Education about AIDS for students in junior high/senior high school grades should be developed and presented taking into consideration the following information.

The virus that causes AIDS, and other health problems, is called human immunodeficiency virus, or HIV.

The risk of becoming infected with HIV can be virtually eliminated by not engaging in sexual activities and by not using illegal intravenous drugs.

Sexual transmission of HIV is not a threat to those uninfected individuals who engage in mutually monogamous sexual relations.

HIV may be transmitted in any of the following ways: a) by sexual contact with an infected person (penis/vagina, penis/rectum, mouth/vagina, mouth/penis, mouth/rectum); b) by using needles or other injection equipment that an infected person has used; c) from an infected mother to her infant before or during birth.

A small number of doctors, nurses, and other medical personnel have been infected when they were directly exposed to infected blood.

The following are at increased risk of having the virus that causes AIDS and consequently of being infectious: a) persons with clinical or laboratory evidence of infection; b) males who have had sexual intercourse with other males; c) persons who have injected illegal drugs; d) persons who have had numerous sexual partners, including male or female prostitutes; e) persons who received blood clotting products before 1985; f) sex partners of infected persons or persons at increased risk; and g) infants born to infected mothers.

The risk of becoming infected is increased by having a sexual partner who is at increased risk of having contracted the AIDS virus (as identified previously), practicing sexual behavior that results in the exchange of body fluids (ie, semen, vaginal secretions, blood), and using unsterile needles or paraphernalia to inject drugs.

Although no transmission from deep, open-mouth (ie, "French") kissing has been documented, such kissing theoretically could transmit HIV from an infected to an uninfected person through direct exposure of mucous membranes to infected blood or saliva.

In the past, medical use of blood, such as transfusing blood and treating hemophiliacs with blood clotting products, has caused some people to become infected with HIV. However, since 1985 all donated blood has been tested to determine whether it is infected with HIV; moreover, all blood clotting products have been made from screened plasma and have been heated to destroy any HIV that might remain in the concentrate. Thus, the risk of becoming infected with HIV from blood transfusions and from blood clotting products is virtually eliminated. Cases of HIV infection caused by these medical uses of blood will continue to be diagnosed, however, among people who were infected by these means before 1985.

Persons who continue to engage in sexual intercourse with persons who are at increased risk or whose infection status is unknown should use a latex condom (not natural membrane) to reduce the likelihood of becoming infected. The latex condom must be applied properly and used from start to finish for every sexual act. Although a latex condom does not provide 100% protection—because it is possible for the condom to leak, break, or slip off—it provides the best protection for people who do not maintain a mutually monogamous relationship with an uninfected partner. Additional protection may be obtained by using spermicides that seem active against HIV and other sexually transmitted organisms in conjunction with condoms.

Behavior that prevents exposure to HIV also may prevent unintended pregnancies and exposure to the organisms that cause Chlamydia *infection, gonorrhea, herpes, human papillomavirus, and syphilis.*

Persons who believe they may be infected with the AIDS virus should take precautions not to infect others and to seek counseling and antibody testing to determine whether they are infected. If persons are not infected, counseling and testing can relieve unnecessary anxiety and reinforce the need to adopt or continue practices that reduce the risk of infection. If persons are infected, they should: a) take precautions to protect sexual partners from becoming infected; b) advise previous and current sexual or drug-use partners to receive counseling and testing; c) take precautions against becoming pregnant; and d) seek medical care and counseling about other medical problems that may result from a weakened immunologic system.

More detailed information about AIDS, including information about how to obtain counseling and testing for HIV, can be obtained by telephoning the AIDS National Hotline (toll free) at 800-342-2437; the Sexually Transmitted Diseases National Hotline (toll free) at 800-227-8922; or the appropriate state or local health department (the telephone number of which can be obtained by calling the local information operator).

CURRICULUM TIME AND RESOURCES

Schools should allocate sufficient personnel time and resources to assure that policies and programs are developed and implemented with appropriate community involvement, curricula are well-planned and sequential, teachers are well-trained, and up-to-date teaching methods and materials about AIDS are available. In addition, it is crucial that sufficient classroom time be provided at *each* grade level to assure that students acquire essential knowledge appropriate for that grade level, and have time to ask questions and discuss issues raised by the information presented.

PROGRAM ASSESSMENT

The criteria recommended in the foregoing "Guidelines for Effective School Health Education to Prevent the Spread of AIDS" are summarized in the following nine assessment criteria. Local school boards and administrators can assess the extent to which their programs are consistent with these guidelines by determining the extent to which their programs meet each point shown below. Personnel in state departments of education and health also can use these criteria to monitor the extent to which schools in the state are providing effective health education about AIDS.

1. To what extent are parents, teachers, students, and appropriate community representatives involved in developing, implementing, and assessing AIDS education policies and programs?

2. To what extent is the program included as an important part of a more comprehensive school health education program?
3. To what extent is the program taught by regular classroom teachers in elementary grades and by qualified health education teachers or other similarly trained personnel in secondary grades?
4. To what extent is the program designed to help students acquire essential knowledge to prevent HIV infection at each appropriate grade?
5. To what extent does the program describe the benefits of abstinence for young people and mutually monogamous relationships within the context of marriage for adults?
6. To what extent is the program designed to help teenage students avoid specific types of behavior that increase the risk of becoming infected with HIV?
7. To what extent is adequate training about AIDS provided for school administrators, teachers, nurses, and counselors—especially those who teach about AIDS?
8. To what extent are sufficient program development time, classroom time, and educational materials provided for education about AIDS?
9. To what extent are the processes and outcomes of AIDS education being monitored and periodically assessed?

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APPENDIX I

The President's Domestic Policy Council's Principles for AIDS Education

The following principles were proposed by the Domestic Policy Council and approved by the President in 1987:

> Despite intensive research efforts, prevention is the only effective AIDS control strategy at present. Thus, there should be an aggressive Federal effort in AIDS education.
>
> The scope and content of the school portion of this AIDS education effort should be locally determined and should be consistent with parental values.
>
> The Federal role should focus on developing and conveying accurate health information on AIDS to the educators and others, not mandating a specific school curriculum on this subject, and trusting the American people to use this information in a manner appropriate to their community's needs.
>
> Any health information developed by the Federal Government that will be used for education should encourage responsible sexual behavior—based on fidelity, commitment, and maturity, placing sexuality within the context of marriage.
>
> Any health information provided by the Federal Government that might be used in schools should teach that children should not engage in sex and should be used with the consent and involvement of parents.

APPENDIX II

The Extent of AIDS and Indicators of Adolescent Risk

Since the first cases of acquired immunodeficiency syndrome (AIDS) were reported in the United States in 1981, the human immunodeficiency virus (HIV) that causes AIDS and other HIV-related diseases has precipitated an epidemic unprecedented in modern history. Although in 1985, fewer than 60% of AIDS cases in the United States were reported among persons residing outside New York City and San Francisco, by 1991 more than 80% of the cases will be reported from other localities.[1]

It has been estimated that from 1 to 1.5 million persons in the United States are infected with HIV,[1] and, because there is no cure, infected persons are potentially capable of infecting others indefinitely. It has been predicted that 20%–30% of individuals currently infected will develop AIDS by the end of 1991.[1] Fifty percent of those diagnosed as having AIDS have not survived for more than about 1.5 years beyond diagnosis, and only about 12% have survived for more than 3 years.[2]

By the end of 1987, about 50,000 persons in the United States had been diagnosed as having AIDS, and about 28,000 had died from the disease.[2] Blacks and Hispanics, who make up about 12% and 6% of the US population, respectively, disproportionately have contracted 25% and 14% of all reported AIDS cases.[3] It has been estimated that during 1991, 74,000 cases of AIDS will be diagnosed, and 54,000 persons will die from the disease. By the end of that year, the total number of deaths caused by AIDS will be about 179,000.[1] In addition, health care and supportive services for the 145,000 persons projected to be living with AIDS in that year will cost our Nation an estimated $8–$10 billion in 1991 alone.[1] The World Health Organization projects that by 1991, 50–100 million persons may be infected worldwide.[4] The magnitude and seriousness of this epidemic requires a systematic and concerted response from almost every institution in our society.

A vaccine to prevent transmission of the virus is not expected to be developed before the next decade, and its use would not affect the number of persons already infected by that time. A safe and effective antiviral agent to treat those infected is not expected to be available for general use within the next several years. The Centers for Disease Control,[5] the National Academy of Sciences,[6] the Surgeon General of the United States,[7] and the US Department of Education[8] have noted that in the absence of a vaccine or therapy, educating individuals about actions they can take to protect themselves from becoming infected is the most effective means available for controlling the epidemic. Because the virus is transmitted almost exclusively as a result of behavior individuals can modify (eg, by having sexual contact with an infected person or by sharing intravenous drug paraphernalia with an infected person), educational programs designed to influence relevant types of behavior can be effective in controlling the epidemic.

A significant number of teenagers engage in behavior that increases their risk of becoming infected with HIV. The percentage of metropolitan teenage girls who had ever had sexual intercourse increased from 30%–45% between 1971 and 1982. The average age at first intercourse for females remained at approximately 16.2 years between 1971 and 1979.[9] The average proportion of never-married teenagers who have ever had intercourse increases with age from 14 through 19 years. In 1982, the percentage of never-married girls who reported having engaged in sexual intercourse was as follows: approximately 6% among 14-year-olds,[10] 18% among 15-year-olds, 29% among 16-year-olds, 40% among 17-year-olds, 54% among 18-year-olds, and 66% among 19-year-olds.[11] Among never-married boys living in metropolitan areas, the percentage who reported having engaged in sexual intercourse was as follows: 24% among 14-year-olds, 35% among 15-year-olds, 45% among 16-year-olds, 56% among 17-year-olds, 66% among 18-year-olds, and 78% among 19-year-olds.[9,12] Rates of sexual experience (eg, percentage having had intercourse) are higher for black teenagers than for white teenagers at every age and for both sexes.[11,12]

Male homosexual intercourse is an important risk factor for HIV infection. In one survey conducted in 1973, 5% of 13- to 15-year-old boys and 17% of 16- to 19-year-old boys reported having had at least one homosexual experience. Of those who reported having had such an experience, most (56%) indicated that the first homosexual experience had occurred when they were 11 or 12 years old. Two percent reported that they currently engaged in homosexual activity.[13]

Another indicator of high-risk behavior among teenagers is the number of cases of sexually transmitted diseases they contract. Approximately 2.5 million teenagers are affected with a sexually transmitted disease each year.[14]

Some teenagers also are at risk of becoming infected with HIV through illicit intravenous drug use. Findings from a national survey conducted in 1986 of nearly 130 high schools indicated that although overall illicit drug use seems to be declining slowly among high school seniors, about 1% of seniors reported having used heroin and 13% reported having used cocaine within the previous year.[15] The number of seniors who injected each of these drugs is not known.

Only 1% of all the persons diagnosed as having AIDS have been under age 20;[2] most persons in this group had been infected by transfusion or perinatal transmission. However, about 21% of all the persons diagnosed as having AIDS have been 20–29 years of age. Given the long incubation period between HIV infection and symptoms that lead to AIDS diagnosis (3 to 5 years or more), some fraction of those in the 20- to 29-year-age group diagnosed as having AIDS were probably infected while they were still teenagers.

Among military recruits screened in the period October 1985–December 1986, the HIV seroprevalence rate for persons 17–20 years of age (0.6/1,000) was about half the rate for recruits in all age groups (1.5/1,000).[16] These data have led some to conclude that teenagers and young adults have an appreciable risk of infection and that the risk may be relatively constant and cumulative.[17]

Reducing the risk of HIV infection among teenagers is important not only for their well-being but also for the children they might produce. The birth rate for U.S. teenagers is among the highest in the developed world;[18] in 1984, this group accounted for more than 1 million pregnancies. During that year the rate of pregnancy among sexually active teenage girls 15–19 years of age was 233/1,000 girls.[19]

Although teenagers are at risk of becoming infected with and transmitting the AIDS virus as they become sexually active, studies have shown that they do not believe they are likely to become infected.[20,21] Indeed, a random sample of 860 teenagers (ages 16–19) in Massachusetts revealed that, although 70% reported they were sexually active (having sexual intercourse or other sexual contact), only 15% of this group reported changing their sexual behavior because of concern about contracting AIDS. Only 20% of those who changed their behavior selected effective methods such as abstinence or use of condoms.[20] Most teenagers indicated that they want more information about AIDS.[20,21]

Most adult Americans recognize the early age at which youth need to be advised about how to protect themselves from becoming infected with HIV and recognize that the schools can play an important role in providing such education. When asked in a November 1986 nationwide poll whether children should be taught about AIDS in school, 83% of Americans agreed, 10% disagreed, and 7% were not sure.[22] According to information gathered by the United States Conference of Mayors in December of 1986, 40 of the Nation's 73 largest school districts were providing education about AIDS, and 24 more were planning such education.[23] Of the districts that offered AIDS education, 63% provided it in 7th grade, 60% provided it in 9th grade, and 90% provided it in 10th grade. Ninety-eight percent provided medical facts about AIDS, 78% mentioned abstinence as a means of avoiding infection, and 70% addressed the issues of avoiding high-risk sexual activities, selecting sexual partners, and using condoms. Data collected by the National Association of State Boards of Education in the summer of 1987 indicated that a) 15 states had mandated comprehensive school health education; eight had mandated AIDS education; b) 12 had legislation pending on AIDS education, and six had state board of education actions pending; c) 17 had developed curricula for AIDS education, and seven more were developing such materials; and d) 40 had developed policies on admitting students with AIDS to school.[24]

The Nation's system of public and private schools has a strategic role to play in assuring that young people understand the nature of the epidemic they face and the specific actions they can take to protect themselves from becoming infected—especially during their adolescence and young adulthood. In 1984, 98% of 14- and 15-year-olds, 92% of 16- and 17-year-olds, and 50% of 18- and 19-year-olds were in school.[25] In that same year, about 615,000 14- to 17-year-olds and 1.1 million 18- to 19-year-olds were not enrolled in school and had not completed high school.[26]

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HIV Counseling Program

Since July 1985, the AIDS Institute has conducted a counseling and antibody testing program to provide free and anonymous services for individuals concerned about their human immunodeficiency virus (HIV) antibody status. These alternate site services are currently available in 19 upstate New York counties and in New York City.

Between July 1985 and January 29, 1988, the program received 137,410 hotline calls and made 43,866 pretest appointments during which HIV information was shared. To date, approximately 31,000 antibody tests have been conducted through the alternate site counseling and testing program.

Health care providers may advise patients who want information and referral regarding anonymous HIV antibody counseling and testing to call the hotline number in their public health region and ask for the HIV counselor.

Rochester Public Health Region	716-423-8081
Syracuse Public Health Region	315-428-4736
Buffalo Public Health Region	716-847-4520
Nassau County	516-535-2004
Suffolk County	516-348-2999
New Rochelle Public Health Region	914-632-4133
Albany Public Health Region	518-457-7152
New York City:	
Bronx	212-716-3350
Brooklyn	718-797-9110
Queens	718-262-9100
Manhattan (Harlem)	212-694-0884

People may also call the following hotlines provided by the New York State Department of Health:

1-800-462-1884	Monday-Friday, 8 a.m.-8 p.m.
(AIDS tapes and information)	Saturday-Sunday, 10 a.m.-6 p.m.
1-800-872-2777	Monday-Friday, 4 p.m.-8 p.m.
(HIV counseling and referral)	Saturday-Sunday, 10 a.m.-6 p.m.

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Lymphocytic interstitial pneumonitis in adult HIV infection

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Pulmonary disease associated with acquired immunodeficiency syndrome (AIDS) is common and may be secondary to infectious and noninfectious processes.[1] One of the noninfectious processes is lymphocytic interstitial pneumonitis (LIP). LIP is an uncommon disorder that was first described by Carrington and Liebow[2] in 1966, and is characterized by a diffuse peribronchial and interstitial infiltration of the lung by lymphocytes and plasma cells.[3] LIP has been recognized as a common pulmonary complication in cases of pediatric AIDS and appears to be present in as many as 75% of these cases.[4] The present Centers for Disease Control (CDC) classification includes LIP as a diagnostic criterion for pediatric AIDS.[5]

The occurrence of LIP in adults with human immunodeficiency virus (HIV) infection has recently been noted.[6-10] In this report we describe two additional cases of LIP in adults with HIV infection. Also presented is an analysis from the literature of 14 cases of adults with LIP and HIV infection.

CASE REPORTS

Case 1. A 37-year-old man with a history of bisexuality and intravenous drug abuse was admitted to Metropolitan Hospital on March 11, 1987, with a two-month history of cough, shortness of breath, pleuritic chest pain, fever, and a 12-kg weight loss. His medical history was otherwise unremarkable.

On admission, the patient was afebrile. There was 1-2 cm diameter nontender lymphadenopathy in the cervical, axillary, and inguinal regions. Auscultation of the lungs revealed inspiratory rales and egophony in the right posterior and axillary lung fields. Abdominal, skin, and joint examinations were within normal limits. The chest radiograph (Fig 1) showed right middle and lower lobe infiltrates with a prominent right hilum as well as a focus of previously noted left lung calcification. A panel of delayed type skin tests did not elicit any reaction.

A hemogram revealed a white blood cell count of 6,700/mm^3; platelet count, 333,000/mm^3; and hemoglobin, 10.9 g/dL. The differential white blood cell count showed 75% lymphocytes. Arterial blood gas levels were pH, 7.4; P_{CO_2}, 35 mm Hg; and P_{O_2}, 95 mm Hg. The IgG count was 4,000 mg/dL (normal, up to 1,700 mg/dL), the IgM was 68 mg/dL (normal, 30-360 mg/dL), and the IgA was 125 mg/dL (normal, 50-410 mg/dL). No monoclonal peak was detected on serum protein electrophoresis. A bone marrow aspirate was within normal limits. Antinuclear antibody, C3, C4, and rheumatoid factor levels were within normal limits. The Epstein-Barr viral capsid antigen (EB-VCA) IgG antibody titer was 1:320, while the cytomegalovirus IgG titer was 1:16. Peripheral blood studies showed a total helper T cell concentration of 1,240/mm^3 with a T4/T8 ratio of 1:9 (both within normal reported limits). Pulmonary function tests showed a forced vital capacity (FVC) of 2.74 L (70% predicted), forced expiratory volume at one second (FEV$_1$) of 3.68 L (74% predicted), total lung capacity (TLC) of 4.58 L (74% predicted), and diffusion capacity for carbon monoxide (DLCO) of 26.3% (98% predicted). HIV antibody was demonstrated by ELISA and Western blot techniques.

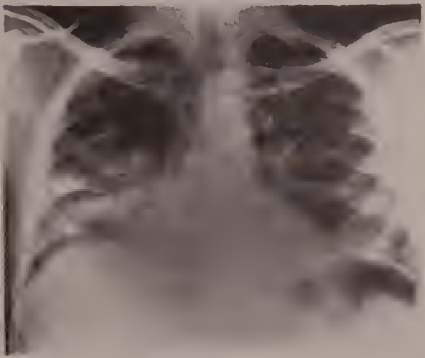

FIGURE 1. Posteroanterior chest film showing right middle and lower lobe infiltrates in case 1.

A transbronchoscopic biopsy of the right lower lobe showed infiltration of peribronchial areas and alveolar septae by mature small lymphocytes and scattered plasma cells with some areas of lymphocytic aggregates. Immunoperoxidase staining for light chains showed the presence of both kappa and lambda chains. This was interpreted as consistent with LIP. No *Pneumocystis carinii* or acid-fast organisms were found. A gallium scan showed bilateral lung uptake with more prominent uptake in the right lung (especially the right hilum). A computed tomogram of the chest showed right lung parenchymal consolidation with air bronchograms, as well as right hilar and mediastinal adenopathy (Fig 2). A mediastinoscopic biopsy of this lymph node showed a pattern of reactive hyperplasia with plasmacytosis and tangible macrophages within reactive follicular centers.

The patient was discharged from the hospital on prednisone, 60 mg/day. He was asymptomatic on 15 mg/day of prednisone at three months after discharge, and his chest film showed complete clearing of all infiltrates at that time. Steroid medication was completely tapered two weeks later, and the patient has remained asymptomatic six months after discharge. A repeat T lymphocyte analysis showed 412 T4 cells/mm^3 and 900 T8 cells/mm^3.

Case 2. A 31-year-old man with a history

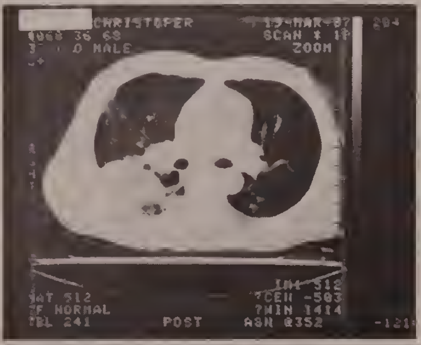

FIGURE 2. Computed tomograph of the chest demonstrating air bronchograms in the area of consolidation of the right lung in case 1.

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of intravenous drug abuse (cocaine) was admitted to Metropolitan Hospital in March 1986 with complaints of fever, productive cough, hemoptysis, and a 14-pound weight loss over the past six months. His wife had recently died from AIDS.

On admission, he was febrile with a temperature of 38°C, he had cervical, supraclavicular, axillary, and inguinal adenopathy (1-2 cm), and he had rales in the posterior right lung fields. A chest radiograph revealed right middle and lower lobe alveolar infiltrates. The white blood cell count was 7,100/mm^3 with 24% neutrophils, 73% lymphocytes, and 3% eosinophils. The hematocrit was 35.8%. The serum asparate aminotransferase level was 90 U/L; albumin, 3.3 g/dL; and globulins, 7.0 g/dL. Arterial blood gas results on room air were pH, 7.43; P_{CO_2}, 32 mm Hg; and P_{O_2}, 87 mm Hg. Intermediate strength purified protein derivative was nonreactive, and sputum cultures for bacteria and mycobacteria were negative. HIV antibody testing was positive.

Right lower lobe transbronchial biopsy revealed diffuse infiltration of alveolar septae and peribronchial connective tissue with lymphocytic cells. No lymphoid follicles were identified. Light chain stains were polyclonal. No *Pneumocystis carinii* were identified in the biopsy or washings. Pulmonary function tests showed a FEV_1 of 3.37 L (82% predicted); FVC of 4.12 L (79% predicted), TLC of 5.71 L (90% predicted), and DLCO of 15.6% (60% predicted). A gallium scan revealed bilateral increased lung uptake.

The patient was treated with prednisone, 60 mg/day, with resolution of fever and cough and increase in body weight. The right lower lobe infiltrate resolved. He was admitted to the hospital in July 1986 for treatment of salmonella sepsis and in November 1986 for treatment of staphylococcal pneumonia.

On January 29, 1987, the patient was readmitted for headaches, changes in mental status, and fever. On admission, he was afebrile and found to have nuchal rigidity, cervical adenopathy, and a 2/6 systolic ejection murmur. The white blood cell count was 5,100 mm^3, and the platelet count was 240,000/mm^3. The cerebrospinal fluid was positive for cryptococcal antigen at a titer of 1:1,024. The patient was treated with amphotericin and cefuroxime. On January 30, 1987, respiratory failure developed, and an endotracheal tube was inserted. The patient subsequently died on February 7, 1987. Post-mortem examination was not performed.

DISCUSSION

Sixteen cases associated with adult HIV infection,[6-10] including the two cases reported here, were reviewed. Three patients from one series[6] appear to have been reported previously in another report.[11] The characteristics of the reported adult LIP cases associated with HIV infection are shown in Table I.

LIP is thought to be part of a spectrum of benign lymphoid disorders of the lungs which diffusely involves alveolar septae.[12] Other lymphoproliferative disorders that can involve the lung (and have been described in association with AIDS) include non-Hodgkin lymphoma and angioblastic lymphadenopathy.[13,14] Benign lymphoid disorders of the lung such as LIP differ from lymphomatous involvement of the lung in that there is no evidence of light chain monoclonality, and the mononuclear infiltrates are always polymorphic in the former, whereas they may be monomorphic in lymphoma.[12,15] Both angioblastic lymphadenopathy and pulmonary lymphoma can have interstitial lymphocyte infiltration, and definitive differentiation between LIP and these two other disorders may on occasion require histologic evaluation of lymph nodes or more lung parenchymal tissue.

LIP is regarded as one of several chronic interstitial pulmonary patterns according to a schema of Liebow and Carrington.[3] In LIP, there is alveolar septal and peribronchial lymphocyte infiltration, as well as lymphoid aggregates that may contain micronodules often positioned around a central, thick-walled, periodic acid-Schiff-positive venule. Occasionally, germinal centers may be seen within the nodules, and epithelioid transformation of macrophages may be present within the germinal centers. Pulmonary fibrosis and noncaseating granulomas may be seen.[12] Similar histopathologic findings of lymphoid nodules, giant cells, and granuloma-like collections of mononuclear cells have been reported in a series of pediatric AIDS-associated LIP cases.[16] In the adult LIP cases reviewed

TABLE I. Patient Profiles of Adult HIV-Associated LIP

Patient No. (Ref. No.)	Chest Radiograph	H/S ratio (T4)	Age-yr (Race/ Nationality)	Infection	Lymphoid Involvement
1(6)	Diffuse reticulonodular	NS[†] (NS)	32 (African)	Five bacterial	RP,HA Sp,Hp
2(6)	Diffuse reticulonodular	0.23 (155)	53 (African)	Two bacterial	RP,PA
3(6)	Diffuse reticulonodular	0.27 (480)	28 (Haitian)		RP,PA
4(6)	Diffuse reticulonodular	0.28 (410)	40 (African)	One bacterial	RP,PA
5(6)	Diffuse reticulonodular	0.25 (310)	24 (Haitian)		RP,PA
6(6)	Diffuse reticulonodular	0.95 (450)	30 (Haitian)	Two bacterial	RP,HA PA
7(6)	Diffuse reticulonodular	0.60 (1,065)	32 (Haitian)		RP,PA
8(7)	Bilateral reticulonodular	0.2 (NS)	32 (Black)	*Mycobacterium avium/intracellulare*	PA,Hp Sp
9(7)	Bilateral reticulonodular	0.6 (NS)	56 (Black)	Presumed *mycobacterium tuberculosis*	Sp
10(7)	Bilateral reticulonodular	0.26 (NS)	46 (Black)	*Pneumocystis carinii*	
11(8)	Bilateral reticulonodular	0.7 (318)	43 (Not stated)	Esophageal candidiasis	Hp, Sp
12(present case)	Right middle and lower lobe infiltrates	NS	31 (Black)	Cryptococcus, Two bacterial	PA
13(present case)	Right middle and lower lobe infiltrates	1.9 (1,240)	37 (Black)	—	HA, PA
14(9)	Bilateral reticulonodular	0.45 (717)	30 (Black)	—	PA
15-16(10)	NS	NS	NS (Haitian)	NS	NS

[*] H/S ratio, T-helper to suppressor lymphocyte ratio; T4(CD4) lymphocytes per cubic millimeter; PA, peripheral adenopathy; HA, hilar adenopathy; Sp, splenomegaly; Hp, hepatomegaly; RP, retroperitoneal.

[†] NS, not stated.

herein, germinal centers, lymphoid nodule presence, and fibrosis in the pulmonary interstitium were reported, but there were no reports of giant cells, follicular or large cell hyperplastic changes, or granulomas. The evolution of adenopathic follicular hyperplasia into follicular involution/atrophy seen in HIV-associated peripheral lymph nodes[17] has not been reported in HIV-associated LIP. Recently, nonspecific pneumonitis has been described[1,18] as another noninfectious pulmonary manifestation of HIV infection which can be histologically distinguished from LIP because of a more mixed cellular infiltrative pattern with less well formed lymphoid aggregates, such as follicles and germinal centers. Two Haitian patients with miliary chest film patterns and diffuse lymphoid nodules on histologic analysis have been described in association with diminished CD4/8 (T4/8) peripheral blood lymphocyte ratios.[12] This may represent another pattern of pulmonary lymphoid involvement in HIV infection.

The clinical features of the patients reviewed in this report showed several interesting findings. The spirometric findings showed restrictive patterns in nine of ten cases studied (total lung capacity range, 44–90% predicted; DLCO range, 30–98% predicted) and obstructive patterns in one case.[6] On radiographic studies reported prior to the discovery of AIDS, LIP had been noted to have both interstitial and alveolar patterns.[12] The two patients described here showed lateralizing and discrete infiltrates, with documented air bronchograms in one. This confirms that both patterns (Table I) can occur in HIV-associated cases as well. In the 12 cases in which race was stated, all patients were black. None of the cases in the French series[6,9] was associated with intravenous drug abuse or homosexuality. Whether there is a genetic predisposition to LIP in adults is not clear. Four of the 16 patients had died at the time of the report (one each from failure to thrive, salmonella infection, cryptococcal infection, and *Pneumocystis carinii* pneumonia). Bacterial infections were sometimes recurrent and included salmonella, *Haemophilus influenzae*, staphylococcus, and streptococcus. Clinical AIDS[5] was described in three subjects at the time of diagnosing LIP; opportunistic infections consistent with AIDS were diagnosed after LIP in four patients, three of whom were treated with corticosteroids.

Treatment of LIP in the pre-AIDS era usually involved high-dose corticosteroid administration.[19] In the cases reviewed here, treatment and LIP response were not stated in all instances. Five patients were treated with corticosteroids, and their respiratory parameters and/or symptoms improved. In one series, patients' conditions were reported to be stable without steroid therapy. One case described by Grieco et al[8] had spontaneous improvement of respiratory symptoms and chest film appearance without steroid administration. In pediatric AIDS-associated LIP, both corticosteroids and gammaglobulin administration have been used with some success in improving respiratory parameters.[20] In the pre-LIP era, in LIP cases associated with hypogammaglobulinemia, there was reported improvement of LIP with gammaglobulin replacement therapy.[19]

The immunopathogenesis of LIP is not known, but pre-AIDS cases have often been associated with a variety of disorders with autoimmune components such as Sjögren syndrome, dysgammaglobulinemia, chronic active hepatitis, pernicious anemia, myasthenia gravis, and hypogammaglobulinemia. Polyclonal B cell stimulation and lymphoid tissue hyperplasia also seem to be common in LIP. In pre-AIDS literature on LIP, hypergammaglobulinemia was found in more than 90% of non-hypogammaglobulinemic patients in one series.[19] In our review, 92% (12/13) of patients with HIV-associated LIP had hypergammaglobulinemia. In this institution, hypergammaglobulinemia is seen in about 50–70% of the cases, while hypergammaglobulinemia in persistent generalized adenopathy secondary to HIV infection has been reported to occur in 31–88% of patients.[21] B cell tropic viruses are known to infect AIDS patients possibly secondary to an impaired T lymphocyte regulatory mechanism, and may be responsible in part for stimulating polyclonal immunoglobulin production[22] in these patients. Indeed, viral genomes such as Epstein-Barr virus DNA have been identified in some pediatric AIDS-associated LIP tissues.[23] Nine cases reviewed here were studied for EBV-VCA IgG, and five had titers of greater than 1:320.[6,23] Extrapulmonary lymphoid hyperplasia has been reported in pre-AIDS LIP cases.[19] This appears also to be present in HIV-associated adult LIP, with adenopathy/splenomegaly present in 13 of 14 cases reported. Whether LIP represents one of several sites of a generalized lymphoproliferative response in HIV infection or whether there is a specific pulmonary immunogenic agent that provokes a localized response is a matter of speculation at this time.

Immunophenotypic studies of tissue sections in LIP have not shown any consistent patterns in pre-AIDS or HIV-associated cases. The latter have shown patterns ranging from mixed T and B cells,[24] to predominantly T cells with both T8 (CD8) and T4 (CD4) populations,[7] to predominantly T8 cells.[7,9] Pre-AIDS LIP cases have been reported with both T cell and B cell predominance.[8,9] Bronchoalveolar lavage findings in two HIV-associated cases[6,9] demonstrated decreased T4/T8 lavage lymphocyte ratios. Decreased ratios were identified in corresponding peripheral blood studies.

From this review of adult HIV-associated LIP, we submit an approach to managing patients in whom LIP is a diagnostic consideration—ie, those presenting with subacute or chronic respiratory symptoms and either lateralizing or bilateral infiltrates on chest radiographs. Since LIP is a diffuse interstitial process, transbronchial biopsy appears to be a reasonable first line method of obtaining adequate histopathologic and microbiologic specimens. Concurrent washings and specimens for mycobacterial cultures/stains, *Pneumocystis carinii* stains, and cytologic studies should be obtained. The biopsy specimens should be examined by periodic acid-Schiff staining as well as light chain immunoperoxidase stains to exclude malignant disorders. If uncertainty regarding malignancy still exists, a computed tomogram may detect mediastinoscopically accessible lymph nodes for further histopathologic examination, thus potentially obviating the need for open lung biopsy. Management considerations in these cases must take into account the observation that some patients will have spontaneous resolution and/or improvement of their pulmonary symptomatology. The administration of corticosteroids in an already immunocompromised patient should be individualized on the basis of concurrent disease, symptoms, and degree of respiratory impairment; and the length and dose of steroid therapy should be reviewed frequently and minimalized if possible. Although nonspirometric gauges of interstitial lung disease may be employed to follow these patients, such as quantitative bronchoalveolar lavage and gallium scanning,[25,26] concurrent infectious processes may complicate the interpretation of these tests. Clearly, it is of paramount importance to recognize and treat the infectious complications of AIDS in these patients. Also, the possibility of malignant transformation of de novo pulmonary lymphoma development should be considered.[12,15,27]

Because of the relatively few cases of adult HIV-associated LIP, many questions still exist regarding the prognosis, histopathology, and treatment of these patients. However, it seems clear that LIP constitutes part of the spectrum of pulmonary disorders that affect HIV-infected adults.

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Thymoma, *Pneumocystis carinii* pneumonia, and AIDS

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Since the initial descriptions of acquired immunodeficiency syndrome (AIDS), it has become evident that the disorder predisposes patients not only to opportunistic infections, but also to malignancies. Large series have reported a 33-40% incidence of neoplastic disease in patients with AIDS;[1,2] the overwhelming majority of these malignancies are Kaposi sarcoma.[3] An additional 4-10% of patients have non-Hodgkin lymphomas, usually of the aggressive B cell type. Case reports of related solid tumors have recently appeared, and although cause and effect have not been proven, the incidence of solid tumors in patients with AIDS may be as high as 5%.[4,5]

We report what appears to be the first case of thymoma in a patient with AIDS. The case is particularly interesting as included in the differential diagnosis is common variable immunodeficiency (Good syndrome), an uncommon immunodeficiency state associated with 5-10% of thymomas.[6]

CASE REPORT

The patient, a 48-year-old man, was in excellent health until two weeks prior to admission, when fever, anorexia, and a cough productive of yellow sputum developed. His symptoms progressed, and when shortness of breath developed, the patient was referred to a physician. He denied a history of intravenous drug abuse, blood transfusions, or homosexual encounters, but admitted to frequent contacts with prostitutes. He had never been hospitalized before and was not taking any medications.

On physical examination, the patient was noted to be cyanotic, diaphoretic, and in respiratory distress. He was febrile to 104°F, had oral thrush, and, on inspiration, rales were detected in both lung fields. The examination did not reveal adenopathy, organomegaly, or skin lesions.

Routine hematologic tests were signifi-

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cant for an elevated white blood cell count with a shift to the left, a hemoglobin of 13.8 g/dL, and a normal platelet count. Blood chemistry studies were significant for a total protein of 6.0 g/dL, an albumin of 2.2 mg/dL, a lactate dehydrogenase level of 580 IU/L, and mildly elevated liver enzyme levels. Results of an arterial blood gas test on room air were pH, 7.49; Pco_2, 32 mm Hg; Po_2, 49 mm Hg. A chest film revealed bilateral diffuse interstitial infiltrates and a mediastinal mass approximately 6 cm in diameter.

The patient was admitted to the hospital and was started on trimethoprim/sulfamethoxazole for possible *Pneumocystis* pneumonia. A computed tomographic (CT) scan of the chest revealed a well-defined, homogeneous, noncalcified mass in the anterior mediastinum with no evidence of adenopathy or local invasion (Fig 1). Despite oxygen supplementation and the addition of antituberculous drugs to his regimen, the patient remained febrile, with worsening hypoxia that necessitated intubation.

On the third hospital day, a mediasternotomy was performed with biopsy of the mass and adjacent lung. On gross examination the lesion appeared smooth and tan-colored with an intact capsule. On microscopic analysis, it consisted of spindle-shaped epithelial cells with scattered aggregates of normal-appearing mature lymphocytes (Fig 2). Immunoperoxidase staining showed the spindle cells to be positive for cytokeratin, and well-formed desmosomes were noted between the cells on electron microscopy. The appearance and staining of the mediastinal mass were consistent with spindle cell thymoma, and the intact capsule indicated that the lesion was benign. Silver stains of lung tissue for *Pneumocystis* organisms were positive, while stains for acid-fast bacilli and fungi were negative.

Evaluation of the patient's immune status revealed cutaneous anergy and a strongly positive human immunodeficiency virus (HIV) titer on ELISA. Gammaglobulin levels were as follows: IgG, 1,090 mg/dL (normal, 750-1,750 mg/dL); IgA, 616 mg/dL (normal, 100-385 mg/dL); IgM, 87.4 mg/dL (normal, 50-250 mg/dL).

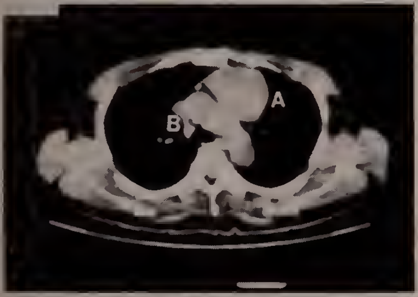

FIGURE 1. Computed tomographic scan of the chest demonstrating a large mediastinal mass (A) anterior to the great vessels (B).

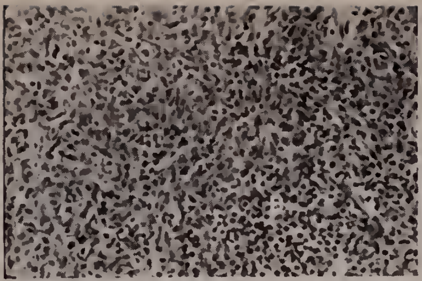

FIGURE 2. Biopsy specimen from the anterior mediastinal mass. Demonstrated are aggregates of spindle-like epithelial cells (A) and mature lymphocytes (B); hematoxylin-eosin stain, original magnification × 400.

Over the next several days, the patient's condition deteriorated, with worsening hypoxia and progressive hypotension. Despite aggressive resuscitation and the administration of pentamidine, the patient died on the seventh day of hospitalization.

DISCUSSION

The documentation of an associated opportunistic infection in a patient with a recognized risk factor and a positive HIV titer are diagnostic of acquired immunodeficiency syndrome. As such, this appears to be the first reported case of thymoma in a patient with AIDS. We do not necessarily propose a cause-and-effect relationship between AIDS and thymoma. As with other case reports involving solid tumors, there is no direct evidence that HIV infection induced or allowed the induction of the patient's tumor. AIDS has become so common that it will now be diagnosed in patients with concomitant but unrelated disorders. However, the predilection of HIV for infecting T lymphocytes and the role of thymus-derived T cells in the immune system may make the development of thymoma and other solid tumors more likely in patients with AIDS.

Although the patient presented had AIDS, included in the differential diagnosis is Good syndrome, a parathymic immunodeficiency state. Good syndrome is a rather uncommon disorder characterized immunologically by hypogammaglobulinemia (particularly of IgG and IgA), cutaneous anergy, and decreased populations of pre-B, B, and T-helper lymphocytes.[7,8] T-suppressor cell populations appear to be increased, leading to speculation that this is the primary immunologic defect.[8] Clinically, patients are on the average 50 years of age and of either sex.[9] They suffer from recurrent infections involving the respiratory tract, gastrointestinal tract, and skin,[7,8] and *Pneumocystis carinii* pneumonia has been described.[10] On pathologic evaluation, three quarters of thymomas in Good syndrome are of the spindle cell type, and most are benign.[8] The prognosis is poor, as most patients succumb to their infections within several years of diagnosis.[9] In contrast to other parathymic disorders, thymectomy in Good syndrome does not affect the course of the disease; in fact, immunodeficiency can develop many years after removal of a thymoma.[8,10]

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Saccharomyces fungemia in a patient with AIDS

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Saccharomyces cerevisiae is a yeast that is more commonly known as brewer's yeast. Commercial uses include beer and wine production, health food supplementation, and, more recently, hepatitis B vaccine production by recombinant DNA techniques. Occasionally, *S cerevisiae* has been isolated from clinical specimens and rarely has been a cause of serious infection. Case reports are few, and *S cerevisiae* has frequently been isolated in association with more common pathogenic organisms. We describe a case of *S cerevisiae* septicemia in association with acquired immunodeficiency syndrome (AIDS).

CASE REPORT

A 37-year-old woman with a history of intravenous drug and alcohol abuse sought medical attention in 1985 because of herpes zoster infection and oral candidiasis. The total T4 lymphocyte count was 324/mm^3 with a T4/T8 ratio of 0.73. Antibody against human immunodeficiency virus (HIV) was present. During the following 18 months her renal function deteriorated, with the serum creatinine level ranging from 4 to 6 mg/dL.

One week prior to admission, headache, generalized edema, ascites, pleural effusions, cough, shortness of breath, and fever developed. Examination revealed oral thrush, lymphadenopathy, decreased breath sounds on the right side, hepatomegaly, and anasarca. Serum creatinine and blood urea nitrogen levels were 18.8 mg/dL and 106 mg/dL, respectively. Chest roentgenography showed a right middle lobe infiltrate.

Treatment included antibiotics and peritoneal dialysis. During treatment a pericardial effusion developed and the patient's pleural effusions worsened. Pericardial biopsy and pleural fluid stains and cultures were negative.

The patient continued to have temperature elevations. She denied any ophthalmologic complaints and there were no overt signs of infection. Four weeks after admission three sets of blood cultures drawn during a 24-hour period grew *Saccharomyces cerevisiae*. Amphotericin B therapy was begun. Ophthalmologic examination revealed foci of fluffy yellow exudates in the right eye and chorioretinal necrosis of the left eye. Follow-up blood cultures were negative, and the fever subsided. The patient received a total dose of 2,100 mg of amphotericin B. Examination one month after completion of amphotericin B demonstrated disappearance of retinal exudates and chorioretinal scarring at the previous location of necrosis. The patient remained without evidence of saccharomyces infection. However, full-blown AIDS subsequently developed, and she died ten months later.

DISCUSSION

S cerevisiae is generally believed to be nonpathogenic.[1] In experimental studies, subcutaneous inoculation was neither lethal nor invasive for both control and cortisone-treated mice.[2] Although *S cerevisiae* was cultured from some visceral organs six days after challenge in both groups of mice, at 30 days there was no evidence of persistent infection.[2] Colonization and pathogenicity studies have established the safety of *S cerevisiae* as a candidate for a host vector system in recombinant DNA experiments. Colonization could not be established by oral or intravenous inoculation of control or cortisone-treated cynomolgus monkeys.[3]

Saccharomyces is believed to be an occasional, normal commensal in the gastrointestinal tract.[4] It has been isolated from sputum and tracheal aspirates of patients with tuberculosis.[5]

Kiehn et al[6] reviewed 3,340 yeast cultures from cancer patients over a 15-month period and isolated *S cerevisiae* from 19 sputum samples, including one tracheal aspirate and one lung tissue culture. Eng et al[1] described an immunosuppressed patient with dysphagia from whom both *S cerevisiae* and *Candida tropicalis* were isolated from the esophagus. In other patients, the authors[1] isolated *S cerevisiae* from pleural fluid, a kidney, urine, and from a Cowper gland. In many of the reported cases, another pathogenic organism was concomitantly isolated.[1] *S cerevisiae* has also been responsible for postoperative peritoneal infection.[7]

There have been three previous reports of *S cerevisiae* septicemia. The first case report in 1970 described a patient with prosthetic valve endocarditis.[8] The second case involved a 68-year-old man who ingested brewer's yeast in large quantities. The patient recovered without treatment when he stopped ingesting brewer's yeast.[9] The third case of *S cerevisiae* septicemia was in a burn patient on hyperalimentation.[10] In two of the aforementioned cases, fundoscopic examination was reported as normal.

The patient described in this report may have been predisposed to disseminated fungal infection secondary to underlying HIV infection and end-stage renal disease. Invasive fungal infections have previously been described in patients with HIV infection. Dissemination has occurred with *Candida albicans*, *Histoplasma capsulatum*,[11,12] *Coccidioides immitis*,[13] and *Cryptococcus neoformans*.[14,15] In addition, fungal septicemia has been reported in patients receiving peritoneal dialysis.[16] Contrary to the clinical experience with AIDS patients, in whom disseminated fungal infections are difficult to eradicate, this patient showed apparent cure after completing a regimen of 2,100 mg of amphotericin B.

The source of *S cerevisiae* fungemia in this patient is not clear. Dialysate fluid, pleural fluid, pericardial fluid, and sputum failed to grow the organism. Urine and stool were not sent for culture. The lack of a recognizable source is similar to that in cases of salmonellosis[17] or cryptococcosis[15] in patients with AIDS.

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We conclude that although *Saccharomyces cerevisiae* is a rare pathogen, it may cause serious invasive infection, especially in patients at risk. When isolated from the bloodstream, *S cerevisiae* cannot be dismissed as a nonpathogen and should now be added to the list of opportunistic pathogens that infect patients with AIDS.

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Candida pneumonia secondary to an acquired tracheoesophageal fistula in a patient with AIDS

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Fungal pneumonias represent approximately 5% of all pulmonary infections occurring in patients with acquired immunodeficiency syndrome (AIDS).[1] The most commonly observed cause of fungal pneumonia is *Cryptococcus neoformans*, followed by *Histoplasma capsulatum* or *Coccidioides immitis*, depending on the geographic location.[2] Despite the prevalence of oral thrush and esophageal candidiasis in patients with AIDS, pneumonia caused by *Candida albicans* is extremely uncommon. The following case report represents, to our knowledge, the first description of a candidal pneumonia secondary to aspiration through a tracheoesophageal fistula in a patient with AIDS.

CASE REPORT

A 36-year-old man with a history of intravenous drug abuse was admitted to Beth Israel Medical Center complaining of a three-week history of severe retrosternal chest pain, weight loss, shortness of breath, and a cough productive of white sputum. The diagnosis of AIDS had been made ten months prior to admission, when the patient was found to have esophageal candidiasis on upper endoscopy. He was initially treated with intravenous amphotericin B and then maintained on ketoconazole, which he took sporadically. Although significant weight loss had occurred, he denied any recent fevers, chills, or night sweats.

Physical examination on admission revealed a cachectic male in no apparent distress. The blood pressure was 100/70 mm Hg, the pulse was 80/min, the respiratory rate was 20/min, and he was afebrile. Examination of the head and neck was unremarkable, and no oral thrush was evident. Moist rales were heard at the base of the left lung posteriorly. The rest of the examination was unrevealing with the exception of a tender, erythematous rectal mucosa with a palpable fissure. A test for occult blood in the stool was negative.

Laboratory evaluation was significant for a white blood cell count of 1,100/mm^3, with a differential of 40% polymorphonuclear leukocytes, 10% band forms, 10% lymphocytes, and 40% monocytes. The hematocrit was 20.3%. Serum chemistry analysis revealed a protein content of 8.2 g/dL; albumin of 2.8 g/dL; alkaline phosphatase of 318 IU/L; and lactate dehydrogenase of 329 IU/L. A chest film revealed an elevated right hemidiaphragm, a left lower lobe retrocardiac infiltrate, and air along the left paratracheal border (Fig 1). There was no evidence of hilar or mediastinal adenopathy, and the heart size was normal. Tests of sputum were negative on Gram stain and culture, and no acid-fast bacilli were seen on smear.

The patient was initially treated with mycostatin oral suppositories and viscous lidocaine for his complaints of retrosternal pain, which was presumably due to the candidal esophagitis. On the sixth hospital day, his rectal temperature spiked to 104°. Physical examination revealed persistent rales at the base of the left lung. After blood and sputum cultures were drawn, he was started on intravenous clindamycin, 600 mg every six

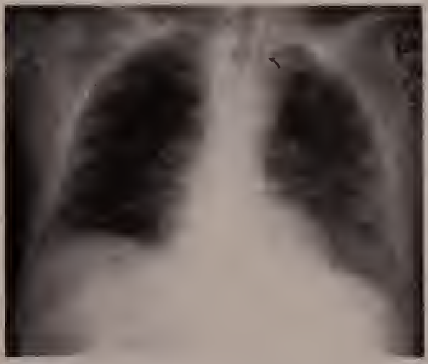

FIGURE 1. Anteroposterior chest film showing a left lower lobe infiltrate and air along the left paratracheal border (arrow).

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hours, and tobramycin, 80 mg every eight hours. His temperature continued to spike for the next 48 hours despite antibiotic therapy. Arterial blood gas studies revealed a pH of 7.46; P_{CO_2}, 27 mm Hg; Pa_{O_2}, 56 mm Hg, and a saturation of 91.2%. Repeat chest film showed worsening of the left lower lobe infiltrate. The antibiotics were changed to intravenous trimethoprim/sulfamethoxazole, 960 mg every six hours, and erythromycin, 750 mg every six hours. On the tenth hospital day, the patient underwent a flexible fiberoptic bronchoscopy. A small opening with irregular borders was noted on the left posterolateral aspect of the trachea, and methylene blue swallowed by the patient was observed to egress from this orifice. Candidal mucous plaques were noted along the left main stem and lower lobe bronchus. Transbronchial biopsies from the left lower lobe revealed pseudohyphae and fungal spores invading the pulmonary parenchyma. The specimens were consistent with *Candida* species. Special stains for acid-fast bacilli were negative. Subsequently the patient was started on antifungal therapy with intravenous amphotericin B, 20 mg/day intravenously. Despite excellent initial improvement, his overall clinical status continued to deteriorate, the dyspnea worsened, and he died on the 23rd hospital day.

DISCUSSION

The most common fungus isolated from respiratory secretions of patients with AIDS is *Candida albicans*, usually representing oropharyngeal colonization.[1] Even with the increased frequency of esophageal candidiasis in AIDS, candidal pneumonia has rarely been reported, except when secondary to hematogenous dissemination as a preterminal event. Marchevsky et al[3] reviewed the pathology of 70 patients with AIDS and pulmonary involvement. Specimens included transbronchial biopsies and autopsy tissue. Only three cases of invasive candidiasis were found, characterized by the presence of pseudohyphae and spores invading both the bronchial wall and lung parenchyma.

The case reported here is unique in two respects: it identifies a rare case of possible endogenous candidal pneumonia; and the portal of entry, presumably through a tracheoesophageal fistula secondary to longstanding candida esophagitis, has not been previously reported in a patient with AIDS. As with other immunocompromised hosts, the usual portal of entry is via hematogenous spread or aspiration. The first reported case of a tracheoesophageal fistula with an infectious etiology was described by Obrecht et al[5] in a patient with promyelocytic leukemia who was found to have invasive candidiasis and aspergillosis of the esophagus, with fungal organisms protruding from the fistula into the right mainstem bronchus. Sehhat et al[6] also reported a case of esophageal moniliasis, with the formation of an esophagopulmonary fistula and right lung abscess in a 28-year-old farmer who had no underlying chronic illness.

Although the patient described here initially improved with amphotericin B treatment, it is not certain that his terminal course was due to respiratory failure from a persistent candidal pneumonia with chronic aspiration through a tracheoesophageal fistula. Even in patients with diffuse pulmonary candidiasis secondary to hematogenous spread, the terminal event has usually been bacterial sepsis. However, given the frequency with which candidal esophagitis occurs in the population of AIDS patients, physicians should be cognizant of the potential for development of a tracheoesophageal fistula and fungal aspiration pneumonia.

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QUESTIONS AND ANSWERS ON AIDS

By Lyn Robert Frumkin, MD, and John Martin Leonard, MD. 208 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $19.95 (paperback)

This book is a clearly written monograph which achieves the author's intent to "enable health care workers to acquire general but comprehensive information about AIDS." The 133 pages of text are written in a question-and-answer format and address many of the issues one often encounters in discussions with health care professionals and others who have an interest in acquired immunodeficiency syndrome. In general, the monograph is simply and directly written.

Despite a rapidly expanding knowledge base, most information presented is current and accurate as of this writing. Occasionally some apparently factual information is imprecise or incorrect. While the authors acknowledge the lack of epidemiologic data regarding the efficacy of diaphragms and condoms in reducing AIDS virus transmission, they conclude "concurrent use of a spermicide with a diaphragm . . . may be expected to provide added protection against AIDS virus transmission." Since diaphragms may not protect the vaginal mucosa or be protective in other types of intercourse, this recommendation lacks scientific and practical support. Recent studies have also examined the ability of latex but not animal intestine condoms to create a barrier to human immunodeficiency virus penetration. This information, which may be quite important, is not included.

The risk of AIDS to hemophiliac patients is thoroughly discussed, as well as recommendations for reducing their risk through use of cryoprecipitate and desamino-D-arginine vasopressin (DDAVP). A minor point needing clarification is that DDAVP is a synthetic vasopressin and not a blood product, as the authors state.

With the exception noted above, this volume is technically correct and easily readable. A glossary of terms, a useful index, and a reference list help those readers who wish to address questions concerning AIDS in depth.

This book is a comprehensive but concise review that begins with a definition of AIDS, the cause of AIDS (human immunodeficiency virus), and a discussion of the much greater problem of fairly widespread but asymptomatic HIV infection. HIV antibody testing, epidemiology, health care worker issues, and a catalog of resources and research centers for dealing with AIDS as well as a brief review of ethical and social dilemmas that this disease raises complete the scope of the text. This publication is relatively inexpensive and may be a useful tool in a wide range of hospital-based and community educational programs, where an easily read, written resource is needed to complement other teaching aids.

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LIVING WITH AIDS AND HIV

By David Miller. 136 pp, illustrated. Dobbs Ferry, NY, Sheridan House Inc, 1987. $14.50 (paperback)

As the title of this book implies, this is a guide to dealing with the problems that accompany infection by the human immunodeficiency virus (HIV). It is not a textbook; rather, it is meant to be read by patients and paramedical personnel. In addition, it is aimed at those who will be dealing with the disease on a personal basis—individuals such as lovers, spouses, friends, and coworkers.

The author, David Miller, is a London-based psychologist with substantial experience in counseling AIDS patients and their loved ones. He played a major role in founding the first comprehensive workshops in Great Britain for medical and health staff working with patients with HIV infections.

The book is divided into seven chapters. The first two chapters deal primarily with the biology of the disease. The HIV antibody test is explained, as are the clinical manifestations of the disease. The third and fourth chapters deal with adapting to the news that one is seropositive, as well as practical issues such as what to do about the workplace and infection control in the home. The next two chapters discuss the psychological adjustments that are necessary as well as the inevitable issues that arise in personal relationships. The last chapter covers some religious issues and sums up with some brief case studies.

This book is an excellent resource for those who are dealing with this disease personally or who know someone who is ill with it. It is written clearly and intelligently, and biological concepts are explained without complicating medical jargon. Nonetheless, I would not recommend this book for someone with less than a high school education. The author clearly assumes that the reader has a broad social and educational background from which to draw. An additional liability is that the book has a British perspective. Even though all the concepts are applicable here, many of the resources referred to in this book are from Great Britain.

Nonetheless, physicians will find this a useful primer to recommend to their patients who wish to learn more about this disease. It has often been said that education is our only vaccine against HIV infection. This book is clearly one of the components of this vaccine.

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THE ESSENTIAL AIDS FACT BOOK. WHAT YOU NEED TO KNOW TO PROTECT YOURSELF, YOUR FAMILY, ALL YOUR LOVED ONES

By Paul Harding Douglas and Laura Pinsky. 63 pp. New York, Simon & Schuster Inc, 1987. $3.98 (paperback)

This book represents an ambitious attempt to synthesize existing information about AIDS and HIV infection into a concise and comprehensible format, directed toward a nontechnical audience. Given its size (63 pages), the authors do remarkably well in covering most of the major topics including patterns of illness, modes of transmission, treatment, risk reduction and HIV antibody testing. Although the medical aspects of HIV infection are not covered in depth, and very little is said about the consequences of HIV infection in the central nervous system, the amount of detail is probably adequate for the intended audience. Most of the information presented in this book is accurate and up to date, although a statement that "only three health workers are known to have become infected with the HIV virus," is no longer correct.

The primary importance of this book is in its clear and concise treatment of sexual risk reduction and HIV antibody testing. Concerning the former, readers are given information about the risks associated with specific sexual activities in direct and nonjudgmental language. Sexual activities are divided into three categories: high risk, lower risk, and no risk. The section on sexual transmission also stresses the fact that reducing one's number of sexual partners does not provide adequate protection from HIV infection if an individual continues to engage in "high-risk" activities. Although the authors do emphasize that "intuition" is unreliable as a means of assessing a potential partner's risk, they are somewhat noncommital in their statement that individuals with multiple partners "should probably avoid high-risk sexual activities." While it is difficult to generalize sexual risk reduction guidelines for the population at large, it is probably more appropriate to advise that sexual risk reduction be employed in all situations where one or both partners are unaware of their serostatus. The section on condoms is adequate, but does not address any of the potential barriers to condom use, especially the nearly ubiquitous concern that they interfere with sexual pleasure.

HIV antibody testing is reviewed from the perspective of the client rather than the health care provider, and there is an extensive and well reasoned discussion of the pros and cons of being tested, along with the need for adequate pretest counseling. Special emphasis is placed on the psychological implications of positive HIV test results, and individuals are cautioned to consider the adverse consequences of learning that they are seropositive. Although the statement, "if you cannot be tested anonymously, do not be tested," is supported by concerns about confidentiality of results and potential psychosocial liabilities, some readers may find it excessively dogmatic. The only major omission in the discussion of HIV antibody testing is a failure to address the subject of false-positive ELISA results. Nonetheless, it is probable that this section of the book will be useful to many readers who are trying to understand the benefits and liabilities of testing.

The final section of the book contains a brief but fairly complete description of the medical symptoms often associated with HIV infection and an outline of the psychological problems associated with AIDS.

Although this book is not intended for everyone, it should prove to be quite helpful to young adults, particularly those who are sexually active. It provides a reliable and easy to read summary of most of the major issues related to HIV infection, as well as concise guidelines to prevent the sexual transmission of the virus.

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AIDS: PRINCIPLES, PRACTICES AND POLITICS

By Inge B. Corless, PhD, and Mary Pittman-Lindeman, DrPH. 252 pp. New York, Hemisphere Publishing Corp, 1988. $15.95 (hardcover)

The purpose of *AIDS: Principles, Practices and Politics* is presumably represented in its title—an overview of many of the issues involved in this complex epidemic. One can find no clear statement of purpose either in the foreword by Mayor Dianne Feinstein of San Francisco or in the introduction by the co-editors. As the jacket states, this volume encompasses all areas of concern: the AIDS virus, physiological aspects, treatment and epidemiology, psychosocial, sociocultural and ethical aspects, political aspects, the media and AIDS, and the implications for future developments. Therein lies the problem with this book. Examination of a subject as complex as this disease presents enormous problems to any editor or author who wishes to deal comprehensively with these problems.

The editors appear to be well qualified to bring together a volume on this disease. Inge B. Corless is on the faculty of the School of Nursing at the University of North Carolina, while Mary Pittman-Lindeman is Director of Planning for the San Francisco Department of Health. Their contributors are also well known in the field of AIDS and include Dr Paul Volberding from San Francisco General Hospital, Dr Dean Echenberg from the San Francisco Department of Public Health, and Sister Patrice Murphy from St Vincent's Hospital in New York City.

The various contributions appear to cover every known issue that those of us involved in AIDS deal with. There are several chapters on the clinical aspects of the disease, including infection in children, as well as a number of chapters dealing with the ethics of the disease, and chapters that chiefly appear to deal with a philosophical approach to concepts of plague.

We are left with a series of chapters that only begin to touch on some of the problems, without leaving the reader satisfied that many subjects have been fully dealt with. If the reader is truly interested in a comprehensive approach to many of the issues touched on here, other resources are available in the burgeoning literature on this disease. It is texts such as this that give credence to the warning: We cannot be all things to all people.

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AIDS: PUBLIC POLICY DIMENSIONS

Edited by John Griggs. 308 pp. New York, United Hospital Fund, 1987. $32.50 (paperback)

AIDS: Public Policy Dimensions is based on the proceedings of a national conference held early in 1986 to explore the numerous public policy issues related to the ongoing epidemic of HIV infection. After a comprehensive yet concise overview of AIDS and HIV infection, the book is divided into eight sections dealing with AIDS and health policy; the politics of AIDS; AIDS and the school system; AIDS and the blood supply; the impact of AIDS on both acute medical services and community care services; the financial implications of AIDS; and its long term policy implications.

Because this book represents the policy analyses of many individuals, it is somewhat more reiterative than would be a similar analysis by a single author. However, the diversity of perspectives presented by such a format more than compensates for this repetition. The brevity and clarity of individual presentations makes this book quite easy to read, and, in general, most of the authors keep rhetoric to a minimum. Readers should be advised that several of the contributors have rather strong feelings about various political implications of the AIDS epidemic, and their perceptions about the federal response to the AIDS crisis are not always flattering. Nonetheless, this book does offer the reader a nearly comprehensive analysis of the nonmedical issues related to AIDS.

Particularly impressive are the sections dealing with the response of acute medical care facilities and community organizations to the epidemic of HIV infection. These are presented in a case study format by representatives from areas of the nation already experiencing difficulties in keeping pace with the exhaustive medical and financial needs of persons with AIDS. Throughout this section of the book it becomes apparent that it is not just the scientific community which has been asked to respond in innovative ways to the challenge of AIDS. Community service organizations, health care administrators, medical economists, health care planners, and health insurance organizations have all been confronted with a tremendous challenge regarding AIDS. Many of the contributors in this section of the book underscore the absolute need for integration of medical services with existing community-based agencies that provide social support services—both as a means of insuring comprehensive care, and also as a mechanism for containing costs. The phenomenon of shifting health care costs to individuals with AIDS and to community-based agencies that are largely supported by volunteer effort is examined from a critical perspective, as is the potential impact that AIDS will have on traditional mechanisms of financing for health care costs in America.

Persons who are interested in developing an appreciation for the multitude of ways in which AIDS will influence our society at large as well as its specific institutions will certainly find *AIDS: Public Policy Dimensions* to be a thought provoking and satisfying book. The ethical, political, and economic questions raised by its authors are undoubtedly as complex as the multitude of scientific, medical, and therapeutic issues related to this epidemic.

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Promising results with drugs for chemoprophylaxis of *Pneumocystis carinii* pneumonia in AIDS

Pneumonia caused by *Pneumocystis carinii* infection develops in more than half of patients with AIDS and results in death in more than 20% of these patients. Previous studies have shown that the combination of sulfamethoxazole and trimethoprim is very effective in treating and preventing *P carinii* infection among immunosuppressed patients without AIDS. Authors Fischl, Dickinson, and La Voie from the University of Miami School of Medicine used these findings as the basis of their experimentation with these drugs in patients with AIDS. The authors recently reported the promising findings of their prospective study to determine the safety, efficacy, and patient tolerance of long term sulfamethoxazole and trimethoprim therapy to prevent *P carinii* pneumonia among patients with AIDS (*JAMA* 1988; 259:1185-1189).

The study incorporated 60 patients with newly diagnosed Kaposi sarcoma, confirmed by biopsy, who exhibited no other opportunistic infections and who had not received any prior antiretroviral or immunomodulator therapy, cytotoxic chemotherapy, or chemoprophylactic therapy for *P carinii* pneumonia. Thirty patients selected on a random basis received no suppressive therapy, serving as a control group, while 30 patients received sulfamethoxazole, 800 mg, and trimethoprim, 160 mg, both given orally twice a day. Patients in the drug treatment group also received oral leucovorin calcium, 5 mg/day—a folate compound not affected by the antifolate activity of sulfamethoxazole and trimethoprim, which can cause bone marrow suppression. Patient follow-up extended for a minimum of 24 months.

There were no reported cases of *P carinii* pneumonia among the patients receiving sulfamethoxazole and trimethoprim suppressive therapy, while *P carinii* pneumonia developed in 16/30 patients (53%) in the control group during the study period. Furthermore, *P carinii* pneumonia developed in four of five patients in whom sulfamethoxazole and trimethoprim therapy was discontinued because of toxic reactions to the medication.

The survival data also proved interesting. Eighteen of 30 patients (60%) in the treatment group died during the course of the study, while 28/30 patients (93%) in the control group died. The causes of death included other opportunistic infections, Kaposi sarcoma, and high-grade B-cell lymphoma, as well as *P carinii* pneumonia (eight patients) in the control population. The authors reported a statistically significant ($p < 0.002$) difference in the mean length (+ standard error) of survival for patients in the treatment group (22.9 ± 2.6 months) compared to the control group (12.6 ± 1.5 months).

Fifteen of 30 patients (50%) reported adverse reactions to the sulfamethoxazole and trimethoprim treatment; the most common reaction was mild to moderate erythroderma. Some patients also described an altered sense of taste, nausea with occasional vomiting, pruritis, and erythroderma associated with fever; leukopenia was also noted.

At the completion of the follow-up period, the authors concluded the following: "The findings of this study demonstrate that sulfamethoxazole and trimethoprim therapy is effective in preventing *P carinii* pneumonia in patients with AIDS-associated Kaposi's sarcoma. . . . Based on these findings, any patient with HIV infection at high risk for *P carinii* pneumonia should be considered for chemoprophylaxis." Their data suggest that sulfamethoxazole and trimethoprim therapy provides protection against *P carinii* pneumonia only during the course of treatment.

Despite the effectiveness of this drug combination, there were toxic reactions. Future research should address the efficacy and safety of lower doses and intermittent dosing. The authors also advocate studies of the long term safety and efficacy of chemoprophylaxis among asymptomatic patients with HIV infection and marked immunosuppression.

The effect of zidovudine on antigen levels in HIV infection

The drug zidovudine or AZT (3′-azido-3′-deoxythymidine) inhibits HIV replication in vitro, and can reduce HIV antigen levels in the serum of patients with AIDS or AIDS-related complex (ARC). "Persistence of human immunodeficiency virus antigen (HIV-Ag) in serum and cerebrospinal fluid (CSF) is strongly associated with rapid progression to the acquired immunodeficiency syndrome (AIDS)," observed de Wolf et al in a recent article in *The Lancet* (1988; 1:373-376). The authors sought to determine whether a simplified regimen of zidovudine therapy given early in HIV infection could lower serum HIV-Ag levels in symptom-free subjects, and thus help prevent progression to AIDS and the development of opportunistic infections and tumors.

Zidovudine is quite toxic and is not appropriate for use in all seropositive individuals. For patients with AIDS or ARC, the current dose schedule for zidovudine dictates administration of the drug every four hours, because of its short half-life. The authors note that subjects without symptoms may not be willing to comply with such a regimen.

A volunteer population of 18 homosexual men with persistent HIV antigen presence and no symptoms was randomly split into three groups of six. Group A received zidovudine alone at doses of 250 mg/6 hr or 500 mg/12 hr. Group B received zidovudine (250 mg/6 hr or 500 mg/12 hr) and acyclovir (800 mg/6 hr or 1,600 mg/12 hr). Group C received acyclovir alone (800 mg/6 hr) for a period of 8 weeks followed by zidovudine alone (500 mg/6 hr or 500 mg/12 hr). Acyclovir, an antiherpetic agent, potentiates the replication inhibiting activity of zidovudine in vitro. Seven men with persistent HIV antigen presence did not wish to undergo drug therapy, and they served as control subjects.

In Group A, five of six men exhibited reduced serum HIV-Ag levels during zidovudine therapy. The decline was statistically significant in three men, and three men

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NEWS BRIEFS are compiled and written by Vicki Glaser.

became HIV-Ag seronegative. Five of six subjects in Group B had significantly reduced levels of HIV-Ag, and four subjects became HIV-Ag seronegative. In Group C, acyclovir therapy produced no significant changes in serum HIV-Ag in five men; one subject exhibited a significant increase in HIV-Ag level. After zidovudine administration, HIV-Ag levels had significantly decreased in five subjects, with two becoming seronegative. In the control group, six of seven men had higher serum HIV-Ag levels; the level stayed stable in one subject.

Four subjects reported adverse effects of the drug treatment, including myalgia, insomnia, and impotence.

These results suggest that four-hour dosing is not imperative to inhibit viral replication. Zidovudine also appeared to stop immunologic deterioration. The authors reported a "striking regression of lymph node sizes" in the treatment groups. Acyclovir produced no apparent benefits, but, as the authors note, the sample population was small.

De Wolf et al conclude that "although little is known about the long-term efficacy or toxicity of the drug, we believe that the data presented here make a case for larger, placebo-controlled, trials of zidovudine in those subjects with CDC group II and III disease who are at high risk of progressing rapidly to AIDS."

Study of polio vaccine's effects on AIDS faces roadblock

Polio vaccines have demonstrated some ability to relieve the cancer lesions, herpes infections, swollen lymph nodes, and fatigue that can plague victims of AIDS (*Morning Call*, March 15, 1988, pp 1, 5). Researchers in Los Angeles, Calif, who developed this experimental treatment had planned to initiate a scientific study to evaluate how the vaccine affects the symptoms and opportunistic infections associated with AIDS. However, the Food and Drug Administration (FDA) has not approved polio vaccines as a treatment for AIDS, and the vaccine's manufacturer (Connaught Laboratories, Swiftwater, Pa), has refused to provide the vaccine to physicians who are using it to treat AIDS. The FDA and company officials support this decision, while AIDS advocate groups view the action as yet another bureaucratic obstacle in the search for treatments and a cure for AIDS.

Researchers from UCLA and Los Angeles County, who coordinated the planned study, appealed to Israel for the four liters of vaccine needed to perform the tests. AIDS treatment requires administration of the vaccine three times a week. Continued use of the vaccine has rid some patients of AIDS symptoms for as long as two years.

Connaught Laboratories is the only producer of the older injectable polio vaccine in the US. The company's new, improved version of the vaccine has not yet been tested against AIDS, but the company has also limited its distribution to physicians using it for polio prevention.

One of the researchers has charged that Connaught is withholding the old vaccine, which cannot be patented, to ensure that the company can enjoy the profits from the new vaccine, if it proves successful against AIDS. Company spokesmen have denied such accusations, stating that they have not patented the improved vaccine and that their actions, decided on together with the FDA, were based on laws governing the supply of drugs for approved purposes.

FDA moves ahead in the fight against AIDS

Frank E. Young, MD, PhD, Commissioner of Food and Drugs, of the Food and Drug Administration, presented a statement to the Presidential Commission on the Human Immunodeficiency Virus Epidemic on February 19, 1988. In his presentation, Young described how the FDA regulates drugs, vaccines, blood products, and medical devices; new procedures aimed at expediting the review of AIDS therapies and diagnostics; past accomplishments against the AIDS epidemic; and the FDA's future role in the war against AIDS. Following are some highlights:

- New procedures for reviewing AIDS drugs make them the FDA's highest priority and make investigational drugs more widely available.
- The first new drug application for AIDS, for zidovudine (or AZT), was approved in 3½ months—record time. The FDA has received 179 applications for approval to test 120 new AIDS drugs, diagnostics, and vaccines, and has approved 90% (154) of these thus far.
- Under the authority of the Orphan Drug Act, the FDA has designated AIDS therapies as eligible for tax incentives and grants for products of limited commercial value.
- The FDA has approved clinical studies for two AIDS vaccines.
- Several new blood screening products are under expedited review.
- In conjunction with the CDC, the FDA has developed recommendations for HIV testing of semen and organ and tissue transplants.
- The FDA regulates AIDS-related medical devices, such as condoms and rubber gloves, clinical devices that could transmit HIV, clinical devices to minimize risk of transmission, and devices to treat patients with AIDS. The FDA investigates fraudulent products intended for AIDS patients and those afraid of contracting the disease.
- The FDA sponsors an educational effort aimed at improving public knowledge about AIDS and ways to combat the disease.

California to determine incidence of HIV infection in newborns

The California Department of Health Services has announced a three-month program to test all babies born in the state for human immunodeficiency virus (HIV) (*Modern Healthcare*, February 19, 1988, p 49). Department officials hope the results will provide a reliable estimate of the incidence of HIV infection throughout the state. If a baby is infected, then the mother must also be infected with the AIDS virus.

The state's purpose in testing the approximately 120,000 newborns is to collect data for statistical analysis. All testing will be anonymous, and state officials will not notify parents or physicians of the results.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089-1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

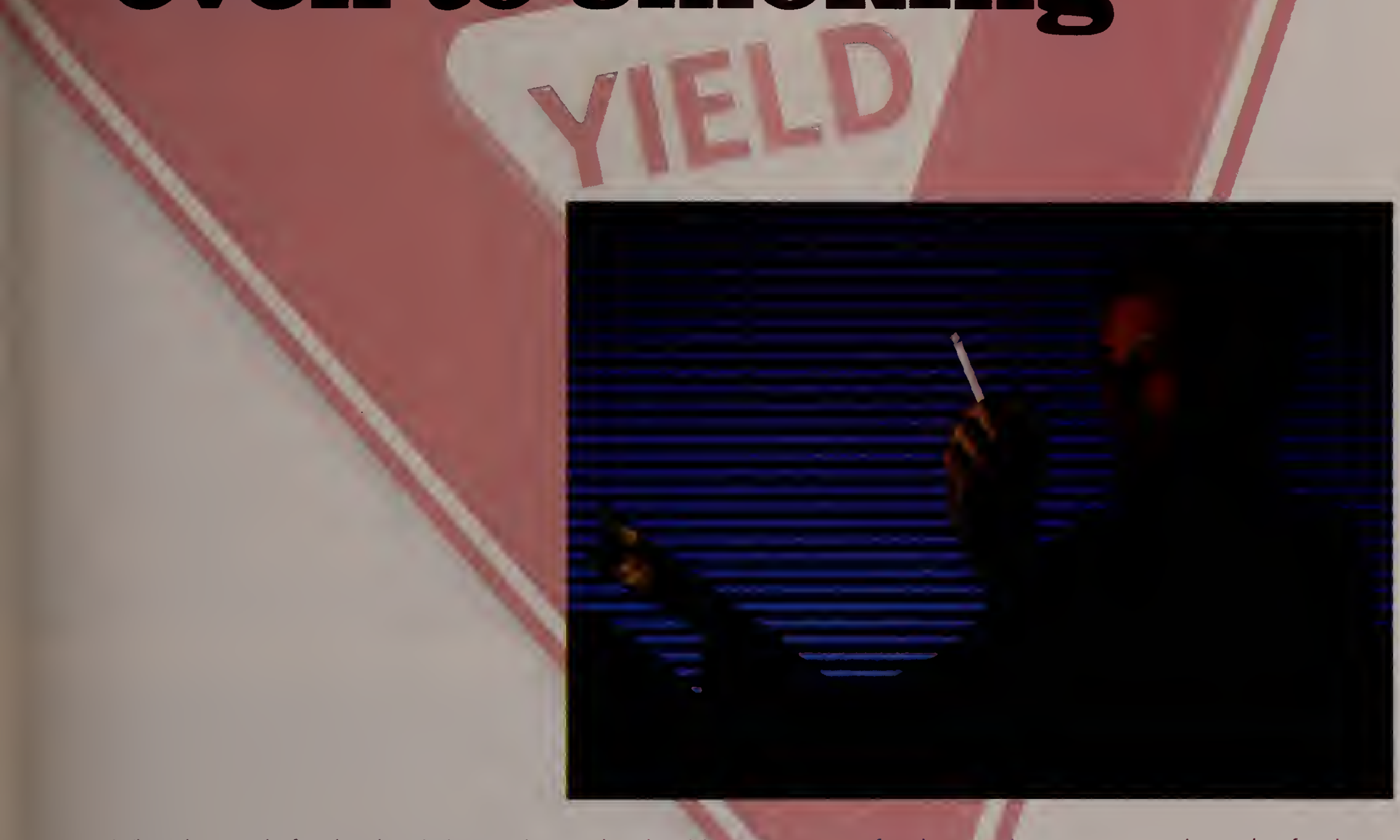
1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83: 925-927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337-338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Ulcer therapy that won't yield, even to smoking



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Ulcer healing rates:
(at four weeks of therapy)[5]

Sucralfate:	
All patients	79.4%
Smokers	81.6%*
Cimetidine:	
All patients	76.3%
Smokers	62.5%

*Significantly greater than cimetidine smoker group ($P<.05$).

Carafate has a unique, nonsystemic mode of action that enhances the body's own ulcer healing ability and protects the damaged mucosa from further injury.

When your ulcer patient is a smoker, prescribe the ulcer medication that won't go up in smoke: safe, nonsystemic Carafate.

Nothing works like

CARAFATE®
sucralfate/Marion

Please see adjoining page for references and brief summary of prescribing information.

0825A8

CARAFATE®
(sucralfate) Tablets

BRIEF SUMMARY

CONTRAINDICATIONS
There are no known contraindications to the use of sucralfate

PRECAUTIONS
Duodenal ulcer is a chronic, recurrent disease While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration

Drug Interactions: Animal studies have shown that simultaneous administration of CARAFATE (sucralfate) with tetracycline, phenytoin, digoxin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract The clinical significance of these animal studies is yet to be defined However, because of the potential of CARAFATE to alter the absorption of some drugs from the gastrointestinal tract, the separate administration of CARAFATE from that of other agents should be considered when alterations in bioavailability are felt to be critical for concomitantly administered drugs

Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose) There was no evidence of drug-related tumorigenicity A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment Mutagenicity studies were not conducted

Pregnancy: Teratogenic effects Pregnancy Category B Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate There are, however, no adequate and well-controlled studies in pregnant women Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4 7%)

Constipation was the most frequent complaint (2 2%) Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo

OVERDOSAGE
There is no experience in humans with overdosage Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose Risks associated with overdosage should, therefore, be minimal

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x ray or endoscopic examination

HOW SUPPLIED
CARAFATE (sucralfate) 1-gm tablets are supplied in bottles of 100 (NDC 0088 1712 47) and in Unit Dose Identification Paks of 100 (NDC 0088-1712-49) Light pink scored oblong tablets are embossed with CARAFATE on one side and 1712 bracketed by C's on the other

Issued 1/87

References:
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3 Brandstaetter G, Kratochvil P *Am J Med* 79 (suppl 2C) 36-38, 1985
4 Marks IN, Wright JP, Gilinsky NH, et al *J Clin Gastroenterol* 8 419-423, 1986
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PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

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ARMY RESERVE MEDICAL PROFILE NO.7

Soldier being examined for effects of high-altitude cerebral edema.

ALLAN J. HAMILTON, M.D.

Neurosurgical Resident and Research Fellow, Massachusetts General Hospital, Boston, Massachusetts. Captain, U.S. Army Reserve.

EDUCATION Ithaca College, B.A. (Magna Cum Laude); Hamilton College (Pre-med); Harvard Medical School.

RESIDENCY General Surgical Internship. Neurosurgical Residency, Massachusetts General Hospital.

CONTINUING EDUCATION Neurology and Neurosurgery Research Fellowship Training, National Institutes of Health.

OUTSTANDING ACHIEVEMENTS Olsen Memorial Fellowship, National Masonic Medical Research Foundation; Albert Schweitzer Fellowship, International Albert Schweitzer Foundation; Harvard Medical School Cabot Prize for Best Senior Thesis; recently published article, "Who Shall Live and Who Shall Die" in Newsweek Magazine.

"The work I'm doing in the Army Reserve fits perfectly with my academic research interests in civilian life. The Army is very concerned with the effects of high-altitude cerebral edema, which is a mirror model of cerebral hypoxia, something I deal with every day in our neurosurgical intensive care unit. I couldn't ask for a smoother transition. And that's true for a lot of Reserve physicians. All we really do is change our clothes, not our mindset.

"Some of the projects the Army is undertaking are on the cutting edge of research. For example, I'm currently involved in developing for the Army a prototype of a non-invasive intracranial pressure-monitoring device that we hope will allow us to measure pressure changes as the brain swells—without drilling holes in the skull. If we can get our design to work, such a device could revolutionize high-altitude medicine as well as civilian neurosurgical care.

"The quality of medicine and the caliber of people I've been associated with in the Army Reserve are, without question, equal to civilian hospitals. In fact, I'm giving serious consideration to applying for an active duty academic position in Army Medicine when my residency ends at Massachusetts General."

Find out more about the medical opportunities in the Army Reserve. Call toll free 1-800-USA-ARMY.

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No other estrogen proven effective for osteoporosis

Only conjugated estrogens tablets have established efficacy in both osteoporosis[1] and vasomotor symptoms* at 0.625 mg/day. No other estrogen, oral or transdermal, has established clinical evidence or minimum effective dose in both indications.

No estrogen proven safer

PREMARIN is the most extensively tested estrogen, with an unsurpassed record of long-term safety.
And clinical evidence shows a significantly reduced risk of endometrial hyperplasia when cycled with a progestin.[2]

PREMARIN®
(conjugated estrogens tablets)

Most trusted for more reasons

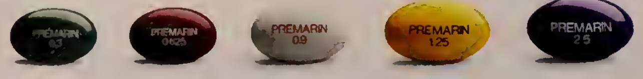
*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary
of prescribing information.

For moderate-to-severe vasomotor symptoms and for osteoporosis

PREMARIN®

(conjugated estrogens tablets)



0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

The appearance of these tablets is a trademark of Ayerst Laboratories.

For atrophic vaginitis

PREMARIN®

(conjugated estrogens)

Vaginal Cream

0.625 mg/g

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream, in a nonliquefying base

1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA.
Three independent, case-controlled studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case-controlled studies reported that the risk of endometrial cancer in estrogen users was about 4.5 to 13.9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible. When prolonged treatment is medically indicated, the patient should be reassessed on at least a semi-annual basis to determine the need for continued therapy. Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy. There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equi-estrogenic doses.
2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY
The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring. It has been shown that females exposed in utero to diethylstilbestrol, a nonsteroidal estrogen, have an increased risk of developing, in later life, a form of vaginal or cervical cancer that is ordinarily extremely rare. This risk has been estimated as not greater than 4 per 1,000 exposures. Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix. Although these changes are histologically benign, it is not known whether they are precursors of malignancy. Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes. Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb-reduction defects. One case-controlled study estimated a 4.7-fold increased risk of limb-reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb-reduction defects in exposed fetuses is somewhat less than 1 per 1,000. In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well-controlled studies that progestogens are effective for these uses. If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine. It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters. Tablets are available in 0.3 mg, 0.625 mg, 0.9 mg, 1.25 mg, and 2.5 mg strengths of conjugated estrogens. Cream is available as 0.625 mg conjugated estrogens per gram.
INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause. (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) Osteoporosis (abnormally low bone mass). Atrophic vaginitis. Kraurosis vulvae. Female castration.

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae.

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).
Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized. Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia. Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes. Whether this will provide protection from endometrial carcinoma has not been clearly established. There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens. (See PRECAUTIONS.) The choice of progestin and dosage may be important, product labeling should be reviewed to minimize possible adverse effects.
CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions. 1. Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease. 2. Known or suspected estrogen-dependent neoplasia. 3. Known or suspected pregnancy (see Boxed Warning). 4. Undiagnosed abnormal genital bleeding. 5. Active thrombophlebitis or thromboembolic disorders. 6. A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).
WARNINGS: Estrogens have been reported to increase the risk of endometrial carcinoma (see Boxed Warning). However, a recent large, case-controlled study indicated no increase in risk of breast cancer in postmenopausal women. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement, it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction. Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users. An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives. If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization. Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use. They should be used with caution in patients with cerebral vascular or coronary artery disease. Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism, and thrombophlebitis. When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk.

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock. Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use. A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives. For this reason, diabetic patients should be carefully observed. Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases.
PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed. Conditions influenced by fluid retention, such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc. Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed. Pre-existing uterine leiomyomata may increase in size during estrogen use. The pathologist should be advised of estrogen therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated. Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia or in young patients in whom bone growth is not yet complete. If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

The following changes may be expected with larger doses of estrogen:

a. Increased sulfobromophthalein retention.

b. Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3, increased norepinephrine-induced platelet aggregability.

c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T_4 by column, or T_4 by radioimmunoassay. Free T_3 resin uptake is decreased, reflecting the elevated TBG; free T_4 concentration is unaltered.

d. Impaired glucose tolerance.

e. Decreased pregnanediol excretion.

f. Reduced response to metyrapone test.

g. Reduced serum folate concentration.

h. Increased serum triglyceride and phospholipid concentration.

As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk.

Long-term, continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. However, in a recent, large case-controlled study of postmenopausal women there was no increase in risk of breast cancer with use of conjugated estrogens.
ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea, premenstrual-like syndrome, amenorrhea during and after treatment, increase in size of uterine fibromyomata, vaginal candidiasis, change in cervical erosion and in degree of cervical secretion; cystitis-like syndrome, tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating, cholestatic jaundice; chloasma or melasma which may persist when drug is discontinued, erythema multiforme, erythema nodosum; hemorrhagic eruption, loss of scalp hair; hirsutism, steepening of corneal curvature; intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea, increase or decrease in weight; reduced carbohydrate tolerance, aggravation of porphyria; edema, changes in libido.
ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females.
DOSAGE AND ADMINISTRATION:
PREMARIN® Brand of conjugated estrogens tablets, USP
1. *Given cyclically for short-term use only.* For treatment of moderate-to-severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0.3 mg to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible. Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals.
2. *Given cyclically:* Osteoporosis. Female castration. Osteoporosis—0.625 mg daily. Administration should be cyclic (eg, three weeks on and one week off). Female castration—1.25 mg daily, cyclically. Adjust upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level that will provide effective control.

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.
PREMARIN® Brand of conjugated estrogens Vaginal Cream
Given cyclically for short-term use only. For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three- to six-month intervals.

Usual dosage range: 2 g to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.
References:
1. Lindsay R, Hart DM, Clark DM. The minimum effective dose of estrogen for prevention of postmenopausal bone loss. *Obstet Gynecol* 1984;63:759-763. **2.** Studd JWW, Thom MH, Paterson MEL, et al: The prevention and treatment of endometrial pathology in postmenopausal women receiving exogenous estrogens, in Pasetto N, Paoletti R, Ambrus JL (eds): *The Menopause and Postmenopause.* Lancaster, England, MTP Press Ltd, 1980, chap 13.

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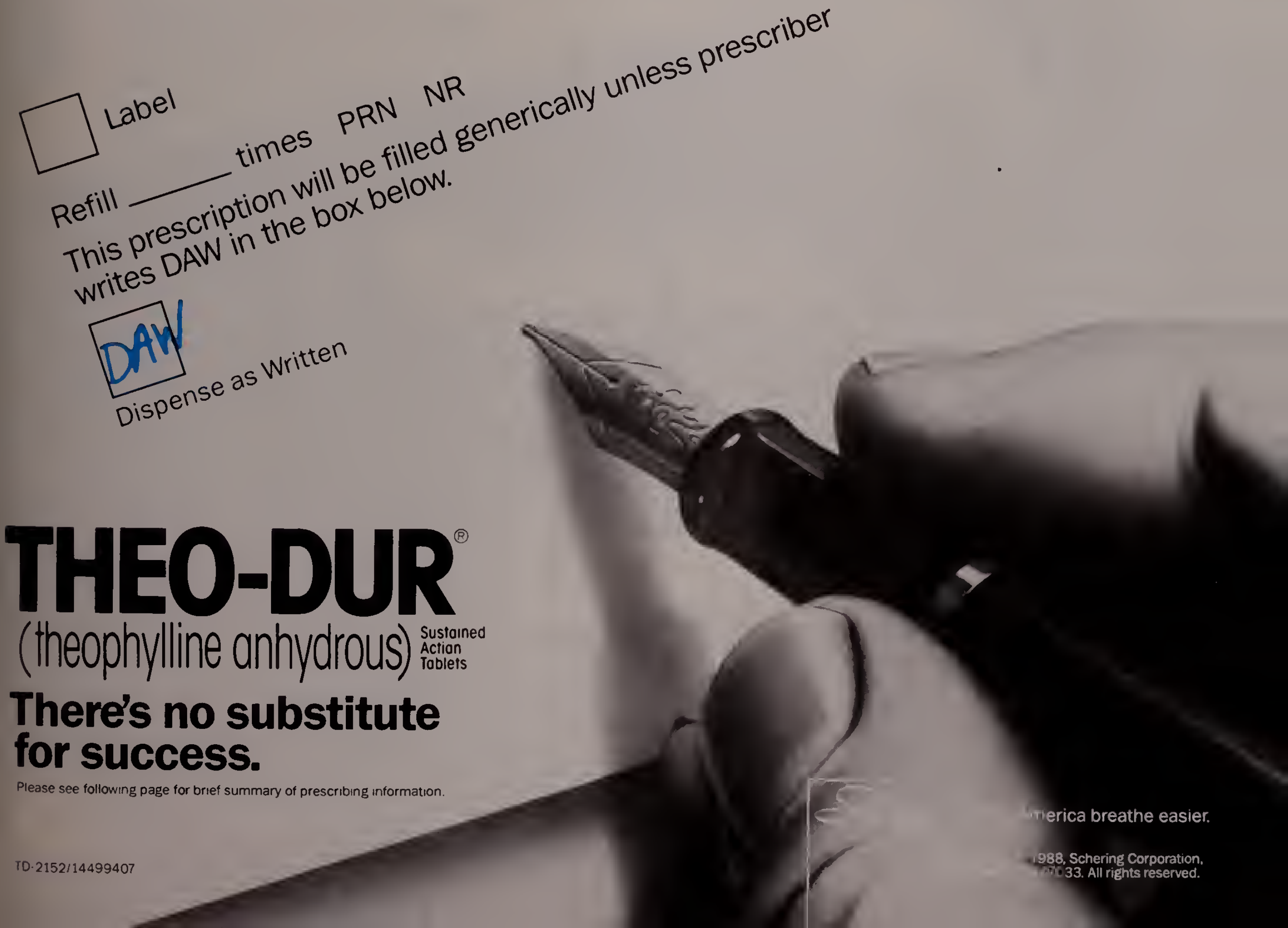

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DAW

Dispense as Written

THEO-DUR®
(theophylline anhydrous) Sustained Action Tablets

There's no substitute for success.

Please see following page for brief summary of prescribing information.

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TD-2152/14499407

THEO-DUR®

THEOPHYLLINE (Anhydrous)
Sustained Action Tablets

INDICATIONS: THEO-DUR is indicated for relief and/or prevention of symptoms of asthma and for reversible bronchospasm associated with chronic bronchitis and emphysema

CONTRAINDICATIONS: THEO-DUR is contraindicated in individuals who have shown hypersensitivity to theophylline or any of the tablet components

WARNINGS: Status asthmaticus should be considered a medical emergency and is defined as that degree of bronchospasm which is not rapidly responsive to usual doses of conventional bronchodilators Optimal therapy for such patients frequently requires both *additional medication* parenterally administered, and *close monitoring*, preferably in an intensive care setting

Although increasing the dose of theophylline may bring about relief, such treatment may be associated with toxicity The likelihood of such toxicity developing increases significantly when the serum theophylline concentration exceeds 20 mcg/ml Therefore, determination of serum theophylline levels is recommended to assure maximal benefit without excessive risk

Serum levels above 20 mcg/ml are rarely found after appropriate administration of recommended doses However, in individuals in whom theophylline plasma clearance is reduced *for any reason*, even conventional doses may result in increased serum levels and potential toxicity Reduced theophylline clearance has been documented in the following readily identifiable groups 1) patients with impaired renal or liver function, 2) patients over 55 years of age, particularly males and those with chronic lung disease, 3) those with cardiac failure from any cause, 4) neonates, and 5) those patients taking certain drugs (macrolide antibiotics and cimetidine) Decreased clearance of theophylline may be associated with either influenza immunization or active infection with influenza

Reduction of dosage and laboratory monitoring is especially appropriate in the above individuals Less serious signs of theophylline toxicity (i e nausea and restlessness) may occur frequently when initiating therapy, but are usually transient, when such signs are persistent during maintenance therapy, they are often associated with serum concentrations above 20 mcg/ml Unfortunately, however, serious side effects such as ventricular arrhythmias, convulsions or even death may appear as the first sign of toxicity without any previous warning Stated differently *serious toxicity is not reliably preceded by less severe side effects*

Many patients who require theophylline may exhibit tachycardia due to their underlying disease process so that the cause/effect relationship to elevated serum theophylline concentrations may not be appreciated

Theophylline products may cause dysrhythmia and/or worsen pre-existing arrhythmias and any significant change in rate and/or rhythm warrants monitoring and further investigation

The occurrence of arrhythmias and sudden death (with histological evidence of necrosis of the myocardium) has been recorded in laboratory animals (minipigs, rodents and dogs) when theophylline and beta agonists were administered concomitantly, although not when either was administered alone The significance of these findings when applied to human usage is currently unknown

PRECAUTIONS: THEO-DUR TABLETS SHOULD NOT BE CHEWED OR CRUSHED.

General: Theophylline half-life is shorter in smokers than in non-smokers. Therefore, smokers may require larger or more frequent doses Morphine and curare should be used with caution in patients with airway obstruction as they may suppress respiration and stimulate histamine release Alternative drugs should be used when possible Theophylline should not be administered concurrently with other xanthine medications Use with caution in patients with severe cardiac disease, severe hypoxemia, hypertension, hyperthyroidism, acute myocardial injury, cor pulmonale, congestive heart failure, liver disease, in the elderly (especially males) and in neonates In particular, great caution should be used in giving theophylline to patients with congestive heart failure. Frequently, such patients have markedly prolonged theophylline serum levels with theophylline persisting in serum for long periods following discontinuation of the drug Individuals who are rapid metabolizers of theophylline, such as the young, smokers, and some non-smoking adults, may not be suitable candidates for once-daily dosing These individuals will generally need to be dosed at 12 hour or sometimes 8 hour intervals. Such patients may exhibit symptoms of bronchospasm near the end of a dosing interval, or may have wider peak-to-trough differences than desired

Use theophylline cautiously in patients with history of peptic ulcer Theophylline may occasionally act as a local irritant to the G I tract although gastrointestinal symptoms are more commonly centrally mediated and associated with serum drug concentrations over 20 mcg/ml

Information for Patients: The physician should reinforce the importance of taking only the prescribed dose and time interval between doses THEO-DUR tablets should not be chewed or crushed When dosing THEO-DUR on a once daily (q24h) basis, tablets should be taken whole and not split As with any controlled-release theophylline product, the patient should alert the physician if symptoms occur repeatedly, especially near the end of the dosing interval

DRUG INTERACTIONS: **Drug-Drug**: Toxic synergism with ephedrine has been documented and may occur with some other sympathomimetic bronchodilators. In addition, the following drug interactions have been demonstrated.

Drug	Effect
Theophylline with lithium carbonate	Increased excretion of lithium carbonate
Theophylline with propranolol	Antagonism of propranolol effect
Theophylline with cimetidine	Increased theophylline blood levels
Theophylline with troleandomycin, erythromycin	Increased theophylline blood levels

Drug-Food: *THEO-DUR 100 mg Sustained Action Tablets* have not been adequately studied to determine whether their bioavailability is altered when given with food Available data suggest that drug administration at the time of food ingestion may influence the absorption characteristics of theophylline controlled-release products resulting in serum values different from those found after administration in the fasting state.

A drug-food effect, if any would likely have its greatest clinical significance when high theophylline serum levels are being maintained and/or when large single doses (greater than 13 mg/kg or 900 mg) of a controlled-release theophylline product are given

THEO-DUR (200, 300, and 450 mg) Sustained Action Tablets The rate and extent of absorption of theophylline from THEO-DUR 200 mg, 300 mg, and 450 mg tablets when administered fasting or immediately after a moderately high fat content breakfast is similar

Drug-Laboratory Test Interactions: When plasma levels of theophylline are measured by spectrophotometric methods, coffee tea, cola beverages, chocolate, and acetaminophen contribute falsely high values.

Carcinogenesis, Mutagenesis, and Impairment of Fertility: Long-term animal studies have not been performed to evaluate the carcinogenic potential, mutagenic potential, or the effect on fertility of xanthine compounds

Pregnancy: Category C—Animal reproduction studies have not been conducted with theophylline It is not known whether theophylline can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Xanthines should be given to a pregnant woman only if clearly needed

Nursing Mothers: It has been reported that theophylline distributes readily into breast milk and may cause adverse effects in the infant Caution must be used if prescribing xanthine to a mother who is nursing, taking into account the risk-benefit of this therapy

Pediatric Use: Safety and effectiveness of THEO-DUR administered

1 Every 24 hours in children under 12 years of age, have not been established
2 Every 12 hours in children under 6 years of age, have not been established

ADVERSE REACTIONS: The most consistent adverse reactions are usually due to overdose and are

1 *Gastrointestinal* nausea vomiting epigastric pain, hematemesis diarrhea
2 *Central nervous system* headaches, irritability, restlessness, insomnia, reflex hyperexcitability, muscle twitching, clonic and tonic generalized convulsions
3 *Cardiovascular* palpitation, tachycardia extrasystoles, flushing, hypotension circulatory failure, ventricular arrhythmias
4 *Respiratory* tachypnea
5 *Renal* albuminuria, increased excretion of renal tubular and red blood cells, potentiation of diuresis
6 *Other* rash hyperglycemia and inappropriate ADH syndrome

OVERDOSAGE: **Management**: If potential oral overdose is established and seizure has not occurred

A Induce vomiting
B Administer a cathartic (This is particularly important if sustained-release preparations have been taken)
C Administer activated charcoal

If patient is having a seizure

A Establish an airway
B Administer oxygen
C Treat the seizure with intravenous diazepam 0 1 to 0 3 mg/kg up to 10 mg
D Monitor vital signs maintain blood pressure and provide adequate hydration

Post Seizure Coma:

A Maintain airway and oxygenation
B If a result of oral medication follow above recommendations to prevent absorption of the drug, but intubation and lavage will have to be performed instead of inducing emesis, and the cathartic and charcoal will need to be introduced via a large bore gastric lavage tube
C Continue to provide full supportive care and adequate hydration while waiting for drug to be metabolized In general the drug is metabolized sufficiently rapid so as not to warrant consideration of dialysis however if serum levels exceed 50 mcg/ml charcoal hemoperfusion may be indicated

CAUTION Federal law prohibits dispensing without prescription For full prescribing information, see package insert

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Francisco. Contact: Continuing Education S/P, University of California, Box 0446, San Francisco, CA 94143. Tel: (415) 476-4194.

ILLINOIS

June 8–10. **Recent Advances in the Diagnosis and Management of Malignant Disease.** Searle Conference Center, Chicago. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

PENNSYLVANIA

June 13–17. **Critical Issues in Tumor Microcirculation Angiogenesis and Metastasis: Biological Significance and Clinical Relevance.** 35 Cat 1 Credits. Adamson Wing, Baker Hall, University of Pittsburgh. Contact: R. Hilda Diamond, Associate Director, Biomedical Engineering Program, DH 2313, Carnegie Mellon University, Pittsburgh, PA 15213-3890.

SOUTH CAROLINA

June 1–4. **Ninth Conference on Computer Applications in Radiology.** 21 Cat 1 Credits. Mariner's Inn, Hilton Head. Contact: Ms Janice Ford, Continuing Education Coordinator, Department of Radiology, Hospital of the University of Pennsylvania, 3400 Spruce St, Philadelphia, PA 19104. Tel: (215) 662-6904 or 662-6982.

June 20–25. **Intensive Review of Family Medicine.** 43½ Cat 1 Credits. Wild Dunes Ocean Resort Hotel, Charleston. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

TEXAS

June 16–18. **AIDS and Infectious Disease Update.** Hilton Resort, South Padre Island. Contact: Scott and White Office of Continuing Medical Education, 2401 S 31st St, Temple, Texas 76508. Tel: (817) 774-2350.

AROUND THE WORLD

CANADA

June 1–4. **Medical Complications of Pregnancy.** Park Plaza Hotel, Toronto, Ontario. *June 27–30.* **Critical Care Medicine—1988.** Banff Park Lodge, Banff, Alberta. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

June 3–4. **Fundamentals of Geriatric Psychiatry.** Medical Sciences Building, University of Toronto. Contact: Continuing Education, Faculty of Medicine, Medical Sciences Building, University of Toronto, Toronto, Ontario, Canada M5S 1A8. Tel: (416) 978-2718.

FRANCE

June 11–16. **American and European Views on Critical Care.** 24 Cat 1 Credits. Hotel Martinez, Cannes. Contact: Professional Seminars/University of Miami, PO Box 012318, Miami, FL 33101. Tel: (305) 547-6411.

KENYA

June 19–July 3. **Diagnosis and Treatment of Infectious Diseases.** 25 Cat 1 Credits. Inter-Continental Hotel, Nairobi. Contact: Danny Flood, Director, Public Relations, Albert Einstein College of Medicine/Montefiore Medical Center, Office of Continuing Medical Education, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674. □

Before prescribing, see complete prescribing information in SK&F LAB CO. literature or PDR. The following is a brief summary.

Contraindications: There are no known contraindications to the use of 'Tagamet'.

Precautions: While a weak antiandrogenic effect has been demonstrated in animals, 'Tagamet' has been shown to have no effect on spermatogenesis, sperm count, motility, morphology or in vitro fertilizing capacity in humans.

In a 24-month toxicity study in rats at dose levels approximately 9 to 56 times the recommended human dose, benign Leydig cell tumors were seen. These were common in both the treated and control groups, and the incidence became significantly higher only in the aged rats receiving 'Tagamet'.

Rare instances of cardiac arrhythmias and hypotension have been reported following the rapid administration of 'Tagamet' HCl (brand of cimetidine hydrochloride) Injection by intravenous bolus.

Symptomatic response to 'Tagamet' therapy does not preclude the presence of a gastric malignancy. There have been rare reports of transient healing of gastric ulcers despite subsequently documented malignancy.

Reversible confusional states have been reported on occasion, predominantly in severely ill patients.

'Tagamet' has been reported to reduce the hepatic metabolism of warfarin-type anticoagulants, phenytoin, propranolol, chlordiazepoxide, diazepam, lidocaine, theophylline and metronidazole. Clinically significant effects have been reported with the warfarin anticoagulants; therefore, close monitoring of prothrombin time is recommended, and adjustment of the anticoagulant dose may be necessary when 'Tagamet' is administered concomitantly. Interaction with phenytoin, lidocaine and theophylline has also been reported to produce adverse clinical effects.

However, a crossover study in healthy subjects receiving either 'Tagamet' 300 mg. q.i.d. or 800 mg. h.s. concomitantly with a 300 mg. b.i.d. dosage of theophylline (Theo-Dur®, Key Pharmaceuticals, Inc.), demonstrated less alteration in steady-state theophylline peak serum levels with the 800 mg. h.s. regimen, particularly in subjects aged 54 years and older. Data beyond ten days are not available. (Note: All patients receiving theophylline should be monitored appropriately, regardless of concomitant drug therapy.)

Lack of experience to date precludes recommending 'Tagamet' for use in pregnant patients, women of childbearing potential, nursing mothers or children under 16 unless anticipated benefits outweigh potential risks; generally, nursing should not be undertaken in patients taking the drug since cimetidine is secreted in human milk.

Adverse Reactions: Diarrhea, dizziness, somnolence, headache, rash. Reversible arthralgia, myalgia and exacerbation of joint symptoms in patients with preexisting arthritis have been reported. Reversible confusional states (e.g., mental confusion, agitation, psychosis, depression, anxiety, hallucinations, disorientation), predominantly in severely ill patients, have been reported. Gynecomastia and reversible impotence in patients with pathological hypersecretory disorders receiving 'Tagamet', particularly in high doses, for at least 12 months, have been reported. Reversible alopecia has been reported very rarely. Decreased white blood cell counts in 'Tagamet'-treated patients (approximately 1 per 100,000 patients), including agranulocytosis (approximately 3 per million patients), have been reported, including a few reports of recurrence on rechallenge. Most of these reports were in patients who had serious concomitant illnesses and received drugs and/or treatment known to produce neutropenia. Thrombocytopenia (approximately 3 per million patients) and a few cases of aplastic anemia have also been reported. Increased serum transaminase and creatinine, as well as rare cases of fever, interstitial nephritis, urinary retention, pancreatitis and allergic reactions, including hypersensitivity vasculitis, have been reported. Reversible adverse hepatic effects, cholestatic or mixed cholestatic-hepatocellular in nature, have been reported rarely. Because of the predominance of cholestatic features, severe parenchymal injury is considered highly unlikely. A single case of biopsy-proven periportal hepatic fibrosis in a patient receiving 'Tagamet' has been reported.

How Supplied: Tablets: 200 mg. tablets in bottles of 100; 300 mg. tablets in bottles of 100 and Single Unit Packages of 100 (intended for institutional use only); 400 mg. tablets in bottles of 60 and Single Unit Packages of 100 (intended for institutional use only), and 800 mg. Tiltab® tablets in bottles of 30 and Single Unit Packages of 100 (intended for institutional use only).

Liquid: 300 mg./5 ml., in 8 fl. oz. (237 ml.) amber glass bottles and in single-dose units (300 mg./5 ml.), in packages of 10 (intended for institutional use only).

Injection:
Vials: 300 mg./2 ml. in single-dose vials, in packages of 10 and 30, and in 8 ml. multiple-dose vials, in packages of 10 and 25.

Prefilled Syringes: 300 mg./2 ml. in single-dose prefilled disposable syringes.

Plastic Containers: 300 mg. in 50 ml. of 0.9% Sodium Chloride in single-dose plastic containers, in packages of 4 units. No preservative has been added.

ADD-Vantage®* Vials: 300 mg./2 ml. in single-dose ADD-Vantage® Vials, in packages of 25.

Exposure of the premixed product to excessive heat should be avoided. It is recommended the product be stored at controlled room temperature. Brief exposure up to 40°C does not adversely affect the premixed product.

'Tagamet' HCl (brand of cimetidine hydrochloride) Injection premixed in single-dose plastic containers is manufactured for SK&F Lab Co. by Travenol Laboratories, Inc., Deerfield, IL 60015.

* ADD-Vantage® is a trademark of Abbott Laboratories.

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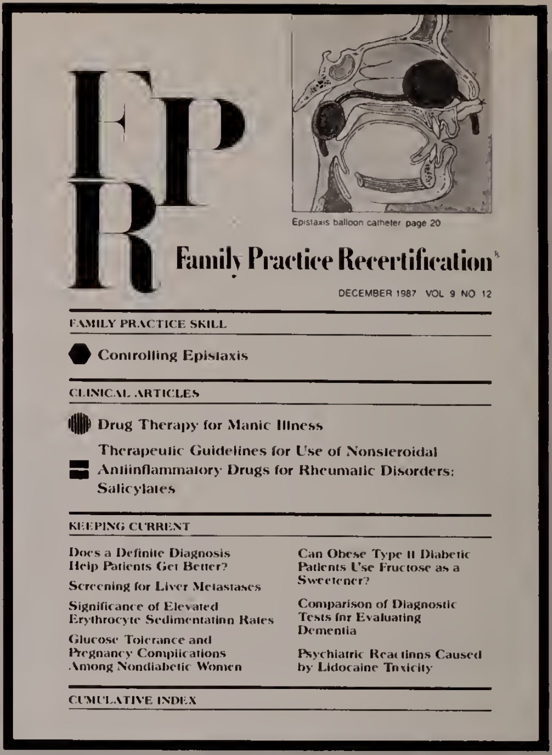

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This new soft-cover manual will be available for distribution by May 1988. **The price schedule for the Manual is as follows: $5.00 for a single copy; $4.00/copy for 2–10 copies; $3.50/copy for more than 10 copies mailed; $3.00/copy for more than 10 copies picked up.**

For further information, please contact: **Jacqueline Messite, M.D., Director, Office of Public Health, the New York Academy of Medicine, 2 East 103rd Street, New York, NY 10029, (212) 876-8200 ext. 250.**

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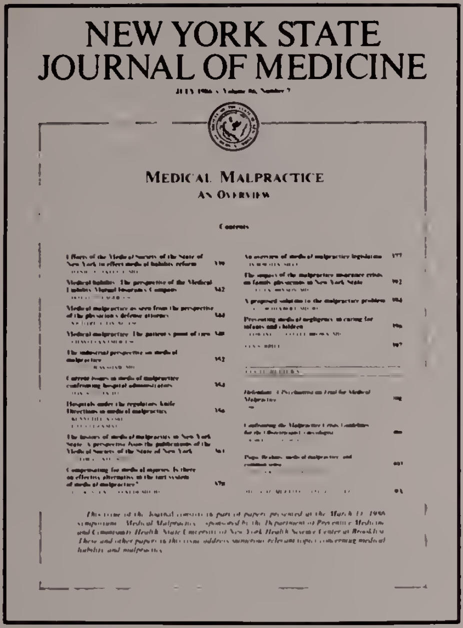
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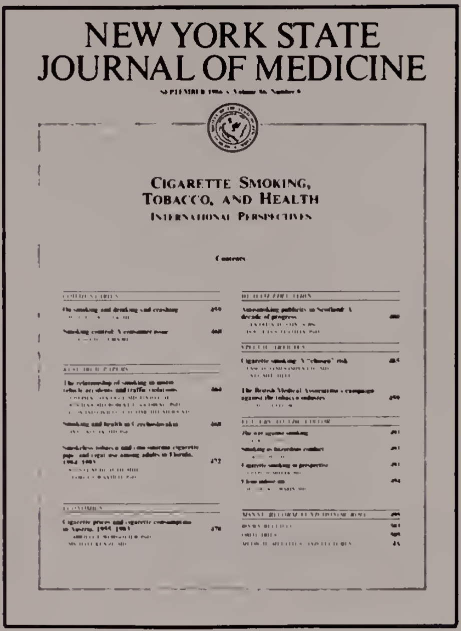
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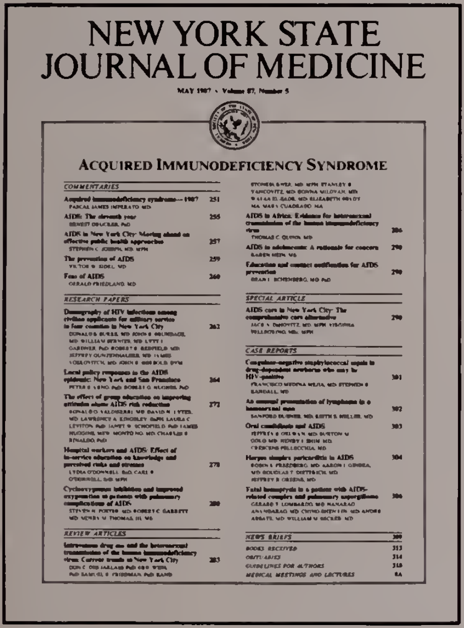
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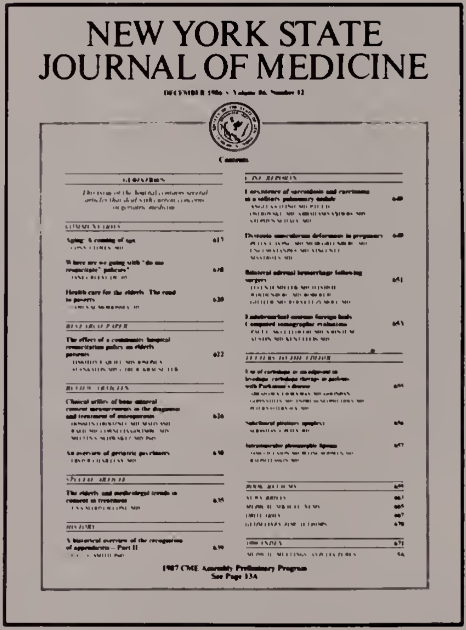
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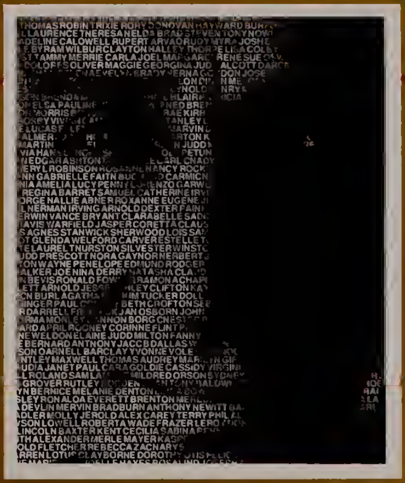

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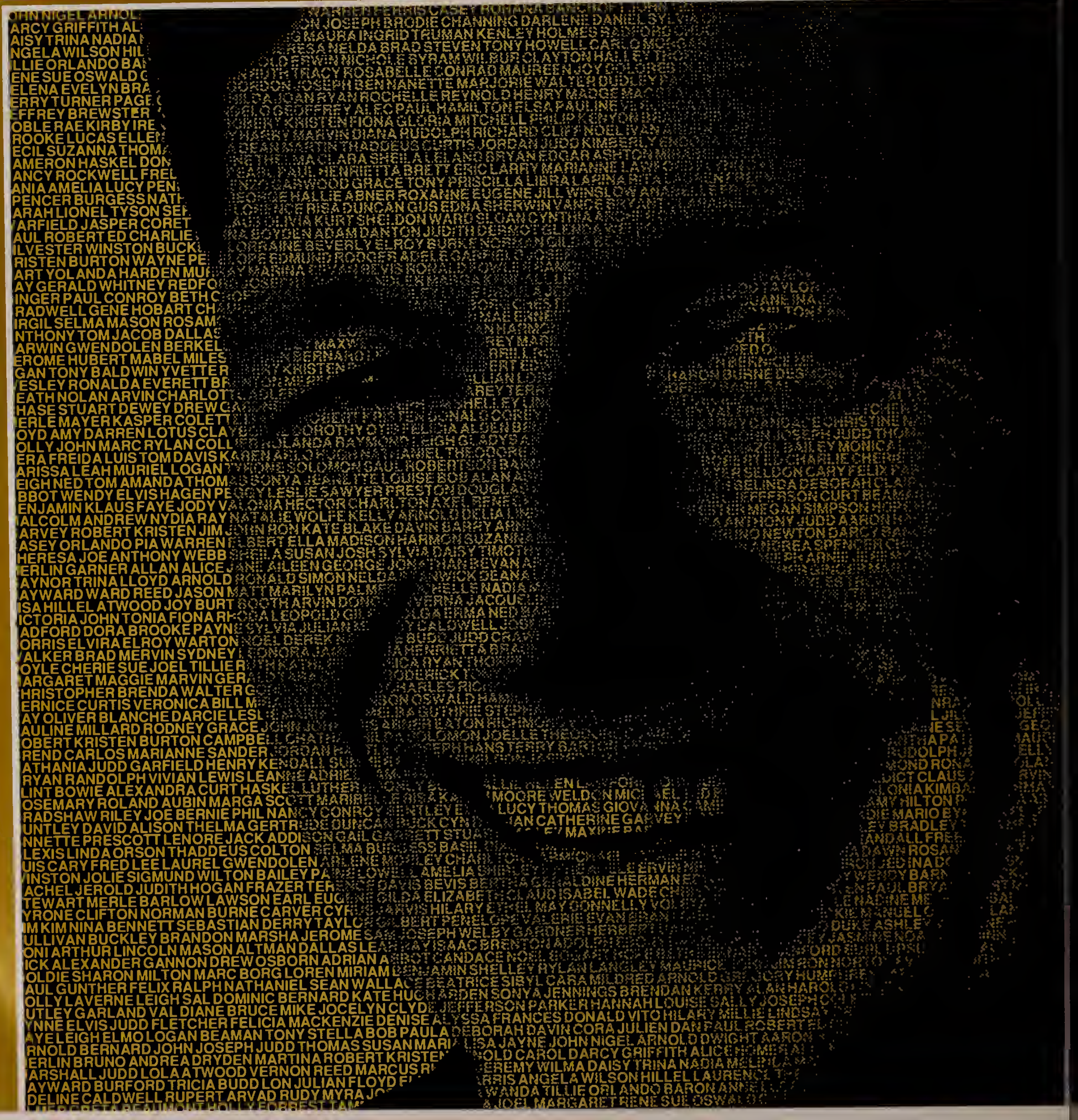
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In a recent survey, 4,120 participating physicians gave us their views[1] on INDERAL LA in the treatment of hypertension, angina and migraine.

INDERAL LA is their preferred beta blocker

...of the nearly three out of four physicians responding to the questionnaire, an impressive 97% rated INDERAL LA good to excellent for overall performance. Virtually all cited efficacy, tolerability, long-term cardiovascular protection and once-daily convenience as important factors in their choosing to prescribe INDERAL LA.

INDERAL LA promotes patient compliance

...Virtually every responding physician rated patient satisfaction with INDERAL LA to be as good as, or better than, other beta blockers.

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree and bronchial asthma.

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INDERAL® LA

(PROPRANOLOL HCl)

LONG ACTING CAPSULES
60, 80, 120, 160 mg

The one you know best keeps looking better

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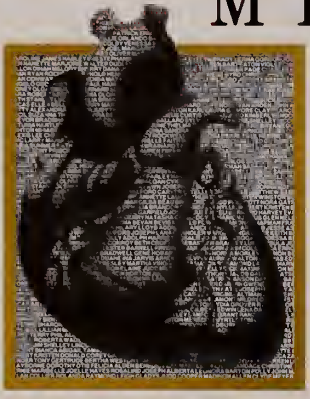

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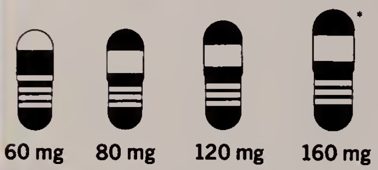

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride. INDERAL LA is available as 60 mg, 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure, and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first-degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected. Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL (propranolol HCl) is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Caution should be exercised when patients receiving a beta blocker are administered a calcium-channel-blocking drug, especially intravenous verapamil, for both agents may depress myocardial contractility or atrioventricular conduction. On rare occasions, the concomitant intravenous use of a beta blocker and verapamil has resulted in serious adverse reactions, especially in patients with severe cardiomyopathy, congestive heart failure, or recent myocardial infarction.

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol.

Ethanol slows the rate of absorption of propranolol.

Phenytoin, *phenobarbitone*, and *rifampin* accelerate propranolol clearance.

Chlorpromazine, when used concomitantly with propranolol, results in increased plasma levels of both drugs.

Antipyrine and *lidocaine* have reduced clearance when used concomitantly with propranolol.

Thyroxine may result in a lower than expected T_3 concentration when used concomitantly with propranolol.

Cimetidine decreases the hepatic metabolism of propranolol, delaying elimination and increasing blood levels.

Theophylline clearance is reduced when used concomitantly with propranolol.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS: INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type.

Central Nervous System: Light-headedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; vivid dreams; an acute reversible syndrome characterized by disorientation for time and place; short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. For immediate formulations, fatigue, lethargy, and vivid dreams appear dose related.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL Tablets to INDERAL LA Capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg-for-mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary, especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—*Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three- to seven-day intervals until optimal response is obtained. Although individual patients may respond at any dosage level, the average optimal dosage appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE—*Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimal migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximal dose, INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of these capsules is a registered trademark of Ayerst Laboratories.

Reference:

1. Data on file, Ayerst Laboratories.

D7295/188

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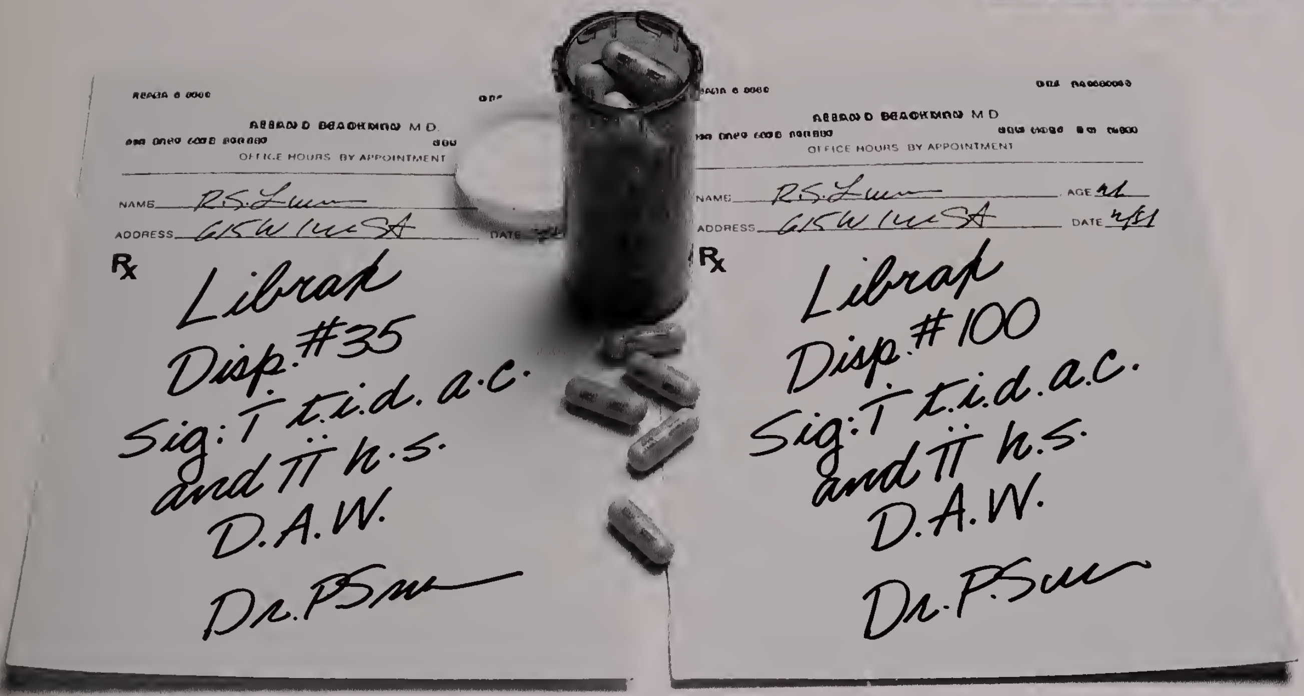

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Rx

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Disp. #35
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and ii h.s.
D.A.W.

NAME R.S. Lum

Rx

Librax
Disp. #100
Sig: ī t.i.d. a.c.
and ii h.s.
D.A.W.

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

> ***Usage in Pregnancy:*** **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.
Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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To insist on the brand, be sure to specify "Dispense as Written."

It's time for the Peacemaker.

In irritable bowel syndrome* anxiety can aggravate intestinal symptoms, which may further intensify anxiety — a distressing cycle of brain/bowel conflict. Librax intervenes with two well-known compounds. The Librium® (chlordiazepoxide HCl/Roche) component safely relieves anxiety. And Quarzan® (clidinium bromide/Roche) provides antisecretory and antispasmodic action to relieve discomfort associated with intestinal hypermotility.

Dual action — for peace between brain and bowel. Because of possible CNS effects, caution patients about engaging in activities requiring complete mental alertness. Specify Adjunctive

LIBRAX®

Each capsule contains 5 mg chlordiazepoxide HCl
and 2.5 mg clidinium bromide

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of peptic ulcer and the irritable bowel syndrome.

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STACKS

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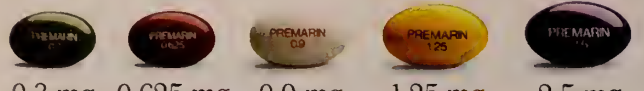

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For atrophic vaginitis

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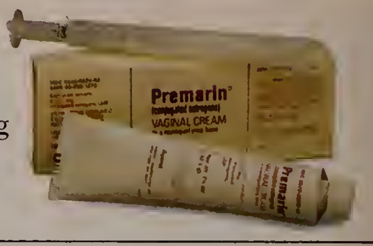
Vaginal Cream

0.625 mg/g



BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream, in a nonliquefying base

1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA.

Three independent, case-controlled studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case-controlled studies reported that the risk of endometrial cancer in estrogen users was about 4.5 to 13.9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible. When prolonged treatment is medically indicated, the patient should be reassessed on at least a semi-annual basis to determine the need for continued therapy. Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy. There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equi-estrogenic doses.

2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY.

The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring. It has been shown that females exposed in utero to diethylstilbestrol, a nonsteroidal estrogen, have an increased risk of developing, in later life, a form of vaginal or cervical cancer that is ordinarily extremely rare. This risk has been estimated as not greater than 4 per 1,000 exposures. Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix. Although these changes are histologically benign, it is not known whether they are precursors of malignancy. Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes. Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb-reduction defects. One case-controlled study estimated a 4.7-fold increased risk of limb-reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb-reduction defects in exposed fetuses is somewhat less than 1 per 1,000. In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well-controlled studies that progestogens are effective for these uses. If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine. It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters. Tablets are available in 0.3 mg, 0.625 mg, 0.9 mg, 1.25 mg, and 2.5 mg strengths of conjugated estrogens. Cream is available as 0.625 mg conjugated estrogens per gram.

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause. (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) Osteoporosis (abnormally low bone mass). Atrophic vaginitis. Kraurosis vulvae. Female castration.

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae.

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized. Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia. Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes. Whether this will provide protection from endometrial carcinoma has not been clearly established. There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens. (See PRECAUTIONS.) The choice of progestin and dosage may be important, product labeling should be reviewed to minimize possible adverse effects.

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions. 1. Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease. 2. Known or suspected estrogen-dependent neoplasia. 3. Known or suspected pregnancy (see Boxed Warning). 4. Undiagnosed abnormal genital bleeding. 5. Active thrombophlebitis or thromboembolic disorders. 6. A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).

WARNINGS: Estrogens have been reported to increase the risk of endometrial carcinoma (see Boxed Warning). However, a recent large, case-controlled study indicated no increase in risk of breast cancer in postmenopausal women. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement, it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction. Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users. An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives. If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization. Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use. They should be used with caution in patients with cerebral vascular or coronary artery disease. Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism, and thrombophlebitis. When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk.

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock. Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use. A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives. For this reason, diabetic patients should be carefully observed. Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases.

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed. Conditions influenced by fluid retention, such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc. Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed. Pre-existing uterine leiomyomata may increase in size during estrogen use. The pathologist should be advised of estrogen therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated. Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia or in young patients in whom bone growth is not yet complete. If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

The following changes may be expected with larger doses of estrogen:

a. Increased sulfobromophthalein retention.

b. Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3, increased norepinephrine-induced platelet aggregability.

c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T_4 by column, or T_4 by radioimmunoassay. Free T_3 resin uptake is decreased, reflecting the elevated TBG, free T_4 concentration is unaltered.

d. Impaired glucose tolerance.

e. Decreased pregnanediol excretion.

f. Reduced response to metyrapone test.

g. Reduced serum folate concentration.

h. Increased serum triglyceride and phospholipid concentration.

As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk.

Long-term, continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. However, in a recent, large case-controlled study of postmenopausal women there was no increase in risk of breast cancer with use of conjugated estrogens.

ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea; premenstrual-like syndrome, amenorrhea during and after treatment, increase in size of uterine fibromyomata, vaginal candidiasis, change in cervical erosion and in degree of cervical secretion; cystitis-like syndrome, tenderness, enlargement, secretion (of breasts), nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice, chloasma or melasma which may persist when drug is discontinued, erythema multiforme; erythema nodosum, hemorrhagic eruption; loss of scalp hair; hirsutism; steepening of corneal curvature, intolerance to contact lenses, headache, migraine, dizziness, mental depression, chorea, increase or decrease in weight, reduced carbohydrate tolerance, aggravation of porphyria; edema, changes in libido.

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females.

DOSAGE AND ADMINISTRATION:

PREMARIN® Brand of conjugated estrogens tablets, USP

1. *Given cyclically for short-term use only.* For treatment of moderate-to-severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0.3 mg to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible. Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals.

2. *Given cyclically.* Osteoporosis. Female castration. Osteoporosis—0.625 mg daily. Administration should be cyclic (eg, three weeks on and one week off). Female castration—1.25 mg daily, cyclically. Adjust upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level that will provide effective control.

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only. For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three- to six-month intervals.

Usual dosage range: 2 g to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

References:

1. Lindsay R, Hart DM, Clark DM: The minimum effective dose of estrogen for prevention of postmenopausal bone loss. *Obstet Gynecol* 1984;63:759-763. **2.** Studd JWW, Thom MH, Paterson MEL, et al: The prevention and treatment of endometrial pathology in postmenopausal women receiving exogenous estrogens, in Pasetto N, Paoletti R, Ambrus JL (eds): *The Menopause and Postmenopause.* Lancaster, England, MTP Press Ltd, 1980, chap 13.

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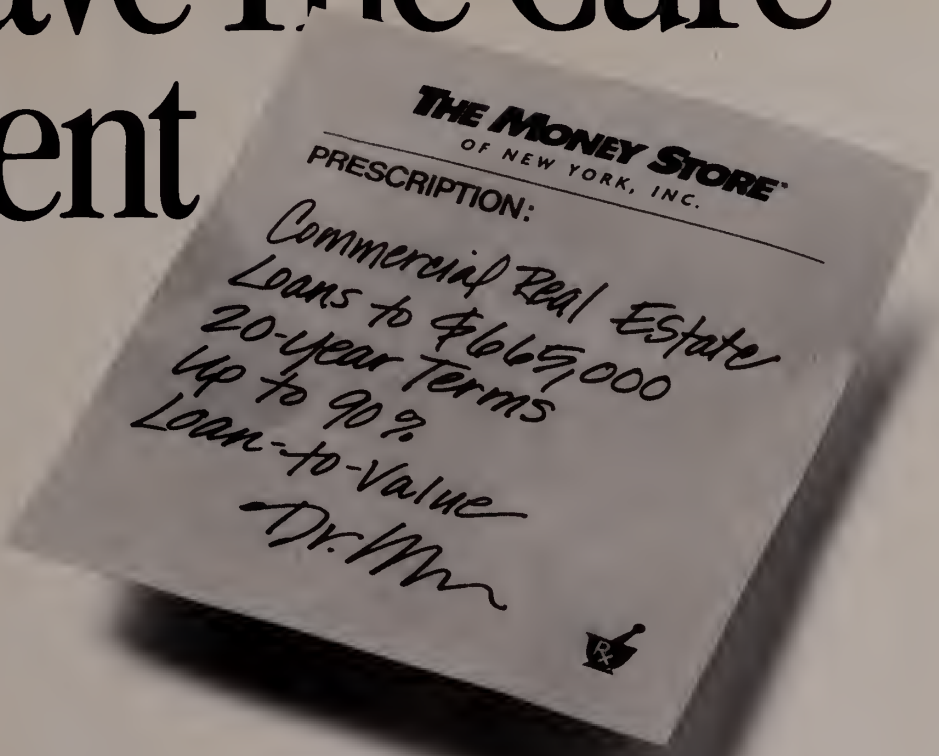

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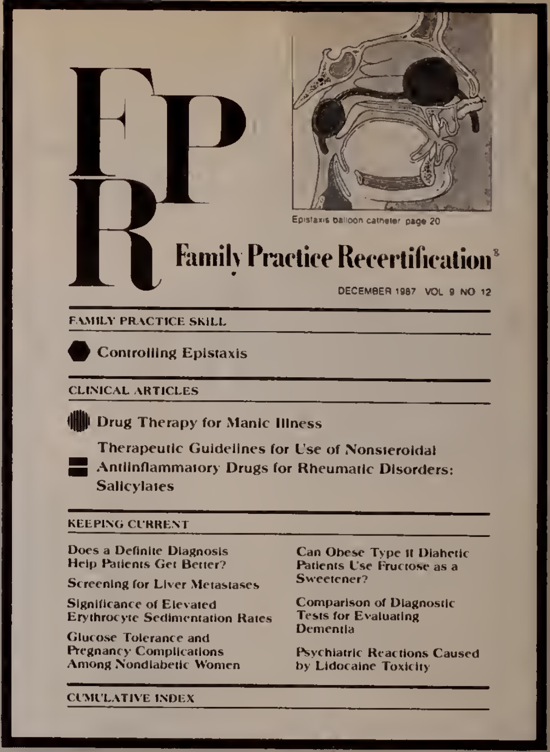

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Contraindications: There are no known contraindications to the use of 'Tagamet'.

Precautions: While a weak antiandrogenic effect has been demonstrated in animals, 'Tagamet' has been shown to have no effect on spermatogenesis, sperm count, motility, morphology or in vitro fertilizing capacity in humans.

In a 24-month toxicity study in rats at dose levels approximately 9 to 56 times the recommended human dose, benign Leydig cell tumors were seen. These were common in both the treated and control groups, and the incidence became significantly higher only in the aged rats receiving 'Tagamet'.

Rare instances of cardiac arrhythmias and hypotension have been reported following the rapid administration of 'Tagamet' HCl (brand of cimetidine hydrochloride) injection by intravenous bolus.

Symptomatic response to 'Tagamet' therapy does not preclude the presence of a gastric malignancy. There have been rare reports of transient healing of gastric ulcers despite subsequently documented malignancy.

Reversible confusional states have been reported on occasion, predominantly in severely ill patients.

'Tagamet' has been reported to reduce the hepatic metabolism of warfarin-type anticoagulants, phenytoin, propranolol, chlordiazepoxide, diazepam, lidocaine, theophylline and metronidazole. Clinically significant effects have been reported with the warfarin anticoagulants; therefore, close monitoring of prothrombin time is recommended, and adjustment of the anticoagulant dose may be necessary when 'Tagamet' is administered concomitantly. Interaction with phenytoin, lidocaine and theophylline has also been reported to produce adverse clinical effects.

However, a crossover study in healthy subjects receiving either 'Tagamet' 300 mg. q.i.d. or 800 mg. h.s. concomitantly with a 300 mg. b.i.d. dosage of theophylline (Theo-Dur®, Key Pharmaceuticals, Inc.), demonstrated less alteration in steady-state theophylline peak serum levels with the 800 mg. h.s. regimen, particularly in subjects aged 54 years and older. Data beyond ten days are not available. (Note: All patients receiving theophylline should be monitored appropriately, regardless of concomitant drug therapy.)

Lack of experience to date precludes recommending 'Tagamet' for use in pregnant patients, women of childbearing potential, nursing mothers or children under 16 unless anticipated benefits outweigh potential risks; generally, nursing should not be undertaken in patients taking the drug since cimetidine is secreted in human milk.

Adverse Reactions: Diarrhea, dizziness, somnolence, headache, rash. Reversible arthralgia, myalgia and exacerbation of joint symptoms in patients with preexisting arthritis have been reported. Reversible confusional states (e.g., mental confusion, agitation, psychosis, depression, anxiety, hallucinations, disorientation), predominantly in severely ill patients, have been reported. Gynecomastia and reversible impotence in patients with pathological hypersecretory disorders receiving 'Tagamet', particularly in high doses, for at least 12 months, have been reported. Reversible alopecia has been reported very rarely. Decreased white blood cell counts in 'Tagamet'-treated patients (approximately 1 per 100,000 patients), including agranulocytosis (approximately 3 per million patients), have been reported, including a few reports of recurrence on rechallenge. Most of these reports were in patients who had serious concomitant illnesses and received drugs and/or treatment known to produce neutropenia. Thrombocytopenia (approximately 3 per million patients) and a few cases of aplastic anemia have also been reported. Increased serum transaminase and creatinine, as well as rare cases of fever, interstitial nephritis, urinary retention, pancreatitis and allergic reactions, including hypersensitivity vasculitis, have been reported. Reversible adverse hepatic effects, cholestatic or mixed cholestatic-hepatocellular in nature, have been reported rarely. Because of the predominance of cholestatic features, severe parenchymal injury is considered highly unlikely. A single case of biopsy-proven periportal hepatic fibrosis in a patient receiving 'Tagamet' has been reported.

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Exposure of the premixed product to excessive heat should be avoided. It is recommended the product be stored at controlled room temperature. Brief exposure up to 40°C does not adversely affect the premixed product.

'Tagamet' HCl (brand of cimetidine hydrochloride) injection premixed in single-dose plastic containers is manufactured for SK&F Lab Co. by Travenol Laboratories, Inc., Deerfield, IL 60015.

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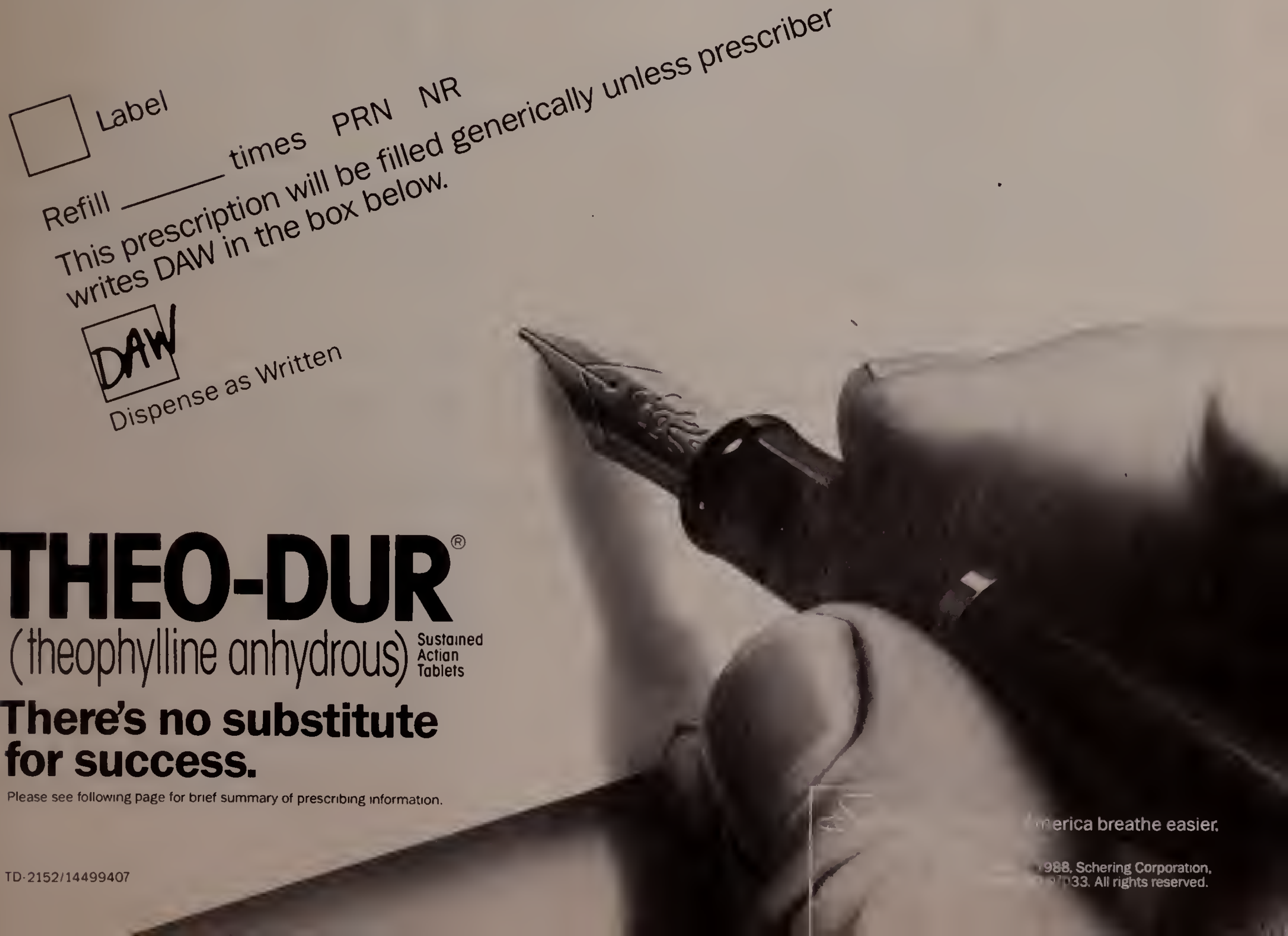

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Please see following page for brief summary of prescribing information.

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THEO-DUR®

THEOPHYLLINE (Anhydrous)
Sustained Action Tablets

INDICATIONS: THEO-DUR is indicated for relief and/or prevention of symptoms of asthma and for reversible bronchospasm associated with chronic bronchitis and emphysema

CONTRAINDICATIONS: THEO-DUR is contraindicated in individuals who have shown hypersensitivity to theophylline or any of the tablet components

WARNINGS: Status asthmaticus should be considered a medical emergency and is defined as that degree of bronchospasm which is not rapidly responsive to usual doses of conventional bronchodilators Optimal therapy for such patients frequently requires both *additional medication*, parenterally administered, and *close monitoring*, preferably in an intensive care setting

Although increasing the dose of theophylline may bring about relief, such treatment may be associated with toxicity The likelihood of such toxicity developing increases significantly when the serum theophylline concentration exceeds 20 mcg/ml Therefore, determination of serum theophylline levels is recommended to assure maximal benefit without excessive risk

Serum levels above 20 mcg/ml are rarely found after appropriate administration of recommended doses. However, in individuals in whom theophylline plasma clearance is reduced *for any reason*, even conventional doses may result in increased serum levels and potential toxicity Reduced theophylline clearance has been documented in the following readily identifiable groups. 1) patients with impaired renal or liver function, 2) patients over 55 years of age, particularly males and those with chronic lung disease, 3) those with cardiac failure from any cause, 4) neonates, and 5) those patients taking certain drugs (macrolide antibiotics and cimetidine) Decreased clearance of theophylline may be associated with either influenza immunization or active infection with influenza

Reduction of dosage and laboratory monitoring is especially appropriate in the above individuals Less serious signs of theophylline toxicity (i e nausea and restlessness) may occur frequently when initiating therapy, but are usually transient, when such signs are persistent during maintenance therapy, they are often associated with serum concentrations above 20 mcg/ml Unfortunately, however, serious side effects such as ventricular arrhythmias, convulsions or even death may appear as the first sign of toxicity without any previous warning Stated differently *serious toxicity is not reliably preceded by less severe side effects*

Many patients who require theophylline may exhibit tachycardia due to their underlying disease process so that the cause/effect relationship to elevated serum theophylline concentrations may not be appreciated.

Theophylline products may cause dysrhythmia and/or worsen pre-existing arrhythmias and any significant change in rate and/or rhythm warrants monitoring and further investigation.

The occurrence of arrhythmias and sudden death (with histological evidence of necrosis of the myocardium) has been recorded in laboratory animals (minipigs, rodents and dogs) when theophylline and beta agonists were administered concomitantly, although not when either was administered alone. The significance of these findings when applied to human usage is currently unknown

PRECAUTIONS: THEO-DUR TABLETS SHOULD NOT BE CHEWED OR CRUSHED

General: Theophylline half-life is shorter in smokers than in non-smokers Therefore, smokers may require larger or more frequent doses Morphine and curare should be used with caution in patients with airway obstruction as they may suppress respiration and stimulate histamine release. Alternative drugs should be used when possible Theophylline should not be administered concurrently with other xanthine medications. Use with caution in patients with severe cardiac disease, severe hypoxemia, hypertension, hyperthyroidism, acute myocardial injury, cor pulmonale, congestive heart failure, liver disease, in the elderly (especially males) and in neonates. In particular, great caution should be used in giving theophylline to patients with congestive heart failure Frequently, such patients have markedly prolonged theophylline serum levels with theophylline persisting in serum for long periods following discontinuation of the drug Individuals who are rapid metabolizers of theophylline, such as the young, smokers, and some non-smoking adults, may not be suitable candidates for once-daily dosing These individuals will generally need to be dosed at 12 hour or sometimes 8 hour intervals Such patients may exhibit symptoms of bronchospasm near the end of a dosing interval, or may have wider peak-to-trough differences than desired

Use theophylline cautiously in patients with history of peptic ulcer Theophylline may occasionally act as a local irritant to the G I tract although gastrointestinal symptoms are more commonly centrally mediated and associated with serum drug concentrations over 20 mcg/ml

Information for Patients: The physician should reinforce the importance of taking only the prescribed dose and time interval between doses THEO-DUR tablets should not be chewed or crushed. When dosing THEO-DUR on a once daily (q24h) basis, tablets should be taken whole and not split As with any controlled-release theophylline product, the patient should alert the physician if symptoms occur repeatedly, especially near the end of the dosing interval

DRUG INTERACTIONS: **Drug-Drug**: Toxic synergism with ephedrine has been documented and may occur with some other sympathomimetic bronchodilators In addition, the following drug interactions have been demonstrated

Drug	**Effect**
Theophylline with lithium carbonate	Increased excretion of lithium carbonate
Theophylline with propranolol	Antagonism of propranolol effect
Theophylline with cimetidine	Increased theophylline blood levels
Theophylline with troleandomycin, erythromycin	Increased theophylline blood levels

Drug-Food: *THEO-DUR 100 mg Sustained Action Tablets* have not been adequately studied to determine whether their bioavailability is altered when given with food Available data suggest that drug administration at the time of food ingestion may influence the absorption characteristics of theophylline controlled-release products resulting in serum values different from those found after administration in the fasting state.

A drug-food effect, if any, would likely have its greatest clinical significance when high theophylline serum levels are being maintained and/or when large single doses (greater than 13 mg/kg or 900 mg) of a controlled-release theophylline product are given

THEO-DUR (200, 300, and 450 mg) Sustained Action Tablets: The rate and extent of absorption of theophylline from THEO-DUR 200 mg 300 mg, and 450 mg tablets when administered fasting or immediately after a moderately high fat content breakfast is similar

Drug-Laboratory Test Interactions: When plasma levels of theophylline are measured by spectrophotometric methods, coffee, tea, cola beverages, chocolate, and acetaminophen contribute falsely high values

Carcinogenesis, Mutagenesis, and Impairment of Fertility: Long-term animal studies have not been performed to evaluate the carcinogenic potential, mutagenic potential, or the effect on fertility of xanthine compounds.

Pregnancy: Category C—Animal reproduction studies have not been conducted with theophylline It is not known whether theophylline can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Xanthines should be given to a pregnant woman only if clearly needed

Nursing Mothers: It has been reported that theophylline distributes readily into breast milk and may cause adverse effects in the infant Caution must be used if prescribing xanthine to a mother who is nursing, taking into account the risk-benefit of this therapy

Pediatric Use: Safety and effectiveness of THEO-DUR administered
1 Every 24 hours in children under 12 years of age, have not been established
2 Every 12 hours in children under 6 years of age, have not been established

ADVERSE REACTIONS: The most consistent adverse reactions are usually due to overdose and are
1 *Gastrointestinal* nausea, vomiting, epigastric pain, hematemesis, diarrhea
2 *Central nervous system* headaches, irritability, restlessness, insomnia, reflex hyperexcitability, muscle twitching clonic and tonic generalized convulsions
3 *Cardiovascular* palpitation, tachycardia, extrasystoles, flushing hypotension, circulatory failure, ventricular arrhythmias
4 *Respiratory* tachypnea
5 *Renal* albuminuria, increased excretion of renal tubular and red blood cells, potentiation of diuresis
6 *Other* rash hyperglycemia and inappropriate ADH syndrome

OVERDOSAGE. **Management**: If potential oral overdose is established and seizure has not occurred
A Induce vomiting
B Administer a cathartic (this is particularly important if sustained-release preparations have been taken)
C Administer activated charcoal
If patient is having a seizure
A Establish an airway
B Administer oxygen
C Treat the seizure with intravenous diazepam, 0 1 to 0 3 mg/kg up to 10 mg
D Monitor vital signs, maintain blood pressure and provide adequate hydration

Post Seizure Coma:
A Maintain airway and oxygenation
B If a result of oral medication follow above recommendations to prevent absorption of the drug, but intubation and lavage will have to be performed instead of inducing emesis, and the cathartic and charcoal will need to be introduced via a large bore gastric lavage tube
C Continue to provide full supportive care and adequate hydration while waiting for drug to be metabolized In general the drug is metabolized sufficiently rapid so as not to warrant consideration of dialysis however, if serum levels exceed 50 mcg/ml charcoal hemoperfusion may be indicated

CAUTION Federal law prohibits dispensing without prescription For full prescribing information, see package insert

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MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

JULY 1988

AROUND THE STATE

ROCHESTER

July 15–17. **Mammography and the Search for Breast Cancer.** 20 Cat 1 Credits. Rochester. Contact: Theresa Wade, 1351 Mt Hope Ave, Rochester, NY 14620. Tel: (716) 442-8432.

SARATOGA SPRINGS

July 7–8. **Advances in Spinal Cord Injury Rehabilitation.** Ramada Renaissance/Saratoga Springs. Contact: Barbara Post, Program Coordinator, Regional Medical Education Program, Sunnyview Hospital and Rehabilitation Center, 1270 Belmont Ave, Schenectady, NY 12308. Tel: (518) 382-4500.

AROUND THE NATION

ALASKA

July 6–13. **Breast Imaging and Ultrasound.** 18 Cat 1 Credits. Cruise the Inland Passage. Contact: Alaska 88, c/o Medical Seminars International, Inc, 21915 Roscoe Blvd, Suite 222, Canoga Park, CA 91304. Tel: (818) 719-7380.

CALIFORNIA

July 15–16. **AIDS/ARC Update 1988.** Civic Auditorium, San Francisco. Contact: Extended Programs in Medical Education, University of California, Room U-569, San Francisco, CA 94113-0742. Tel: (415) 476-4251.

July 28–31. **The Second Annual Symposium on Magnetic Resonance Imaging.** 26 Cat 1 Credits. Ritz-Carleton Resort Hotel, Laguna Niguel. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

(*continued on p 24A*)

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AXID® 300mg

nizatidine

Effective once-nightly
duodenal ulcer therapy available in a

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for better patient compliance

AXID®
nizatidine capsules

Brief Summary. Consult the package insert for prescribing information.

Indications and Usage: Axid is indicated for up to eight weeks for the treatment of active duodenal ulcer. In most patients, the ulcer will heal within four weeks

Axid is indicated for maintenance therapy for duodenal ulcer patients, at a reduced dosage of 150 mg h s after healing of an active duodenal ulcer The consequences of continuous therapy with Axid for longer than one year are not known

Contraindication: Axid is contraindicated in patients with known hypersensitivity to the drug and should be used with caution in patients with hypersensitivity to other H_2-receptor antagonists

Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy

2 Because nizatidine is excreted primarily by the kidney, dosage should be reduced in patients with moderate to severe renal insufficiency

3 Pharmacokinetic studies in patients with hepatorenal syndrome have not been done Part of the dose of nizatidine is metabolized in the liver In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects

Laboratory Tests—False-positive tests for urobilinogen with Multistix® may occur during therapy with nizatidine

Drug Interactions—No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system, therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when nizatidine, 150 mg b i d , was administered concurrently

Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect There was a dose related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high dose males compared to placebo Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups The rate of hepatic carcinoma in the high dose animals was within the historical control limits seen for the strain of mice used The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement compared to concurrent controls, and evidence of mild liver injury (transaminase elevations) The occurrence of a marginal finding at high dose only in animals given an excessive, and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery is not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, and the mouse lymphoma assay

In a two-generation, perinatal and postnatal, fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose, and in Dutch Belted rabbits at doses up to 55 times the human dose, revealed no evidence of impaired fertility or teratogenic effect, but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers—Nizatidine is secreted and concentrated in the milk of lactating rats Pups reared by treated lactating rats had depressed growth rates Although no studies have been conducted in lactating women, nizatidine is assumed to be secreted in human milk, and caution should be exercised when nizatidine is administered to nursing mothers

Pediatric Use—Safety and effectiveness in children have not been established

Use in Elderly Patients—Ulcer healing rates in elderly patients are similar to those in younger age groups The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations. Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo. Among the more common adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs <0 01%), and somnolence (2 4% vs 1 3%) were significantly more common in the nizatidine group A variety of less common events was also reported, it was not possible to determine whether these were caused by nizatidine.

Axid® (nizatidine, Lilly)

Hepatic—Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients possibly or probably related to nizatidine. In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L), and in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid. Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo. Rare reports of gynecomastia occurred.

Hematologic—Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist On previous occasions, this patient had experienced thrombocytopenia while taking other drugs

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine than in placebo patients. Rash and exfoliative dermatitis were also reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported.

Overdosage: There is little clinical experience with overdosage of Axid in humans. If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy Renal dialysis for four to six hours increased plasma clearance by approximately 84%

Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal. Intravenous LD_{50} values in the rat and mouse were 301 mg/kg and 232 mg/kg respectively. PV 2091 AMP [041288]

Axid® (nizatidine, Lilly)

Lilly

Eli Lilly and Company
Indianapolis, Indiana
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COMMENTARIES

The use of adrenal medullary and fetal grafts as a treatment for Parkinson disease

The idea of implanting tissue into the brain to treat neurologic disease is one that continues to elicit disbelief and wonder. The first autologous adrenal medullary to caudate nucleus implant in a patient with Parkinson disease (PD) was performed in Sweden six years ago, but resulted in only limited success.[1,2] The procedure was substantially modified in Mexico with better results.[3,4] This procedure may have opened a new era in the treatment of PD, but it is clear that there are many problems associated with the operation. Fewer than half of the patients who undergo the procedure benefit; in many of these patients the benefits are only modest. Perioperative morbidity is high, and the long term results are unknown. Nonetheless, some patients in whom all other treatments have failed derive substantial benefits. The task is to make the surgery uniformly successful and to reduce its risks.

One factor that is undergoing scrutiny is the adrenal medulla. Experiments with animals indicate that adrenal medullary tissue survives poorly in the brain.[5,6] While some question whether the adrenal medullary tissue has to survive to be effective,[6] most investigators assume survival is necessary. In three autopsies of patients who unsuccessfully underwent adrenal medullary transplants, only one demonstrated a few surviving adrenal cells (H. Hurtig, personal communication, December 1987). Thus, while it is not known if patients who have undergone successful implants have surviving adrenal cells, it has been shown that patients with unsuccessful implants do not have surviving adrenal cells.

The reason that neural transplantation centers on PD is that, among the neurodegenerative disorders, there is in PD the clearest relationship between pathology (loss of substantia nigra neurons), neurochemistry (loss of striatal dopamine), pathophysiology, and clinical symptoms.[7] Moreover, the tissue loss in PD is small and restricted, unlike the more widespread tissue loss in Alzheimer disease. It was thus reasonable to expect that if a transplant could substitute for lost tissue, the chances of a favorable outcome would be best in a disease in which only a small volume of transplanted tissue is required.

Studies using animal models of PD indicate that fetal nigral cells can survive and reverse parkinsonian symptoms when they are transplanted into the striata of these animals.[8–11] The fetal grafts reverse the animals' symptoms through several mechanisms including restoring striatal dopamine neurotransmission, releasing trophic factors, or becoming integrated into the host's neural circuitry.[8–11]

Because transplanting human fetal tissues raises complex ethical issues, investigators have turned to other sources of tissue which might substitute for the fetal nigral cells. One such source is the adrenal medulla, which is embryologically related to the substantia nigra. While some of the disadvantages of using adrenal medullary grafts have been enumerated, using a patient's own tissue avoids the problems of host/graft rejection and the necessity of using immunosuppressive drugs, and bypasses the ethical issues of fetal transplantation. However, in taking the patient's own adrenal medulla, the patient is subjected to two simultaneous operations: a laparotomy and a craniotomy. It is now evident that much of the morbidity associated with the procedure results from doing a laparotomy on a chronically debilitated patient. Moreover, there is now some question as to whether the adrenal medulla is affected in PD[2]—PD may be a disorder of all catecholamine synthesizing tissue including tissue outside the central nervous system.[12]

With these questions in mind, investigators have again turned to the possibility of using fetal tissue. At this time, fetal implants have been performed in the People's Republic of China, Mexico, and Sweden, in that order. The details and results of the operation in China are not known. The details of the surgery in Mexico are as follows.

On September 12, 1987, a 31-year-old woman with a history of repeated abortions due to cervical uterine incompetence had a spontaneous abortion and delivered a 13-week old fetus (calculated from the date of conception). After fetal death was certified by two physicians who were not part of the transplantation team, written consent for cadaver organ transplantation was obtained from the woman and her husband. Two patients with PD had been hospitalized until fetal tissue became available and were now started on cyclosporine, and were operated on within two hours of fetal death. A female patient with PD received both fetal adrenal medullae. A male patient with PD received the fetal substantia nigra. The man was chosen for the nigral implantation because he had disease that was worse on one side, and it was not known whether the nigral implant would have a bilateral effect, like the adrenal implant.

Prior to performing surgery, approval was obtained from the ethics and research committees of the hospital and written consent was obtained from the patients and their relatives. In both patients, the grafted fetal tissue was placed within a cavity of the right caudate nucleus in contact with the cerebrospinal fluid.

On September 12, 1987, and again on January 15, 1988, I visited the Hospital de Especialidades Centro Medico la Raza in Mexico City. I examined the two patients who had undergone the fetal transplants. The first patient was a 35-year-old woman with a four-year history of PD who was becoming increasingly disabled despite medication. She was on Sinemet,^{TM} 750 mg levodopa/75 mg carbidopa. When first examined by me six days after receiving a fetal adrenal medullary implant to the caudate she had been off the medication for six days. She had difficulty cutting her food, dressing herself, turning in bed, and maintaining personal hygiene. She had a sustention tremor of both hands, and rigidity of her neck and both lower extremities. She could not arise from a chair without assistance. She had difficulty walking. On the Unified Parkinson Rating Scale (UPRS), in which "0" represents no disability and 160 indicates complete disability, she scored 39. Before surgery she had scored 71 points. It is noteworthy that she, like many patients undergoing autologous adrenal medullary implants, improved transiently within the first two weeks of surgery. This improvement then disappears and may be followed later by a more sustained improvement.

The second patient was a 50-year-old man with a ten-year history of PD, which was much worse on his left side. He was on Sinemet, 1,000 mg of levodopa/100 mg carbidopa. He was examined six days after an implant of fetal nigra to the right caudate nucleus. Off all medication his speech was impaired and he could not write, feed himself, dress himself, or maintain personal hygiene. He could barely turn in bed. He had a resting tremor of both hands that was much more prominent on the left. He had marked rigidity of the neck and of both upper and lower extremities. He could not arise from a chair unaided. His gait was impaired, and he had postural instability. On the UPRS he scored 90. He was "off" all of the time. Before surgery he suffered from "on/off" phenomena and scored 59 points when "on" and 90 points when "off." At the time of my examination in September the man's condition was worse than the woman's. When she was reexamined by me on January 15, 1988, the woman's handwriting was better, and she had no difficulty in cutting her food, dressing herself, turning in bed, or maintaining personal hygiene. There was a mild sustention tremor of the right hand. There was minimal rigidity of the neck and of both lower extremities. She had minimal difficulty in arising from a chair and no gait difficulty. Her score on the UPRS was 29. She was on 250 mg levodopa, 25 mg carbidopa, and cyclosporine, 1 mg/kg. The man had improved much more than the woman. His speech was normal, he had only minimal difficulty in writing and no difficulty in cutting his food, dressing himself, turning in bed, or performing tasks of personal hygiene. There was a mild resting tremor of both hands, on the left more than on the right. There was minimal rigidity of the neck and of the lower extremities. Finger and hand movements were only minimally slow. He had no difficulty arising from a chair and no difficulty walking. He was "on" all the time. On the UPRS he scored 33. He was on 375 mg levodopa, 37.5 mg carbidopa, and cyclosporine, 1 mg/kg.

Obviously extreme caution must be exercised in commenting on these two patients, but it appears that the fetal nigral implant is more effective than the fetal adrenal one, corroborating the data from animal studies.[8-11] It is surprising that the nigral implant had a bilateral effect. This suggests that in addition to being integrated into the hosts' neural circuitry (the presumed mechanism of restoring function), the nigral graft like the adrenal graft may have a trophic influence as well.

In November and again in December 1987, fetal nigral implants were performed in Sweden (A. Bjorklund, personal communication, January 1988). The operations in Sweden differed from the one in Mexico in that in Sweden, suspensions of nigral cells from embryos less than eight weeks old were injected stereotaxically into one site in the caudate and two sites in the putamen. It is too early to tell if these grafts were successful. Ideally, the fetal tissue should be less than 12 weeks old (from the time of conception), as in the Swedish operations, because after this age in humans there is little neuron replication. In Sweden, women undergoing abortions are asked if they wish to donate the tissue for medical research. The women are told that there is a chance that the tissue will not be used. This is done to prevent women from conceiving solely for the purpose of donating tissue.

Transplants of adrenal medullary to caudate and fetal transplants may have opened a new era. The transplants are forcing neurologists to rethink their ideas about how the brain is organized. Patients with PD improve bilaterally after a unilateral implant. Does this mean that each side of the brain is controlled in part by the other side, or that the grafts secrete trophic factors that diffuse past the midline and stimulate the opposite side? Transplants will force neurologists to play a more active role in managing patients with neurodegenerative diseases. Heretofore, neurologists were passive observers of these disorders, with the exception of PD. But in the future, neurologists may have to select those patients and those disorders that may be amenable to transplantation.

At this time, the adrenal medullary to caudate and the fetal transplants must be viewed for what they are: investigational treatments. The current operations are prototypes that will have to be refined and perfected so that their results become uniform with few complications. While the present grafting techniques are crude, in the future procedures may be developed that will allow purified, enriched, or genetically engineered cells to be used, with the cells producing the specific trophic factors that are necessary for recovery. The use of fetal tissue to treat PD raises great hopes and causes much uneasiness. On one hand, there are potentials for reversing debilitating diseases such as Parkinson disease and conceivably other degenerative disorders. On the other hand, there are visions of the elderly renewing themselves at the expense of the young. Until now the debate over the ethics of fetal transplantation has been a theoretical one. However, the recent reports from China, Mexico, and Sweden now bring this debate into the real world.

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Visual complaints and video display terminals

Due to the widespread use of video display terminals (VDTs) and the frequency of worker complaints of visual difficulties, there naturally arises a concern about potential adverse ocular health effects from the use of VDTs.[1-4] These concerns are of particular interest to ophthalmologists, optometrists, preventive health specialists, occupational medicine physicians, and legislators. Some states and counties have enacted or are considering laws to establish machine performance and ergonomic standards for VDTs.[5] Suffolk County, NY, has mandated that the state pay for eye examinations for all state workers using VDTs.[6] While ocular complaints have provided much of the impetus for such legislation, there is no conclusive scientific evidence indicating a direct relationship between VDTs and ocular disease.[7-9] The question then arises as to why there are so many visual complaints by VDT users.

Visual difficulties associated with VDTs include asthenopic complaints such as eye strain, irritation, blurred vision, headaches, transient color vision problems, and discomfort.[10,11] Identical visual complaints commonly occur after prolonged intensive visual activities and many tasks that do not involve VDTs.[12,13] These non-VDT-associated symptoms are sometimes attributed to temporary changes of visual function including decreased accommodation. Accommodative symptoms are especially prominent in presbyopic patients. These patients have decreased ability to focus up close due to loss of elasticity of their lens. This normally occurs in people in their mid-40s and is readily correctable with glasses. Frequently, patients with conversion insufficiency or poor eye coordination also have difficulties with near vision. This can be remedied by exercises, glasses, or occasionally with muscle surgery. Patients with poor visual acuity due to pathologic conditions also have significant complaints of eye strain. Standards for image quality in VDTs eliminate the screen as a cause of blurred vision. Images should not flicker, and the entire display should be in sharp focus. Functional accommodative range is increased when the size of the pupil is decreased. This is best achieved by proper lighting. Blurring due to eye strain usually resolves within half an hour of resting the eyes and can be alleviated by looking in the distance periodically to relax the focusing mechanism.

Unique to VDTs are several ergonomically related visual effects.[14,15] The viewing distance from the user to the VDT is generally 22 to 26 inches, which is in the intermediate visual range. Presbyopic patients who require reading prescriptions usually focus at a closer range and may experience blurred vision at an intermediate range. In addition, other printed reference material may not be in an appropriate viewing position. To minimize strain on the eyes, work should be placed at the same distance as the screen with lighting of equal intensity. These problems are easily solved with reading glasses, bifocal lenses or, occasionally, trifocal lenses. These must be adjusted for the proper height of the terminal, which should be just below eye level.

Appropriate lighting will minimize visual difficulties in the use of VDTs. In general, background light levels should not be too high. Visual contrast should be maximized between the print and the background color on the screen, and there should be no bright sources of direct or reflected light in the area of the screen. Glare is generally produced by reflection off the screen from overhead lighting, reflections off work surfaces, or excessive background light such as from a window behind the VDT. These factors can be evaluated by turning the VDT screen off and observing any bright reflections. The reflection should be minimized by utilizing an indirect lighting system, downward directed light, or removing or shielding the glare sources. In some cases, it may be easier to install a screen hood on the VDT or to use a glare filter. Sunglasses generally reduce contrast on the screen.

VDTs are excellent dust attracters. This may be due to an electrostatic field in the area of the VDT causing deposition of particles on the screen and the operator. These particles have been thought to be the cause of occasional facial dermatitis.[16-18] They may also be the cause of ocular irritation, making the eyes tear, feel gritty, or turn red. Proper humidity and ventilation in the work place or the use of artificial tears may relieve these complaints.

Transient color vision abnormalities have been reported by multiple authors in patients using green monochromatic display terminals.[19-21] First discovered in 1965, the McCullough effect appears to be a harmless, induced, prolonged, complementary chromotopsia probably related to after-images. Light letters on contrasting background have an apparent pink color. AOHRR pseudo-isochromatic testing shows error on plate three and occasionally plate five. The Farnsworth-Munsell D15 tests have been normal. The phenomenon only occurs in one eye if the other eye is occluded and may last as long as several weeks. Chloropsia can occur after using a monochrome red VDT but will not occur with trichromatic displays. VDTs flicker extremely rapidly so that screen characters appear solid. Lights flickering well below VDT rates have induced epileptic seizures in photosensitive individuals. There have been no reports of VDT-induced seizures, and there is no scientific evidence that those suffering from epilepsy should not work with VDTs.[9,22]

The testing of VDTs has documented that they produce little or no harmful ionizing radiation such as x-rays and less non-ionizing ultraviolet radiation than is produced by fluorescent lighting.[23-27] These levels of radiation are well below acceptable standards of exposure. Anecdotal reports of cataract formation in VDT users have naturally prompted concern. Some of the patients had minor lens opacities that did not alter vision or had exposure to other cataractogens.[28] A study by the National Institute for Occupational Safety and Health indicated no increased incidence of cataracts among VDT users than among nonusers.[29]

Mandatory ocular screening examination of VDT users has been proposed by the Suffolk County Legislature. The American Academy of Ophthalmology recommends routine ocular screening examinations every three years, and certainly any ocular complaints should be addressed. On the other hand, the American Academy of Ophthalmology and the New York State Ophthalmologic Society have taken the position that VDTs pose no ocular hazards. They have stated their opinion that reported ocular problems are not due to the VDT itself, but rather stem from poor ergonomics and the common problems associated with near visual tasks.[7] While screening examinations will detect previously undiagnosed ocular systemic disorders, they will not deal with ergonomic problems which may be better addressed through educational programs or appropriate legislation.

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Carcinoma of the male breast

Carcinoma of the male breast is a relatively rare entity which many physicians may never see in a lifetime of medical practice. The disease represents only about 1% of mammary cancers and therefore has not been studied as extensively as carcinoma of the female breast.[1] Wainwright[2] is generally credited with the first report in the literature of male breast cancer, which appeared in 1927 and proclaimed the lethality of the disease. Despite the popularity of the Wainwright series, recognition for the first report of male breast carcinoma should actually be given to the 14th century British physician John of Arderne.[3] He noted an enlarging mass beneath the right nipple in a priest of Colstone. A local barber-surgeon promised cure, but John would "let no cutting come therenigh."

Unfortunately, carcinoma of the male breast is encountered so infrequently that the experiences of many authors are typically combined in order to define the natural history and results of treatment. The report by Lefor and Numann[4] in this issue of the *Journal* is refreshing in this regard. These investigators were able to document 32 cases of male breast cancer within a specific geographic area over a 27-year period. Pathologic confirmation was achieved in each instance, and follow-up was excellent.

These authors and others have extracted some fundamental features of male breast cancer from their review. These findings may be summarized as follows: Male breast cancer usually presents as an asymptomatic breast mass, and specific diagnosis is frequently delayed. Infiltrating duct carcinoma is the most common histologic cell type, and estrogen receptors are frequently present. Survival after surgical therapy is excellent for early stage lesions; however, once the tumor has reached the axillary nodes, the results of treatment are dismal.

One important feature of male breast masses relates to the frequency of malignancy. In contrast to the female breast, most masses arising in the male breast are malignant.[1] Benign tumors such as fibroadenomas are extremely rare. Clinical assessment of the axilla is inaccurate, and one must rely on axillary dissection and microscopic node evaluation for staging.[5] Histopathologic findings in male breast cancer differ from those of female breast cancer, with an absence of the lobular cell type in the male disease. This feature is presumably a result of the absence of lobules in the male breast.

Traditionally, the survival of males with breast cancer has been thought to be lower than that of females with the disease. This belief has been promulgated by authors who have used crude survival statistics that do not take into account the fact that men with breast cancer tend to present at an older age than females.[6] It is true that the interval between the onset of symptoms and definitive therapy may be longer in men than women, and more men may present with later stage disease. However, survival data within each stage are not significantly different between the sexes.[5]

The relationship of gynecomastia to mammary cancer is not clear. Although cancer of the male breast rarely supervenes on gynecomastia,[7,8] male breasts with carcinoma often demonstrate an unusual amount of stroma and benign ducts. Reports of men developing mammary cancer after prolonged estrogen therapy are not frequent. Carcinoma of the breast that occurs while undergoing estrogen therapy for prostatic cancer is infrequent and is thought to represent metastatic prostatic carcinoma rather than primary breast cancer.[9] The incidence of mammary cancer is increased in male patients with Klinefelter syndrome,[10] but an estrogen-gynecomastia-carcinoma relationship in male patients has yet to be established.

Therapeutic intervention in male breast cancer has been based on empiric suppositions, since the disease is rare enough to make randomized trials impractical. However, several tenets appear to be rational and worthy of consideration. First, the small size of the male breast interdicts any procedure less than complete removal of all breast tissue. Second, radical mastectomy may be required more frequently than in female breast cancer because of the exiguous nature of the male breast tissue and contiguous involvement of the pectoralis major muscle with the malignant process. Third, precise staging with axillary dissection is indicated in the absence of obvious widespread disease, so that adjuvant chemotherapy may be implemented in an effort to achieve the results observed with cancer of the female breast. Finally, the responsiveness of advanced male breast cancer to systemic chemotherapy and directed radiotherapy may be the same as that reported for female breast cancer, and these treatment modalities should be implemented when advanced disease is encountered.[11]

In conclusion, the male breast is an easy organ to examine, and any discrete masses should be assumed to be malignant until proven otherwise. Unfortunately, carcinoma of the male breast is such an infrequently encountered tumor that unfamiliarity with the disease can lead to delays in diagnosis. An increased awareness of the disease may be expected to result in earlier detection and institution of appropriate therapy at a stage when cure may be possible. The article by Lefor and Numann[4] in this issue of the *Journal* represents an important addition to the literature in this regard. Although few new features have been elucidated by the study, it certainly serves to increase awareness of this rare disease entity. In this manner it may be adumbrated that the data of future studies will permanently entomb the pessimistic contentions of Wainwright, as the results of treatment of male breast cancer approach those of its female counterpart.

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Carcinoma of the breast in men

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ABSTRACT. Cancer of the breast in men is uncommon. We retrospectively reviewed 32 cases of male breast cancer. The patients presented at an average age of 66 years with an average delay in diagnosis after appearance of symptoms of 14 months. The tumors were predominantly left sided, usually presenting as a subareolar mass, and most were infiltrating duct cell tumors. Primary therapy remains surgical, reserving limited operation for those with advanced disease. Five-year survival correlated well with stage of disease. Stage I had an associated 88% five-year survival rate, stage II had a 54% survival rate, and only 20% of patients with stage III disease were alive after five years. These survival rates are higher than those reported in earlier studies, and are comparable to survival rates seen in female breast cancer of similar stage. An increased public awareness leading to earlier detection and treatment should further improve survival rates.

(NY State J Med 1988; 88:293–296)

Cancer of the breast in men is an uncommon entity. While carcinoma of the breast accounts for 23% of all malignancies among women, it represents only 0.7% of malignant tumors in men.[1] Approximately 1% of all cases of human breast cancer occur in men. There are an estimated 600 new cases each year in the United States, and 250 men die of the disease annually.[2] The public is generally unaware that malignancy occurs in the male breast. While it is generally held that breast cancer in men has a poorer prognosis than in women, several recent studies[3–9] have shown survival rates similar to those of female breast cancer. In this study we reviewed 32 cases of carcinoma of the male breast to identify the clinical features of the disease and correlate these with survival.

PATIENTS AND METHODS

Between 1956 and 1983, 32 cases of carcinoma of the breast in men were identified from the Syracuse Cancer Registry or records of Syracuse area hospitals in New York State. The Syracuse Cancer Registry was in operation from 1956 through 1975 and provided the data for the earliest available cases. These 32 cases were retrospectively reviewed. Only abbreviated medical records were available for 12 patients in this group. Pathology reports were reviewed for all patients.

Follow-up data were obtained from outpatient clinic charts and from contact with individual physicians. Survival data were evaluated using the life-table method. Patients who died of causes unrelated to the breast malignancy were excluded from analysis at the time of their last evaluation.

RESULTS

The average age of patients in the series at the time of diagnosis was 66.5 years, with a range from 40 to 82.5 years (standard deviation, 10 years). Thirty of the 32 patients were white, and two were black. Presenting symptoms included nipple discharge in one patient and a breast mass in the remaining 31 patients (97%). One patient underwent needle biopsy of a mass in the evaluation of ipsilateral tuberculosis of the breast. The duration of symptoms ranged from the time of initial presentation to as long as ten years (mean, 14 months) before medical attention was sought. One patient had a strong family history of breast cancer in first-degree female relatives. Family history was specifically mentioned as negative for breast cancer in 18 cases.

The tumors were left sided in 20 patients (62%) and right sided in 12 patients (38%). This difference is not statistically significant by chi-square analysis ($p > 0.05$). No patient in this series had bilateral disease. One patient had ulceration of the skin over the lesion, two patients had induration, and three patients had nipple retraction. There were no skin changes in the remaining 26 patients (81%). The mass was subareolar in 27 patients (84%), located in the upper-outer quadrant in four patients (13%), and in the upper-inner quadrant in one patient (3%).

Surgery was the primary mode of therapy in all cases (Table I). Of the seven patients who underwent limited operation, two had metastases at the time of initial evaluation. Of the 25 patients who underwent curative resection, five patients were given postoperative radiation therapy, and two patients had postoperative chemotherapy. The influence of procedure on survival is shown in Figure 1. No statistical difference in survival comparing the two procedures is shown by Wilcoxon rank sum analysis ($p > 0.8$).

Microscopic examination revealed intraductal carcinoma in one

TABLE I. Surgical Procedure Used in Primary Therapy

Procedure	Number of Patients
Limited operation	
Excisional biopsy	1
Excisional biopsy with axillary node biopsy	1
Simple mastectomy	5
Curative operation	
Modified radical mastectomy	9
Radical mastectomy	16
Total	32

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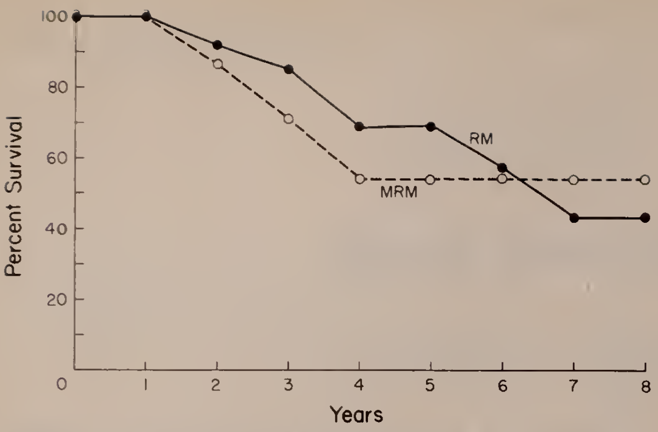

FIGURE 1. The influence of operative procedure on survival (RM, radical mastectomy; MRM, modified radical mastectomy).

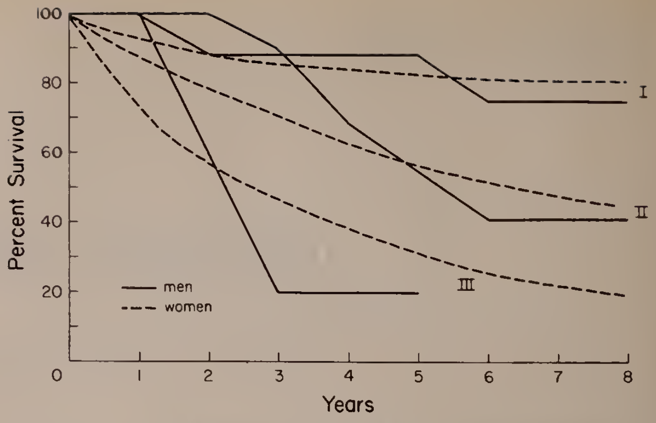

FIGURE 2. The influence of stage of disease on survival in carcinoma of the breast in men (this series) and in women (Ref. 21).

patient (3%), colloid carcinoma in one patient (3%), and infiltrating duct cell carcinoma in the remaining 30 patients (94%). Microscopic examination of axillary nodes was performed in 26 cases. Thirteen of these patients (50%) had axillary nodal metastases. Pathologic staging was possible in these 26 patients. The remaining six patients were clinically staged because of the limited operation performed. Stage of disease at the time of presentation is shown in Table II.

Hormone receptors were assayed in six of 32 patients (19%). Estrogen receptor assay was positive in four of six cases (67%). In addition, progesterone receptor assay was evaluated in three patients and was positive in one (33%).

Metastases were diagnosed at initial evaluation or during the follow-up period in ten patients (31%). The time interval from initial diagnosis to diagnosis of metastases was as long as seven years, with a mean of 35 months. Of these ten patients, nine had histologic evaluation of their axillary nodes at the time of primary treatment. Five of these nine patients (56%) had positive axillary nodes. Six patients were not treated for their metastases. Two patients underwent orchiectomy alone, one patient had radiotherapy alone, and one patient had orchiectomy and radiotherapy. Appearance of metastases preceded death by an average of eight months.

Tumor recurrence was documented in three patients (9%). Two patients had positive axillary nodes at the time of primary treatment. The average interval between treatment of the tumor and recurrence was 29 months. These three patients died an average of 25 months following appearance of recurrent tumor.

Excluding skin malignancies, other malignancies were diagnosed in four patients (13%). One patient had been treated for carcinoma of the colon seven years prior to his breast carcinoma. One patient had been treated with radiotherapy for carcinoma of the vocal cord ten years earlier, followed by laryngectomy for recurrent lesions four years prior to mastectomy. One patient had carcinoma of the prostate treated surgically six years after mastectomy. One patient underwent laryngectomy for carcinoma two years prior to mastectomy, and at autopsy was found to have carcinoma of the prostate.

Current follow-up is available for all patients. Eighteen patients have died, an average of 40 months after diagnosis. Five of these 18 patients (28%) died of causes other than the breast malignancy. Five-year survival by stage of disease at presentation is analyzed by the life-table method and shown in Figure 2. Patients with stage I malignancy had a five-year survival rate of 88%, those with stage II malignancy had a 54% five-year survival rate, and those with stage III had a 20% survival rate. Both patients with stage IV disease died within six months of diagnosis, one dying of unrelated causes (cerebrovascular accident).

TABLE II. Stage of Disease at the Time of Presentation

Stage	Number of Patients
I	9 (28%)
II	15 (47%)
III	6 (19%)
IV	2 (6%)

DISCUSSION

Several major characteristics of breast carcinoma differentiating the disease in men from that in women were set forth in a landmark paper by Wainwright[10] in 1927: breast carcinoma develops at a later age in men; lesions are usually located at the nipple in men; skin involvement is more common in men; axillary metastases occur with the same frequency as in women; tumor types are similar in both sexes; symptoms are present longer before diagnosis in men than women; and the prognosis is worse in men than women.

The average age at the time of presentation in this series is considerably higher than the mean age generally reported for women, as 54–57 years.[2] In contrast to the findings among women, unilateral single masses of the male breast are malignant. Nearly all of the patients in this series presented with a subareolar breast mass.

We are not aware of previous reports of carcinoma that developed in a breast infected with tuberculosis, as found in one of the patients reported here. We suspect that the infection called attention to a co-existing malignancy in this patient, and we doubt there is an etiologic relationship. In studies of female breast cancer, a predilection for the left breast has been observed. This has also been observed in male breast cancer. Although not statistically significant, it is interesting that we observed a left to right ratio of 1.67:1. Norris and Taylor[11] reported a ratio of 1.38:1.

No patient in this series had a family history of male breast cancer, and only one patient reported a family history of breast cancer among female relatives. Although we were unable to obtain family follow-up, we believe it is important to do so. The incidence of breast cancer in female children of males with breast cancer has not been well studied.

Trauma has been considered in the past as a possible etiologic factor in the development of carcinoma of the male breast. One patient in this series presented with a history of trauma. There is little evidence to suggest a cause and effect relationship between trauma and subsequent malignancy. We believe that any positive correlation between trauma and carcinoma of the male breast can be explained, as in women, by the fact that a history of trauma leads to a thorough examination of the patient's breast, revealing a previously undetected malignancy.

We presume the lengthy delays in the diagnosis of breast lesions in these men to be due to lack of attention to the breast by male patients and their physicians. This delay could be shortened with increased public awareness of male breast cancer as well as more frequent, thorough breast examinations in men. Hopefully this will lead to identification of lesions in an earlier stage, more amenable to cure.

In a series of 97 men with carcinoma of the breast, Heller et al[2] found 30% with stage I, 54% with stage II, and 16% with stage III disease at presentation. The stage at presentation in women is reported as 18% stage I, 62% stage II, and 20% stage III.[2] Thus, while the interval between onset of symptoms and definitive diagnosis in men may be longer than that in women, the patients present with similar stages of disease.

Hormone receptor assays in male breast cancer have been done only recently. Only six of 32 patients in this series were evaluated for estrogen receptors; all six of these were done since 1976. Gupta et al[12] reported a series of six men with five (83%) positive assays. The incidence of positive estrogen receptor assay in men is higher than that in women. Friedman et al[13] found clinical response to hormonal manipulation in seven of eight estrogen receptor positive patients. Most men with breast carcinoma have hormonally sensitive tumors. This series is too small and follow-up was not long enough to draw significant conclusions about the predictive value of steroid receptors for tumor responsiveness to hormonal manipulation. We recommend obtaining tissue for steroid receptors in all cases of male breast carcinoma. Quantitation of receptor protein may allow for a more accurate predictive role for the assay.[14]

The primary mode of therapy in male breast carcinoma remains surgical. Limited operation is acceptable therapy in stage IV disease. Clancy and Levien[8] found that, regardless of stage, all patients who underwent radical mastectomy survived ten years, as compared to an overall ten-year survival rate of 57%. However, Ouriel and coworkers[9] found no difference in survival at five years regardless of the procedure performed. Similarly, in this series, the difference in survival was not significantly higher in patients who had undergone radical mastectomy compared with modified radical mastectomy (Fig 1). It is important to point out that formal mastectomy is not required in the evaluation and treatment of gynecomastia, where simple excision of the offending tissue is sufficient. The significant predilection for the subareolar site of breast cancer in men should prompt early biopsy in unilateral breast disease, even if cause for gynecomastia is present. The role of mammography in men with bilateral gynecomastia is unknown but may be valuable.

Many reports of survival in men with breast carcinoma have shown a poor prognosis, especially when compared to data on female breast cancer. Gupta et al[15] have concluded that "most authors agree on poorer prognosis in male breast cancer as compared with female." Panettiere[6] collected several previous studies with five-year survival ranging from 10% to 40%. The overall five-year survival in this series is 63%, similar to the survival reported elsewhere.[8] Five-year survival by stage in women with breast cancer was shown in one study[2] to be 91% in stage I, 87% in stage II, and 51% in stage III. We found five-year survival in men to be 88%, 54%, and 20% for patients in stages I, II, and III, respectively (Fig 2), similar to results in other reports.[2,8,9]

In nearly one third of the patients in this series metastases developed at some time during the course of disease. Radiotherapy gave good symptomatic relief to those patients with osseous metastases. Kantarjian et al[16] noted poor response to hormonal therapy in men with a disease-free interval of less than 12 months, similar to previous results in female patients. Chemotherapy is the initial therapy of choice in these patients. For those patients predicted to benefit from hormonal manipulation on the basis of estrogen receptor assay, orchiectomy is the primary mode of therapy.[1] In a study of 23 men with metastatic disease, Kraybill et al[17] report an overall response rate to orchiectomy of 48%, with an increase in median survival from 24 months in nonresponders to 58 months in patients with response. More recently, tamoxifen has been used in the palliation of advanced disease. Patterson and coworkers[18] report a 48% response rate in 31 patients. In comparison, the overall response rate among more than 1,000 women with advanced disease was 32%. Kantarjian et al[16] reported a 25% response rate to tamoxifen.

Interestingly, malignancies other than the breast tumor developed in four patients (13%) in this series. Two of these were carcinoma of the prostate, diagnosed after the breast malignancy in both patients. Neither patient was treated with steroids. Sobin and Sherif[19] suggest that a direct relationship may exist between breast malignancy and carcinoma of the prostate. Langlands et al[5] reported a similar incidence of second malignancies. They concluded, on the basis of a historical control group, that the probability of developing a second primary lesion is sex-dependent, being more common among men than women with breast cancer.

While our results concur with the first six characteristics of male breast cancer stated by Wainwright, we are encouraged that they do not support his conclusion that carcinoma of the male breast has an inherently worse prognosis than that in women. Patient education is critically important to reduce the mortality of carcinoma of the breast in men. All unilateral breast masses in men must be considered malignant until proven otherwise. Pathologic staging of all patients is essential to direct appropriate therapy. Limited operation is suitable for patients with metastatic disease, but mastectomy including axillary nodal sampling is otherwise the minimal level of acceptable therapy. Although there have been no randomized prospective trials in male breast cancer to examine the influence of the type of procedure on survival, our data suggest that patients who underwent modified radical mastectomy will have survival rates similar to those patients who received radical mastectomy. However, radical mastectomy must be considered with obvious involvement of the pectoral muscles. Hormone receptor assay should be obtained on all tumors. Development of metastatic disease should be treated with chemotherapeutic agents in patients with disease-free intervals of less than 12 months. In patients with longer intervals, hormonal manipulation should be considered as initial therapy.

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See also pp 291–292.

Pap smear screening in elderly high-risk women

The role of the primary care physician

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ABSTRACT. Cervical cancer is the fifth leading cause of cancer deaths for women aged 75 and older. The elderly have been noted to be nonusers and underusers of Papanicolaou (Pap) screening for cervical cancer. There is a need to screen elderly women for cervical cancer in primary care settings. To investigate factors that affect compliance with Pap screening 75 elderly women who were previously offered gynecologic screening examinations in a primary care setting were interviewed. Forty-one accepted the Pap test and 31 refused. Patients who reported that their doctor had very strongly recommended the Papanicolaou smear were more likely to have accepted screening ($p < 0.05$). Selected aspects of the physician-patient relationship and provider gender were not related to compliance. These findings suggest that primary care physicians can improve compliance with Pap screening among certain high-risk patients.

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Cervical cancer is the fifth leading cause of death from cancer for women aged 75 and older.[1] In 1986 there were 14,000 new cases of cervical cancer and 6,800 avoidable deaths from this disease.[1] Women aged 65 and older account for 25% of the cases of invasive cervical cancer and 42% of the deaths due to cervical cancer in this country.[2,3] This disproportionately high incidence of and mortality from cervical cancer among the elderly has been noted in other countries as well.[4,5]

However, cervical cancer screening for the elderly is controversial. The American Cancer Society* recommends the cessation of screening at age 65.[6] This guideline assumes that a woman has had multiple tests prior to the age of 65. This upper age limit was determined by extrapolation from screening programs in British Columbia.[7] In that setting the incidence of abnormal Papanicolaou (Pap) smears was too low in the over-65 age group to justify the costs of screening. However, all of the women included in that study had had at least one prior Pap smear. It may be inaccurate to extrapolate from these data to elderly women in the United States, up to 40% of whom have never had a Pap smear.[8,9] In other countries, as many as 85% of women who have never had a Pap smear are over age 60.[4] Women who have never had a Papanicolaou smear have a 2.7 to 4.0 times increased risk of having invasive cervical cancer when they are screened, compared to women who have had at least one prior Papanicolaou smear.[4,10,11]

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* The revised American Cancer Society recommendations, published in *Ca—A Cancer Journal for Clinicians* (1988; 38(2):127-128), have removed this upper age limit.

It has therefore been suggested that cervical cancer screening programs need to reach older, high-risk women to be effective in decreasing the mortality from this preventable cancer.[8,12,13] Elderly women generally see primary care physicians rather than gynecology specialists.[3] When primary care physicians provide gynecologic care for elderly women, a high prevalence rate of abnormal Pap smears (13.5/1,000) has been observed. However, almost one half of patients do not participate in gynecologic screening when it is offered in a primary care setting.[12] This study explored the factors associated with an elderly woman's participation in primary care screening programs. We investigated the relationship between patient participation in screening and physician gender, selected aspects of the physician-patient relationship, and the strength of the physician's advice to participate. The role of primary care physicians in improving the gynecologic care of elderly patients is discussed.

PATIENTS AND METHODS

The Medical Primary Care Unit (MPCU) of the City Hospital Center at Elmhurst, NY, is an outpatient clinic where more than 60 practitioners (attendings, house staff, and nurses) provide comprehensive care to more than 10,000 patients per year with chronic medical illnesses. Patients visit the MPCU about every three months and are scheduled to see the same provider on each visit. The patient population is approximately 44% white, 16% black, 38% latino, and 2% Asian. Approximately two thirds of the population is female. The average patient age is 65 years.

Gynecologic screening was offered to all women aged 65 years and older who were receiving routine, ongoing care in the MPCU. Characteristics of the patients and the screening program have been described elsewhere.[12] Briefly, all patients were coming to the MPCU for ongoing medical care, and no patients presented with primary gynecologic problems. Historical information was obtained by the patient's provider, and all women were offered screening examinations. Overall, screening was offered to 1,542 women, and 816 participated. The mean age of participants was 74 years (SD ± 6.11 years). The racial distribution was 66% white, 17% black, 15% Hispanic, and 2% Oriental. Seventy-five percent of the women reported having had a Pap smear at some time in their life, but on average, this test had been performed five years before (SD ± 6.99 years). Only 26% of the women reported regular screening, defined as every one to five years, and 25% of the women had never had a Pap smear. The overall prevalence of abnormal Pap smears (for cervical intraepithelial neoplasia, carcinoma in situ, or invasive cancer) was 13.5 per 1,000 (95%, confidence intervals 5.6 to 21.4/1,000).

Logistic regression models revealed that the women who participated were younger, white, more frequently screened in the past, and had a higher hysterectomy rate. Therefore, the nonparticipants (47% of the sample) were likely to have a higher risk for cervical cancer than the participants.

To explore reasons for the high nonparticipation rate observed in the program, a cross-sectional study was conducted in the MPCU between October 7 and November 2, 1985. The sample consisted of all women who attended the clinic between October 14 and October 18, 1985. Criteria for inclusion in the study population were as follows: date of birth before 1920 (determined by birthdate on clinic card); and receiving medical care in the MPCU during the period that the original gynecology screening program had been offered (September 1984 to June 1985).

As patients registered for their routine medical visit, their age was verified by their clinic card. One hundred and sixty-six women were eligible to participate. An attempt was made to locate each woman. One hundred and thirty-nine women were included in the study population; 27 were excluded, having left the MPCU early in the clinic session; and 18 failed to meet the second study inclusion criterion. In a standardized format, interviewers explained that they were medical students conducting a survey on health care in the MPCU, and that all responses were confidential. Patients were informed that the researcher was blind to the identity of their physician, and that participation would not affect their medical care. Informed consent was obtained from 108 patients. Thirteen refused to participate. The most common reasons given for refusal were "in a hurry" and "fatigue" from spending several hours in the MPCU (Table I).

Data were then collected via standardized interview by one of the authors (DT, HP). Interviews were conducted in the language which the patient preferred—English or Spanish. The interviewers were blind as to which patients had previously been offered screening and their prior participation status. Patients were instructed to answer all questions in reference to the physician visited during the time period screening was offered.

The study instrument was a questionnaire containing three sections with a total of 15 items in either a yes, no, or graded response format. The first section ascertained gender of the primary care physician and patient ethnicity. In the second section, selected aspects of the physician-patient relationship were assessed by eight questions distributed among three categories: communication, physician affect, and patient satisfaction.[14-18] Responses were graded on a five-point Likert scale ranging from "never" (value = 1) to "always" (value = 5). The Likert vector was reversed for negative constructs. Mean scores were calculated for each category, and values for all eight questions constituted an overall score. The third section contained a set of questions referring to five preventive health measures, including a Pap test. To blind patients to the actual focus of the study, the hemoccult test, influenza vaccination, rubella screening, and electrocardiogram were included. In this population of elderly patients, electrocardiograms are frequently performed, hemoccult tests and influenza vaccination are often recommended, and rubella immune status is rarely obtained. The inclusion of these measures serves as an approximation of internal validity and as a check for indiscriminate answering. Each of these five measures was explained in a standard manner. Patients were then asked whether their doctor had recommended each measure, and if so, how strongly. Strength of recommendation was recorded on a five-point Likert scale from "never" (value = 1) to "must have" (value = 5). Patients were asked when they last had each of these tests. They were also asked whether they would now, at their physician's recommendation, have a Pap test.

On completion of all interviews, race, participation status, and reported date of last Papanicolaou test were compared to the data obtained in the original gynecology screening study.[12] Patients were identified by medical record number for cross referencing. This comparison served as an approximation of reliability of the data.

Incomplete data were obtained in eight cases, in which the patient prematurely terminated the interview or was unable to answer the questions. Of the 100 women who completed interviews, 25 had not been offered a Pap test in the previous screening program (the original study did not include 20% of the elderly female clinic population due to clerical work overload[12]). Subsequent analyses are confined to the remaining 75 patients.

RESULTS

The study population was 68% white, 21% black, and 11% latino.

TABLE I. Response Rate

Eligible	166
Excluded—left unit early	27
Subtotal	139
Failed to meet study criteria	18
Subtotal	121
Refused	13
Total	108

The mean age was 76.0 years (± SD 6.6 years). Forty-one women (55%) had participated in the previous gynecologic screening program, and 34 (45%) had not. (Hereafter, they are referred to as "participants" and "nonparticipants," respectively). Twenty-four percent of nonparticipants reported never having had a Pap test; 24% reported having had one more than five years ago; 50% reported having had a Pap test in the last five years (2% missing data). All participants, by definition, had had a Pap test within the last year.

Participants had a significantly higher mean score for strength of physician recommendation than nonparticipants ($p = 0.04$, two-tailed student t-test). In addition, a significantly ($p = 0.002$) higher proportion of participants (71%) reported that they would undergo a Pap test now if recommended by their physician, compared to nonparticipants (15%). Physician gender and selected aspects of the physician-patient relationship were not significantly associated with participation status. Table II summarizes the independent variables by participation status.

Overall, a high degree of reliability of patient responses was found. Eighty-eight percent of participants accurately reported having had the Papanicolaou test during the period of the original program. Among the nonparticipants, Papanicolaou smear history was 81% concordant for those women (79%) for whom comparative data were available. Discordant information was reported by five nonparticipants. Of these, two reported having had a Pap test after the period of the original study.

In addition, the validity of patients' responses to the questions on the five preventive health measures was demonstrated by a check on indiscriminate answering. Ninety-six percent (N = 72) of patients accurately reported that their physician had never recommended rubella screening, while 68% (N = 50) of patients said that their physician had recommended an electrocardiogram. Responses regarding recommendations for hemoccult testing (31%) and influenza immunization (56%) fell in between. Responses did not differ significantly for participants and nonparticipants for any of these preventive health measures (Table III).

DISCUSSION

In this urban primary care setting, there was a strong relationship between the strength of the physician's recommendation to have a Papanicolaou test and an elderly woman's participation in screening. However, physician gender and patient-physician relationship were not related to compliance. Also, women who did not participate in screening were less likely than their participant counterparts to do so in the future. These results are consistent with previous findings that a large percentage (50–75%) of nonparticipants had not had regular prior screening, and that one quarter had never been screened,[12] thus constituting a high-risk group.

The findings of this study must be considered in light of several limitations. First, it is possible that women who refused the Pap screening were less interested in general health care issues, and therefore were more likely to refuse the interview. This self-selection would lead to a sample that was disproportionately weighted with participants who might be more health conscious. However, results show that the ratio of participants to nonparticipants in this study was similar to the ratio in the original population, in which all patients were included in the sample. Therefore, it is unlikely that this self-selection occurred. It is also possible that a health conscious group is more responsive to any physician recommendation. A controlled trial would be necessary to test this possibility. Second, questions pertaining to aspects of the physician-patient relationship may not have been sensitive enough to detect small differences in the two groups, given the small sample size. Third, our questionnaire did not account for level of patient education, knowledge regarding the benefits of Pap screening, or utilization of screening offered outside of MPCU. Lastly, due to the retrospective nature of this study, conclusions pertaining to causality and predictive value cannot be drawn.

A strength of the study design is the validation of Pap screening participation through medical records. Although the study design is limited by reliance on patient self-reporting in other areas, we were able to demonstrate a high degree of validity and reliability when the data

TABLE II. Predictors of Patient Participation

	Participants N = 41	Nonparticipants N = 34	*P* value
Age (yr)	76.0 ± SD = 6.8	77.3 ± SD = 6.4	NS*
Race			
White	22	29	
Black	12	4	
Latino	7	1	
Total	41	34	
Recommend Pap	3.07 ± SD = 1.39	2.38 ± SD = 1.44	0.04
Pap now			
Yes	29	11	0.002
No	12	21	
Total	49†	33†	
Physician gender			
Female	17	13	NS
Male	23	21	
Total	40†	34	
Patient-physician relationship			
Global	3.80 ± SD = 0.37	3.71 ± SD = 0.48	NS
Communication	3.57 ± SD = 0.76	3.48 ± SD = 0.65	NS
Physician affect	4.76 ± SD = 0.47	4.63 ± SD = 0.65	NS
Patient satisfaction	3.06 ± 1 = SD = 0.36	3.01 ± 1 = SD = 0.56	NS

* NS, not significant.

† Data missing on one to two patients.

TABLE III. Use of Preventive Health Measures by Study Participants and Nonparticipants

	Participants N = 41	Nonparticipants N = 34	Total N = 75	*P* value
Suggest electrocardiogram				
Yes	28	22	50	NS[*]
No	11	12	23	NS
Total	39[†]	34	73[†]	
Suggest hemoccult testing				
Yes	14	9	23	NS
No	27	25	52	NS
Total	41	34	75	
Suggest influenza immunization				
Yes	21	21	42	NS
No	20	13	33	NS
Total	41	34	75	
Suggest rubella screening				
Yes	3	0	3	NS
No	38	34	72	NS
Total	41	34	75	

[*] NS, not significant.

[†] Data missing on one to two patients.

were cross referenced with those of the prior screening study. Another strength is that the study population was similar to that of the original gynecologic screening program with respect to mean age, racial distribution, ratio of participants to nonparticipants, and history of prior Papanicolaou smear screening.[12]

The findings of this study are supported by similar findings in other settings. In a rural area Celantano et al[3] found that a physician's advice to get a Papanicolaou test was positively related to the recent participation of elderly women in cervical screening.[3] In Britain, Standing and Mercer[19] were able to increase compliance with Pap screening in general practice from 50% to 96% through personal letters, phone calls, and home visits. It has been noted that women who are at high risk for cervical cancer are the least likely to participate in Pap smear screening.[13,19-21] Therefore, the primary care physician plays a pivotal role in reaching high-risk patients,[12,19-22] and is likely the single most important person to influence whether or not a patient seeks out and receives a Pap smear.[21]

The demonstration of an association between physician recommendation and participation provides primary care physicians with an inexpensive tool for increasing compliance with Pap test screening among elderly high-risk patients. Primary care physicians can act as catalysts to cancer prevention and early detection activities for their elderly patients.

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Health and disease in Greenland (Kalaallit Nunaat)

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Greenland, or Kalaallit Nunaat, as it is known in Inuktituk, the linqua franca of the Inuit, is the largest island in the world, covering some 840,000 square miles, of which 708,100 are submerged by ice. The island is 1,650 miles long and 750 miles across at its widest point. Its coastline is indented, sheltering numerous fjords. The ice sheet that covers most of Greenland is the dominant feature of the country. Its average depth is 5,000 feet and in certain areas it is 10,000 feet deep. The ice sheet is in a sense contained by the mountains that rim most of Greenland, and tongues out into the fjords in the form of glaciers. The edges of these glaciers can advance at a velocity of 97 feet a day, and either melt or else calve icebergs. The weight of the ice sheet has depressed the land beneath it to below sea level. The population of Greenland was estimated in 1986 to be 53,406 (Table I).[1-5]

Thoughts of Greenland conjure up images of snow and ice. However, the southwest coast, which is warmed by the Gulf Stream, has moderate temperatures from June through August, reaching as high as 65°F at times. Rainfall, however, can be heavy in the southwest during the summer months, and fog and mist are frequent. Still, the summer climate in the southwest is temperate enough to support sheep and crops such as wheat, cabbage, radishes, lettuce, and potatoes.[4,5]

Greenlandic society has undergone major changes since World War II. By the 1970s most of the population, except for those in the extreme north, had both Eskimo (Inuit) and Danish ancestry. The people of Greenland prefer to be called Greenlanders, and many view the word "Eskimo" as pejorative.[6] The preferred modern word for their language is *Greenlandic.* Gradually, most place names which were originally Danish are being changed to Greenlandic, and frequently both the Danish and Greenlandic names are used simultaneously.[6] Most Greenlanders speak Danish.[6] Danish influence in Greenland began in 1721 when Hans Egede, a Lutheran missionary, landed at the mouth of what is now Godthab (Nuuk) Fjord on the west coast. Eventually the Danes settled in Godthad (Good Hope), now called *Nuuk* in Greenlandic, and in other places along the west coast.[7-9]

Until 1951 Danish immigration to Greenland was not great, most of the Danes in the country being administrators. Until that time entry into Greenland was severely restricted. However, after 1951 the economic monopoly of the Royal Greenland Trading Company was abolished, and in 1953 Greenland's colonial status was terminated, it becoming an integral part of Denmark. Danish immigration increased during the 1950s and 1960s in conjunction with the modernization of the country.[4] Some of the impetus for this modernization arose from Greenland's strategic geographic location, which made it important for radar installations, weather observations, and air communications. The large US air base at Thule in the northwest, established in 1951, exemplifies Greenland's importance to the North Atlantic Treaty Organization (NATO).[5,9]

TABLE I. Population of Greenland, 1986[1,2]

County/Towns	Area in Square Miles	Population
Avanersuaq (Nordgronland)	41,200	
Qaanaq (Thule)		811
Kitaa (Vestgronland)	46,000	
Aasiaat (Egedesminde)		3,499
Ilulissat (Jakobshavn)		4,425
Ivittuut (Ivigtut)		36
Kangaatsiaq (Kangastsiaq)		1,259
Maniitsoq (Sukkertoppen)		4,006
Nanovtalik		2,752
Narsaq (Narssaq)		2,120
Nuuk (Godthab)		11,438
Paamiut (Frederickshab)		2,739
Qaqortoq (Julianehab)		3,291
Qasigiannguit (Christianshab)		1,811
Qeqertarsuaq (Godhavn)		1,062
Sisimiut (Holsteinborg)		4,817
Upernavik		2,198
Ummannaq (Umanaq)		2,582
Tunu (Ostgronland)	44,800	
Illoqqortoormiut (Scoresbysund)		535
Tasilaq (Angmagssalik)		2,794
Total area (ice-free)	131,900	
Permanent ice	708,000	
Total	840,000	53,406*

* Includes 1,231 people not distributed by county.

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This paper presents an overview of Greenland and its principal health problems based on a literature review and observations made during a field trip in 1987.

HISTORY

Humans have lived in Greenland for more than 4,000 years. Four distinct waves of Inuit immigration have been archeologically documented in Greenland. Early settlers came from the west in what is now Canada and fished and hunted their way along the coast.[10-12] These early Inuit settlers lived by hunting seal, walrus, whale, polar bear, and reindeer, a way of life that persisted into the early part of the 20th century.[12]

In 1978 two graves containing the well preserved bodies of six adult females and two children were found under a cliff overhang high in a ravine at Qilakitsoq.[13] The bodies, which were fully clothed, date from the 15th century and have provided anthropologists and archeologists with important information on the Inuit and Inuit life 500 years ago. Of medical interest is the fact that the skull of one of the adult mummies showed evidence of an extensive neoplastic lesion of the nasopharynx.[13] Other well preserved mummies have been found in various areas of Greenland and date from the 16th and 17th centuries. However, those found at Qilakitsoq are remarkable for their state of preservation and for that of their clothing.[13] The types of clothing and the materials from which they were made (furs and skins) were still widely used well into the 20th century.

The Inuit were not the only ones to settle in Greenland. In 982 AD a Viking settler in Iceland, Erik the Red, got into a dispute with a neighbor which led to armed conflict and the neighbor's sons being killed. The local *Althing* (Viking assembly) banished Erik, who decided to sail west. In so doing he came to Greenland, whose west coast he explored over the next three years. It was a land of many fertile fjords and he returned to Iceland seeking colonists. Erik called this new country Greenland because of the color of the lichens and grass that covered many of the slopes along the upper portions of the fjords. Eventually, Erik led 25 ships back to Greenland, only 14 of which successfully made the trip. The settlements he founded were to last 500 years. By the year 1300 there were 3,900 Norse inhabitants on Greenland's west coast, living on 300 farms. The settlers lived in two large settlements, the eastern settlement in the south and the more sparsely populated western settlement 400 miles to the north in the area of Nuuk, modern Greenland's capital. There were some 17 churches and 2 monasteries.[10,14-17]

Erik the Red's son, Leif, sailed westward from Greenland in 1000 AD and came to the coast of North America, eventually setting foot in what he called Vinland. Many contemporary scholars believe this was somewhere in Newfoundland.[15,18]

Life in the Viking settlements on Greenland's west coast must always have been precarious, yet the Norsemen endured there for 500 years, until the early 15th century as far as we know. The ruins of their settlements, churches, and farmsteads litter the southern portion of the west coast. These settlements were in regular contact with one another and with Norway, trading being carried out through the port of Bergen. The last documentation of Norse settlement of Greenland dates to 1408 and concerns a wedding that took place in Hvalsoy Church on September 16 between Torstein Olavsson, a member of a visiting Icelandic ship's crew, and Sigrid Bjornsdatter. There was nothing unusual about this wedding except that it is the last documented event found so far in records concerning the Norse in Greenland.[10,15] After this nothing more is heard about the Norse. When the coasts of Greenland were rediscovered in the late 16th century by John Davis, Martin Frobisher, William Baffin, and others, no Norse were found.[19] By then Inuit were the only inhabitants of Greenland. What ultimately happened to the Viking settlers remains a mystery. However, some scholars postulate that the development of a colder climate with impenetrable ice packs in the seas around Greenland effectively blocked trade contacts and caused the demise of the settlements.[10]

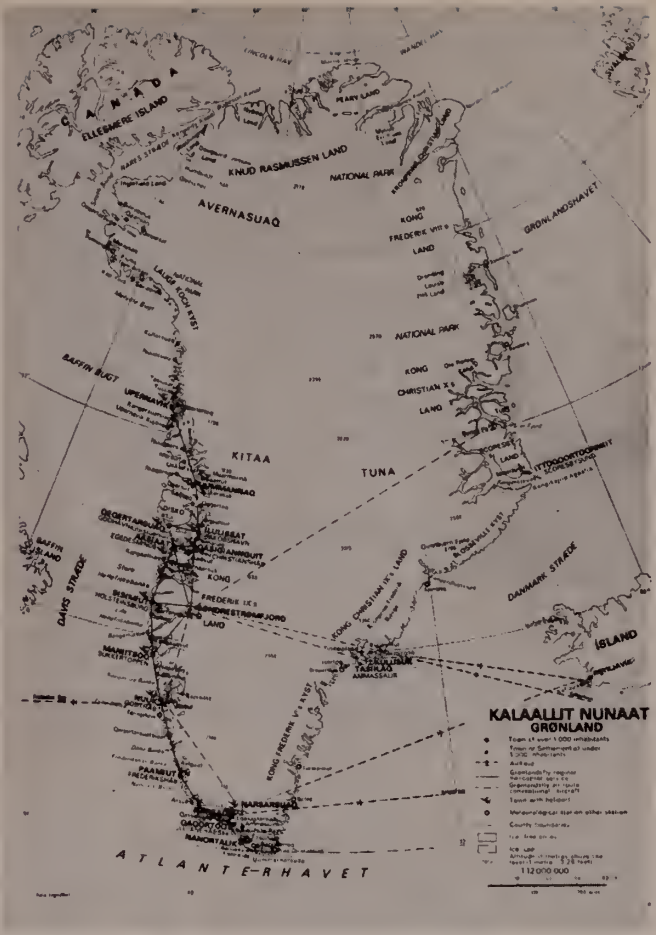

FIGURE 1. Greenland[4] (courtesy of the Danish Tourist Board, New York City).

Between the 16th and 18th centuries English, Dutch, and Basque whalers operated in the waters off Greenland. The Dutch also engaged in limited trade along the west coast, taking out furs and pelts in exchange for wood, iron, beads, and textiles. However, it was not until 1721 that a Danish Lutheran missionary, Hans Egede, landed on the west coast at a place he called Godthab (Good Hope). This settlement eventually became the capital of Greenland. Egede, who in addition to being a missionary was also a teacher, explorer, and trader, set up a number of trading posts.[9] In 1776, Denmark established a trading monopoly over Greenland.[5] At the time of Hans Egede's visit to Greenland, the Inuit population was approximately 5,000.[5]

Danish control of most of Greenland was fairly complete by the late 18th century. However, Norwegian challenges to Danish sovereignty over the uninhabited northeastern portion of the island continued until 1933, when the International Court of Justice at the Hague rejected the Norwegian claims.[5] During World War II, when Denmark was occupied by Germany, Greenland came under the temporary protection of the United States. Until the advent of World War II, Greenland was essentially closed to outsiders, who could only enter with special permission from the Danish government. Danish presence in Greenland largely consisted then of a few administrators and officials of the Royal Greenland Trading Company which enjoyed a monopoly on the island. With the termination of colonial status in 1953 and major initiatives to develop the deep sea fishing industry along the west coast, large numbers of Danes entered the country. In 1953 Greenland became an integral part of Denmark and in 1979 was given home rule with a legislative assembly (Landsting) and a home rule government (Landsstyre) headed by a prime minister.[3] The home rule government has authority over most matters except foreign affairs, defense, and finance.

ECONOMY

Economic changes have had a major impact on Greenland's society. Seal hunting, once the principal mode of existence, has increasingly declined in importance, being largely restricted to the northern and eastern parts of the country.[12] At present a few hundered of Greenland's 54,000 people are still involved in seal hunting.[20] Deep sea fishing—especially for shrimp, prawns, cod, wolf fish, halibut, and salmon—and associated industries employ close to 25% of the population.[2,20] There are several thousand fishermen using over 1,000 motor boats and over 70 fish processing plants employing several thousand people in canning, drying, and freezing processes. Greenland's relatively new deep sea fishing industry expanded largely because of the Danish government's financing of canning and freezing facilities, the construction of docks for oceangoing ships, and the development of local sources of power and fresh-water supplies. The economic advances of the last 20 years have greatly elevated the standard of living.[20] Today the average per capita income in Greenland is $10,000.[2]

Greenlanders, sensitive to international concerns about continued whale and seal hunting, are quick to point out that only adult seals are killed and that whaling is not done on a commercial basis.[20] Greenlanders take 80,000–90,000 adult seals annually. The export market for seal skins has been steadily declining because of the international campaign against the slaughter of baby seals off the Canadian coast. Seal meat and whale products are largely consumed locally. However, Greenlanders also hunt blue fox, white fox, walrus, eider ducks, and other birds.[20] Polar bears, musk ox, eagles, falcon, and snowy owls are largely protected in Greenland. In 1952, 300 tame reindeer were brought to Greenland from Norway. By 1987 their numbers had risen to 5,000, which graze on the slopes of Godthab Fjord. The herd is owned by a cooperative and 1,300 head are slaughtered annually.[5] Reindeer horn is frequently employed by Greenlandic artists for sculpting *tupilaks*, small figures representing malevolent spirits once used for sorcery.[20]

Although the per capita income of Greenland is high at $10,000 a year, the country is still dependent on large subsidies from Denmark that in recent years have amounted to around $4 million annually.[3] The transformation of the indigenous society from one of nomadic hunters to an industrialized one primarily centered on deep sea fishing has largely occurred in the 20th century. The population has progressively become urban, living in towns and settlements along the west coast. With this urbanization have come public and social services which also employ large numbers of people (Table II).

Fish and fish products comprise 77.3% of Greenland's exports and animal products, lead, and zinc account for most of the remainder. Exports are sent to Denmark (75.4%), West Germany (6.9%), Belgium-Luxembourg (4.2%), and the United States (1.7%). Machinery and transport equipment account for 27.2% of the value of imports, food for 18.1%, fuels for 16.8%, and metal products and semimanufactured goods for 16.4%.[2,3] In terms of value, Denmark supplies most of Greenland's imports (59.2%).

TABLE II. Distribution of Greenland's Labor Force and Structure of the Gross Domestic Product, 1985[2,3,5]

Sector	Percent of Total Value of Domestic Product	Labor Force	Percent of Labor Force
Agriculture (fishing, hunting and sheep raising)	16.0	3,222	15.1
Mining and manufacturing	31.6	3,205	15.0
Construction	27.4	3,112	14.5
Transportation and communication	4.8	1,842	8.6
Trade	8.0	2,153	10.1
Public utilities	12.2	293	1.4
Public administration and education		3,233	15.1
Social health services		2,141	10.0
Other		2,177	10.2
Total	100.0	21,378	100.0

MODERNIZATION

Greenland has undergone rapid modernization during this century and especially since the end of World War II. This is exemplified by the fact that in 1985 the Museum of Greenland in Nuuk sponsored a program to teach young men the art of making *kayaks*, once a ubiquitous means of transportation. It was found that in the region of Nuuk, the capital, only a few old men knew how to make these boats.[20] They were asked to teach the art to a group of 50 younger men.[20]

Greenland has compulsory education for nine years and offers four additional years for those who wish to continue. Technical training is available in Greenland but Greenlanders must leave the country for university level training.[5] To facilitate both education and the delivery of other social services the government has encouraged settlement in towns and villages. This too has had an impact on the traditional Greenlandic way of life as evidenced by the country's 100% literacy rate and the fact that 61.7% of the population has had a primary school education.[5] There are 17 branch libraries in the country, local book and newspaper publishers, 10,000 television sets, 13,000 radios, 10,720 telephones, and 1,600 automobiles.[5] Transportation within the country is largely provided by boats of the Royal Greenland Trade Department which sail up and down the west coast and by the large air network of Grondlandsfly which uses S-61 helicopters and Dash-7 and Twin Otter aircraft.

POPULATION

The population of Greenland is roughly 54,000. This is ten times the population estimated to have been living on the island when Hans Egede first visited it in 1721.[5] Approximately 78.8% live in towns and 21.2% in rural settlements. Thus the country's population is overwhelmingly urban.[2,3,21] This progressive urbanization of the population intensified after World War II and reflects the changeover of the society from one of hunters to one of deep-sea fishermen. In 1951, 40% of the population was urbanized.[21] By 1960, 59% was, and by 1979 the figure was 73.3%.[21] Most of the settlements and towns in which people live are located on the coast near the sea, and well over 85% of the total population lives on a narrow strip of mountainous land along the west coast. As urbanization has increased, the number of individual small settlements has declined as people congregate in the larger towns. For instance, in 1960 there were 149 settlements, but by 1972 these had fallen to 127.[21]

FIGURE 2. The center of Nuuk, capital of Greenland.

TABLE III. Vital Statistics, Greenland, 1986[1-3]*

Birth rate	20.0/1,000 population
Legitimate births	32.3%
Illegitimate births	67.7%
Death rate	8.3/1,000 population
Natural increase rate	11.7/1,000 population
Total fertility rate	2.1/per childbearing woman
Marriage rate	6.0/1,000 population
Divorce rate	2.6/1,000 population
Life expectancy at birth	
Males	59.7 years
Females	67.3 years
Sex distribution	
Males	54.3%
Females	45.6%

* *Gronland 1986. The Seventeenth Annual Report.* Copenhagen, Ministry for Greenland, 1987.

Greenland's current birth rate is 20.0/1,000 population, well below the world average of 29.0.[1-3] At this rate the population will double in 62 years (Table III).[1]

Greenland's birth rate has declined dramatically over the past 30 years. In 1960 it was 50.2 per 1,000 population, in 1965 45.7 per 1,000 and in 1970 25.7 per 1,000.[21] This decline is accounted for by family planning programs and the entry of women into the job market. Greenland's rapid population increase in the years just prior to 1960 was largely due to a decrease in mortality rates and an increase in birth rates. Medical interventions such as programs against tuberculosis had a major impact in lowering mortality rates.

Greenland's population is overwhelmingly young (Table IV), the largest proportion being found in the 15–29-year age group. This age group currently accounts for 33.7% of the population. It reflects the high annual population growth rate following medically influenced declines in death rates prior to the establishment of family planning programs. The progressive decline in birth rates has principally involved older women. Fertility rates for teenage girls remain comparatively high. Teenage girls have a high illegitimate birth rate.[20,22] The overall declines in fertility rates in Greenland have taken place at a time of rapid modernization.[23] However, the widespread introduction of contraceptives in 1966 and 1967 has clearly played a major role.[24,25]

It must be kept in mind that the total number of live births per year in Greenland is small (Table V). What has characterized fertility rates is a marked decline among older women and only a modest decline among teenage

TABLE IV. Age Distribution of Greenland's Population, 1986[1,2]

Age Group	Percent
0–15 years	24.7
15–29 years	33.7
30–44 years	23.2
45–59 years	12.6
60–74 years	4.6
75 and over	1.2

TABLE V. Total Births for Selected Years, Greenland, 1965–1984[1,21]

Year	No. of Births
1965	1,650
1972	770
1974	665
1980	1,028
1981	1,051
1982	1,066
1983	985
1984	1,050

girls.[22] In 1980, teenage girls accounted for 22% of all live births compared to 11% in 1960.[22] In 1983 they accounted for 20.1% of live births (Table VI). With the legalization of abortions in 1975, the number of abortions has sharply risen to over 600 a year (Table VII).[22] Over half of these are among teenagers.[22] It appears that teenagers either do not readily use contraceptives, are not encouraged to use them, or else do not understand the necessity for using them. Sex education has historically not been extensive in Greenland.[26] The high number of abortions among teenage girls seems to support the conclusion of Hansen and Smith that this is still the case.[22] In 1986, 67.7% of all live births were illegitimate.[20] The figures for previous years were 21.3% (1950), 33.6% (1960), and 45% (1974). Although Greenland has one of the highest illegitimate birth rates in the world, it must be remembered that many such births take place in stable family settings.[21] Yet teenage pregnancy and illegitimacy continue to be significant medical and social concerns.[20]

OVERALL MORTALITY

Life expectancy for both males and females has dramatically improved over the past several decades.[21] In 1946 it was 32.2 years for males and 37.5 years for females.[21] In 1986 these levels had risen to 59.7 years for males and 67.3 years for females (Table III).

Prior to 1967 no death certificates were used. Since then Danish certificates have been used.[21] Despite the absence of certificates for previous years, it has been possible to reconstruct the major causes of mortality in certain parts of the country for years past from other types of records.

The current death rate for Greenland is 8.3 per 1,000 population.[1] The crude number of annual deaths has ranged from 383 to 470 in recent years (Table VIII). The leading causes of death among young adults are accidents and suicide (Table IX).[21] Malignant neoplasms, accidents, suicide, and cardiovascular disease account for most of the deaths (Table X). During the 1950s Greenland experienced a dramatic decline in mortality, from 24 per 1,000 in 1951 to 8 per 1,000 in 1960, representing a 10% decline per year. In 1951 one third of all deaths in the country were due to tuberculosis. The dramatic decline in mortality rates during the 1950s was due not only to control of this disease but also to an economic development program that carried with it improved standards of living, and preventive and curative health care programs.[27]

TABLE VI. Live Births by Age Group, Greenland, 1983[1]

Age Group	Number of Births	Percent of Births
<15	2	0.2
15–19	198	20.1
20–29	591	60.0
30–39	176	17.9
40–44	18	1.8
Total	985	100.0

TABLE VII. Abortions in Greenland, Selected Years, 1975–1985[1,132]

Year	Number of Abortions
1975	381
1976	359
1977	388
1978	379
1979	450
1980	470
1981	539
1985	654

TABLE VIII. Crude Number of Deaths, Greenland, 1980–1984[1]

Year	Number of Deaths
1980	383
1981	383
1982	425
1983	434
1984	470

TABLE IX. Crude Deaths by Age Group in Years and Sex, Greenland, 1983[1]

	Number of Deaths	
Age Group	Male	Female
<1	24	15
1–9	6	2
10–14	5	2
15–19	13	7
20–24	22	6
25–29	14	3
30–39	22	5
40–49	31	20
50–59	33	19
60–69	37	26
70–79	35	43
80+	18	26
Total	260	174

TABLE X. Major Causes of Mortality, Greenland, 1987[2,20]

Cause	Rate per 100,000 Population
Malignant neoplasms	138.7
Accidents	119.7
Cardiovascular disease	89.3
Suicide	62.7

CAUSES OF MORBIDITY AND MORTALITY

Prior to the advent of extensive contact with Europeans, Greenlanders were subject to the vagaries of the harsh Arctic climate, uncertain food supplies, and the physical risks of hunting and fishing on the ice. Death from starvation and accidents was quite common.[12] Not surprisingly, injuries and deaths from accidents were more common among males who hunted and fished. Contacts with Europeans exposed the native Greenlanders to infectious agents against which they had no herd immunity. The inevitable result was frequent epidemics which often carried high mortality rates. These changing morbidity and mortality trends have been well documented for a number of localities in Greenland since the latter part of the 19th century. The dawn of the 20th century saw morbidity and mortality curves dominated by the infectious diseases, which frequently appeared in episodic epidemic form. As the decades passed the impact of these diseases was lessened by improvements in hygiene, housing, and the development of preventive and curative health services.

Iversen has provided a detailed overview of mortality trends in Greenland from the early part of the century until recently.[27] From 1907 to 1950 the mortality curve was characterized by numerous peaks of 35/1,000 with inter-peaks of 20–25/1,000. The peaks were due to a number of serious epidemics of infectious diseases such as influenza and pertussis.[27] The impact of these high mortality rates on population growth was not as great as one might have expected because the crude birth rate for this period ranged between 37 and 47 per 1,000.[27] Thus the population rose from 13,000 in 1910 to 23,000 in 1950.[27] After 1950, mortality peaks disappeared not because epidemic diseases disappeared, but because of preventive measures and the extension of curative health services.[27]

Robert-Lamblin was able to carefully document the principal causes of mortality over an 80-year period from the late 19th century until 1980 in Angmagssalik (Ammassalik), East Greenland.[28] This east coast settlement was not visited by Europeans until 1884 and a trading post and administrative services were not established until 1894. The changing pattern of mortality in this carefully studied community is a good mirror of what occurred over other time frames in those regions of Greenland that had contacts with Europeans much earlier.

In 1906 the first Greenlandic midwife came to the settlement; in 1932 the first Danish nurse, and in 1944 the first permanent Danish physician.[28] Infectious diseases such as whooping cough, influenza, poliomyelitis, and tuberculosis were very significant causes of mortality in Greenland during the first 50 years of this century.[21,28] Measles epidemics became increasingly common beginning in the early 1950s, as did hepatitis B infection.[21,28,29] The principal reason why Greenlandic society was not decimated by these epidemics of infectious diseases was because of its high fertility rate.

MEASLES

The first documented measles epidemic occurred in Greenland in 1951 and was meticulously studied and reported on by Christensen and colleagues.[29,30] The rapid spread of this epidemic, in which 4,247 cases were recorded, was facilitated by the fact that the index case had contact with large numbers of people.[29,30] He was a Greenlander who had contracted the disease in Denmark and returned by ship to Qaqortoq (Julianehab) in the southern part of the country. Six days after his arrival a welcoming ceremony (*mik*) was held in his honor which large numbers of people attended.[29,30] A total of 77 deaths occurred and the most commonly observed complications were pneumonia, otitis media, encephalitis, and cardiac failure among the elderly.[29] The highest death rates were among males and females above 55 years of age, being 159/1,000 and 104/1,000, respectively.[29,30] Most of the deaths in this age group were attributed to heart failure, tuberculosis, and pneumonia.[29,30] Major epidemics of measles subsequently occurred in Greenland in 1954, 1959, 1963, and 1967.[28] Since the inception of immunization programs, the incidence of the disease has greatly declined. Pedersen et al[31] have recently studied the long term antibody response to measles vaccination in Greenland.

TUBERCULOSIS

Tuberculosis was a major cause of morbidity and mortality from the late 1930s until the early 1960s.[27,32] In 1951 one-third of all deaths in Greenland were due to tuberculosis. By the early 1960s very few deaths from the disease were reported.[27] The reason for this dramatic decline was a national public health campaign against the disease.[27,32] Stein et al[32] correctly characterize the situation with regard to tuberculosis in Greenland prior to World War II when they say that control was hopeless due to ineffective treatment, poor diagnostic facilities, and the inability to isolate active cases. In 1949 the Danish authorities launched a BCG vaccination campaign aimed at all infants and children. This was followed by a BCG vaccination campaign for all newborns.[32] Complete coverage of the population was not easily achieved because people were widely dispersed in small settlements along the western coast. More complete coverage was not obtained until 1955 when an antituberculosis ship, the *Misigssut*, was put into service.[27,32] This ship was able to sail into most coastal settlements where x-ray examinations were performed and sputum specimens tested.

In addition to case finding and BCG vaccination, active cases were hospitalized at a tuberculosis sanatorium that was opened in Nuuk in 1954. Although it had a capacity of 211 beds, it was insufficient to handle the caseload.[32] Between 1951 and 1958 some 1,500 Greenlanders with tuberculosis were treated in Denmark.[32] A chemoprophylactic program was conducted in 1956 and 1957 using 400 mg/day of isoniazid.[32] On follow-up six years later, it was found that the incidence of tuberculosis was reduced 30% in the treated group.[33,34]

The impact of all these efforts was realized over time. Between 1955 and 1965, a total of 2,643 new cases of respiratory tuberculosis were reported in a population that then averaged 27,400. In 1955 the incidence was 23.2/1,000 (526 new cases) and by 1964 it had fallen to 2.7/1,000 (85 new cases). In 1956 incidence rates were lowest for children and higher in older age groups. The 1964 data show much the same pattern.[32] Between 1955 and 1965 the annual incidence of tuberculosis fell by 90%.[32] Stein et al[32] conclude that BCG vaccination played a minor role as it was primarily limited to children. They also conclude that chemoprophylaxis, which was limited to only half of the adult population, had played a minor role. It appears that effective case finding and therapy greatly reduced transmission.[32,35]

HEPATITIS

Both viral hepatitis A and viral hepatitis B have been extensively studied in Greenland by Skinhoj and colleagues and others.[36-41] All forms of hepatitis have been reportable diseases in the country since 1950. From historical records it is clear that viral hepatitis has been in Greenland for many years. Although the etiology is unknown, epidemics of jaundice were described in 1782 and 1829.[36] Geographically localized outbreaks in the 20th century occurred in 1913, 1926, 1934, and notably in 1947–1948.[36] The 1947–1948 epidemic of hepatitis A primarily affected the west coast of the country and left in its wake large numbers of immune individuals. The next epidemic of hepatitis A occurred in 1970–1974 and was well studied by Skinhoj et al.[36] A total of 4,961 cases were reported in this epidemic, affecting 11% of the population (Table XI).[36] The case fatality rate was 0.3%, the clinical course mild, and the fecal-oral route the principal mode of transmission.[36] Ninety-three percent of the cases occurred in individuals 1–25 years of age, indicating widespread immunity among adults, probably resulting from the 1947–1948 epidemic.[36] This was confirmed by the fact that the prevalence rates of antibody to hepatitis A antigen in sera of healthy Greenlanders in 1970 ranged from 100% for those 35 to 56 years of age, to 90% for those 24 to 34 years of age, to only 3% for those 12 to 22 years of age.[36] The true incidence of the 1970–1974 epidemic is difficult to arrive at because gammaglobulin was extensively used at the time in an effort to control the disease.[39]

Prior to the 1970–1974 epidemic the yearly incidence of viral hepatitis averaged 125 cases per 100,000 population.[39] By comparison the rate for the United States for the same period was 30 per 100,000 population.[42] Careful epidemiologic and clinical observations made during the 1970–1974 epidemic in Greenland demonstrated that there was no correlation between epidemic incidence rates by districts and endemic rates.[36] This, coupled with the observation that few cases occurred among adults over 25 years of age, supported the conclusion that the epidemic was due to hepatitis A. Radioimmunoassays of cases revealed antibody to the hepatitis A virus.[36] These findings also indicate that immunity from the previous 1947–1948 epidemic had lasted for a quarter of a century. The hepatitis A virus, unlike the hepatitis B virus, was shown to have a less predictable existence in Greenland and seems to cause explosive epidemics after being introduced from the outside.

TABLE XI. Number of Cases and Incidence of Viral Hepatitis in Greenland, 1947–1974[37]

Years	No. of Cases	Incidence Rate per 100,000 Population
1947–1949	Large epidemic; no. of cases unknown	1,000
1951–1960	372	138
1961–1970	511	130
1971–1974	4,961	2,606

Transmission of hepatitis A was facilitated by increased population densities in west coast settlements and poor levels of sanitation and hygiene. During the long arctic winters people are forced indoors and often live in cramped quarters in conditions of poor personal and environmental hygiene.[20] For most of this century, the epidemiology of hepatitis A in Greenland has been characterized by large scale epidemics occurring at intervals of 15 to 25 years.

The epidemiologic pattern of hepatitis B in Greenland has been one of endemicity. Clinically diagnosed cases are sporadic, predominantly among adults, and lack seasonal fluctuations.[39] In 1974, Skinhoj et al[39] published prevalence rates for HB-Ag in the sera of 2,904 native Greenlanders who had no signs of apparent liver disease. Overall 7.1% had HB-Ag. Prevalence rates varied from 1.2% in the more developed southwest of the country to 12.0% in the isolated eastern region.[39] These rates correlated with socioeconomic and housing conditions and inversely correlated with the reported incidence of hepatitis.[39]

The authors of this study concluded that the prevalence of healthy HB-Ag carriers was determined by the extent of exposure in early childhood, facilitated by overcrowded housing conditions.[39] Clinical hepatitis B appeared to be more prevalent in areas with better living conditions, where more people were susceptible to infection in adult life.[39]

In 1977 Skinhoj published the results of a more extensive survey of prevalence of hepatitis B infection.[40] A total of 1,450 sera from the east, northwest, and southwest were extensively studied for HB_sAg, anti-HB_s, anti-HB_c, HB_eAg, and anti-HB_e. Overall 16.6% were HB_sAg positive and 45.8% had anti-HB_s antibody.[40] The highest rates of HB_sAg (25.4%) and anti-HB_s (55.7%) were in the east (Angmagssalik).[40] The lowest rates of HB_sAg (7.3%) and anti-HB_s (39.2%) were found in the better developed southwest.[40] Skinhoj concluded from this data that the HB_sAg was hyperendemic in certain rural areas of Greenland such as Angmagssalik where socioeconomic and hygienic conditions were poor. He postulated that this situation led to subclinical infection and/or immunization at an early age, resulting in a high frequency of HB_sAg carriers in adults.[37] In the better developed communities of the west coast there were fewer HB_sAg carriers and more adults susceptible to clinical hepatitis.[37] Overall, HB_sAg prevalence in Greenland was 11.4% compared to 6.42% for Eskimos in Alaska and 4.2% for those in Canada.[43-45]

In interpreting their data on the HB_sAg carrier state, one must remember that the carrier state is not static and that each year a certain proportion of positives will become negative. Of some concern is the prevalence rates for HB_sAg for women of childbearing age since they can transmit the disease to their offspring vertically and not just horizontally. Skinhoj has published more recent data that show that among 62 HB_sAg carriers studied, 15 (24%) were HB_eAg positive and 41 (66%) were anti-HB_e positive.[43]

Given the high prevalence rates for HB_sAg in Greenland and the association of the virus with primary liver cancer (PLC), it is remarkable that so few cases of the latter have been documented in the country.[38] Skinhoj et al[38] found in the retrospective study that from 1951 to 1974 there were only 31 reported deaths from cirrhosis in Greenland (Table XII). During the period 1950–1976 there were only 12 reported cases of PLC in native Greenlanders that were histologically verified.[38] The youngest patient was a three-year-old boy and the oldest a 76-year-old woman. The male-to-female ratio for PLC was 1:1 and no clustering of cases by village or family was found.[38] Six of the cases were hepatocellular carcinomas, three were cholangiocellular carcinomas, one was combined, and two were hepatoblastomas.[38] The age specific incidence rates for PLC by sex do not differ for the period 1960–1975 from 1968–1972 data from Denmark.[38] However, age adjusted incidence rates demonstrated a sharp rise from 1.4 for females in 1950–1959 to 7.1 in 1970–1975 and from zero for males in 1950–1959 to 6.3 in 1970–1975.[38]

These data must be viewed with a certain degree of caution. The study was retrospective in nature and the number of histologic specimens few.[38] Death certificate information about cirrhosis was also insufficient for coming to definitive conclusions about etiologic types.[38] Alcoholic cirrhosis may predominate because of widespread alcohol abuse in Greenland where annual consumption is 18.7 liters per adult compared to 11.8 liters in Denmark.[38] Yet health care facilities in Greenland were good during the period under study. During the latter 15 years of the 25 years covered by the study all patients with malignant disease came to medical attention at hospitals and most were referred to either the Queen Ingrid Hospital in Nuuk (founded in 1957) or else to Denmark. The steep rise in PLC cases over time may be due to better detection and reporting. Overall the incidence of PLC is low, no higher than in Europe, where the prevalence of hepatitis B infection at the time was tenfold less.[46] Skinhoj et al conclude that among native Greenlanders, hepatitis B infection per se does not significantly contribute to the development of either cirrhosis or PLC.[38,43]

OTHER INFECTIOUS DISEASES

Infectious diseases are routinely reported to the Chief Medical Officer for Greenland from the 165 public health districts in the country. Table XIII presents the leading causes of death from infectious disease for the ten-year period, 1971–1980. During this period, 3,210 deaths were reported in the country, of which 529 (16%) were due to infectious diseases. Bjerregaard[47] carefully analyzed these data to see if they correlated with a variety of socioeconomic variables, especially housing. He found that average space per inhabitant ranged from 8 to 17 square meters compared to 43 in Denmark. Districts along the southwest and middle west coast had the best housing conditions. Those farther north, around Disko Bay, were intermediate, while the hunting districts in the northeast had the poorest housing conditions. Mortality from infectious diseases per 1,000 inhabitants was found to range from 0.66 in Nuuk, the capital city, to 21.12 in Upernavik in the northwest[48] and 3.20 in Angmagssalik in the east. The latter is an isolated area on the east coast which is almost permanently closed in by ice.[20] There was a direct linear association between higher rates of death from infectious diseases and housing density.[47] The exception for this was deaths from respiratory infections.[47] High rates of housing density correlate with socioeconomic status in Greenland and tend to be regional due to the regional nature of the country's rapid economic development. The southwest and middle west coasts, where the mainstay of the economy, deep sea fishing, is centered, have far better housing, social services, etc, and lower death rates for most infectious diseases. The northwest and the east, remote and out of the economic mainstream, are less favored by this development. There large numbers of people still exist by hunting. These regional variations in death rates for infectious diseases seem to be determined to some extent by socioeconomic variables. Similar variations have also been observed for infant mortality.[49]

TABLE XII. Reported Deaths from Cirrhosis in Greenland, 1951–1975[38]

Years	No. of Deaths Reported	Mortality Rate per 100,000 Population
1951–1955	2	1.60
1956–1960	6	4.00
1961–1965	4	2.22
1966–1970	8	3.81
1971–1975	11	4.73
Total	31	3.28

Herpes simplex and cytomegalovirus infections seem to be acquired in the first five to six years of life, by which time 75% of the population possess antibodies.[43] By one year of age 75% of Greenlanders have antibodies to Epstein-Barr virus and by three years of age all are infected.[43] Varicella is in a stable state of endemicity in Greenland.[43]

The relationship of herpes simplex virus (HSV) infection to cervical carcinoma is controversial. However, the rate of cervical carcinoma in Greenland is significantly higher than in Denmark.[43] The reasons for this increased rate may be multiple and cannot be explained only by rates of HSV infection. Human cases of echinococcosis have not been reported in many years and trichinosis outbreaks have been sporadic. The latter are usually due to the consumption of inadequately cooked bear meat. Yet a 1970 outbreak in Upernavik, involving 56 persons, was traced to the consumption of walrus meat.[21] Infectious mononucleosis is primarily a disease of the nonindigenous population.[50,51] Rabies is relatively rare.[52]

TABLE XIII. Leading Causes of Death Due to Infectious Diseases, Greenland, 1971–1980[47]

Disease	Number of Cases	Percent of Total
Pneumonia	226	43.7
Bronchitis, emphysema, and asthma	115	21.7
Influenza	57	10.9
Tuberculosis	50	9.4
Meningitis (plus meningococcal infection)	24	4.5
Enteritis	11	2.0
Measles	6	1.1
Other	40	7.5
Total	529	100.0

FIGURE 3. Greenlandic children, Paamiut.

Chronic otitis media has long been a significant cause of morbidity.[21] However, precise data about its true incidence are lacking due to the fact that it is not reportable. The impression of many medical authorities is that it is more common in southern Greenland where it is relatively more humid.[20,21] Kristensen, in a survey whose results were published in 1975, estimated that chronic otitis media and tonsillitis involved 10–12% of the population. He postulated that housing, nutrition, clothing, and other factors played a role in producing this rate since no correlation was found with temperature, relative humidity, and precipitation.[21]

More recently Pedersen and Zachau-Christiansen[53] studied acute, chronic, and secretory otitis media in Greenlandic children three to eight years of age. In a study of 142 such children they found that 75% had a previous history of otitis. Many of these children had a history of repeated episodes. Six percent of the children examined had suppurative, chronic otitis and 13% had the sequelae of chronic otitis.[53] Twenty-six percent had reduced middle ear pressure.[53] These figures far exceed those for Danish children. A number of factors have been put forth to explain the high prevalence rates for otitis in Greenlanders. These include the climate, crowded housing conditions with poor ventilation, diet, and socioeconomic variables.[53] Pedersen and Zachau-Christiansen[53] found that children of unemployed mothers from the lower socioeconomic strata had the highest risk of middle ear disease. The long term risk of chronic otitis, hearing loss, is a major concern in Greenland.[53]

SEXUALLY TRANSMITTED DISEASES

Sexually transmitted diseases continue to be major causes of morbidity in Greenland, with several thousand cases of gonorrhea and several hundred of syphilis being annually recorded.[6,20,21,54] Premarital sexual activity among the young and sexual norms that lead to early age for first coitus and multiple partners play a role. Yet sexual norms among Greenlanders and Scandinavians do not seem to differ much.[55]

Gonorrhea. Gonorrhea was first recognized in Greenland in 1864.[54] Until 1913 only a few sporadic cases were diagnosed. Thereafter until 1940 annual incidence varied. Since 1945, prevalence has been higher in Greenland than in Denmark.[54] During the 1960s and 1970s incidence increased sharply, especially among young girls 16–19 years of age.[54] Some 57% of infected teenagers were reinfected within the year, and 31% of the girls and 11% of the boys were treated more than twice a year for gonorrhea.[54]

Mardh et al[56] in a prevalence study of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* infections found that in an outpatient clinic in Nuuk where 262 patients were studied, 92% of females and 70% of males had hemagglutinin antibodies to gonococcal pili; among 63 controls, 30% of males and 10% of females were positive. In the settlement of Uvkusigsat where 150 subjects were tested, 41% of females and 33% of males were positive.[56] Gonorrhea continues to be a serious problem throughout Greenland.[57]

Syphilis. Greenland was surprisingly free of syphilis according to From[54] until the 1970s. Even though occasional outbreaks occurred in 1872, 1947, and 1965 in the south, the country's isolation and access only by ship from Denmark accounted for this state of affairs.[54] Compulsory medical screening of all ship crews and the length of the voyage insured that the incubation period was passed by arrival time. Infected individuals were quarantined.[54] Once the country was opened up, however, the incidence rose, especially in the 1970s.[54] Data for this period show a high prevalence among girls 13–19 years of age.[54] A decline in annual syphilis incidence occurred after 1977.[54] Much of this decline is traceable to early diagnosis and treatment of cases and contacts, thus reducing the size of the pool of infectious persons. Petersen et al[58] carefully studied the pattern of syphilis in Greenland in 1979. They found that of the 294 cases of reported early syphilis, 267 (91%) were diagnosed soon after exposure.[58] This pattern of early syphilis is due to early diagnosis and the extensive routine use of serologic testing of the population.[58] Petersen et al[58] found that the ratio of primary to secondary syphilis was 5:0 compared to 0:9 for Denmark.

Chancroid. In 1977, a major epidemic of chancroid occurred in Greenland.[54] According to From[54] the epidemic did not affect the northern and eastern areas because of long travel times. A total of 975 cases were reported, with large numbers of cases being observed in girls 13–19 years of age.[54] Only 488 cases occurred in 1978, and public health education coupled with the painful nature of the disease caused a steep decline in incidence.[54]

OTHER SEXUALLY TRANSMITTED DISEASES

As of the early 1980s, lymphogranuloma venereum had not yet been encountered.[54] However, evidence of *Chlamydia trachomatis* infection was found in 79% of females and 26% of males attending a Nuuk clinic.[56] Only 12% of females and 5% of male controls showed evidence of infection.[56] In the settlement of Uvkusigsat, 41% of females and 33% of males were positive on testing with a microimmunofluorescence test.[56] Nongonococcal urethritis, genital herpes, genital warts, and scabies are frequently reported in Greenland.[54] Reiter syndrome has been well documented in Greenland by Bardin et al.[59,60] Using 1983 hospital records, these authors found 51 patients with Reiter syndrome in the Egedesminde and Jakobshavor districts.[59] This represented prevalence rates of 1.08% and 0.34% respectively.[59] A review of prevalence and incidence between 1960 and 1983 showed a marked increase over time.[59] Endemic Reiter syndrome is often a post-venereal disease and is strongly associated with the HLA antigen B27 which is phenotypically found in 27% of native Greenlanders compared to 10% of Europeans.[59]

Acquired Immunodeficiency Syndrome. As of July 1987 there were no reported cases of acquired immunodeficiency syndrome (AIDS) in Greenland.[20] However, Pedersen et al[61] reported that as of early 1987 there were three known human immunodeficiency virus (HIV) positive Greenlandic males in Copenhagen who had apparently contracted the infection there. Pedersen et al tested 1,639 serum samples collected in Greenland in January 1986 for antibody to HIV. Of the 1,639 samples, 677 were from men, 893 from women, with a median age range of 31 years. The samples, which came from all major towns and villages in Greenland, were tested by the enzyme linked immunosorbent assay (ELISA), Western blot, and an immunofluorescence test.[61] Thirty (1.9%), including those of two women, were reactive to the Western blot.[61] Further samples from the two women were negative. All samples were negative on immunofluorescence. Of the total group of 1,639, 18 (1%) had active syphilis and 271 (17%) had previously been tested for the disease. Although Pedersen et al[61] did not find any HIV-positive people at that time they expressed concern about the eventual introduction of AIDS into Greenland given its close contacts with Denmark, which relative to other European countries has a high annual incidence.[61]

As Pedersen and colleagues had feared, HIV has now been introduced into Greenland. As of early February 1988, there were seven HIV-infected men in Greenland, all of them either homosexual or bisexual. Five were infected in Denmark and two in Greenland. None of the female contacts of the bisexual men had been found to be HIV-positive as of February 1988 (personal communication, N.S. Pedersen, February 3, 1988).

The relatively high rates of sexually transmitted diseases in Greenland coupled with multiplicity of sexual partners and frequency of sexual contacts create conditions similar to those in sub-Sahara Africa, where heterosexual transmission has become the prevalent mode of spread.[61] Greenland's current strategy for preventing AIDS includes the testing of all pregnant women and of everyone who has contact with the health care system. Since most Greenlanders have regular contact with health care facilities, this should prove to be successful. The aim is to test all adults every six months and to undertake contact tracing of those who are positive (personal communication, N.S. Pedersen, February 3, 1988). If these measures are successfully implemented, transmission of the disease may be effectively reduced. There is a high level of awareness of the disease in Greenland and local bookstores carry a number of popular books in Danish on the subject.[20]

MALIGNANT NEOPLASMS

Malignant neoplasms (Table XIV) are a leading cause of mortality in Greenland, accounting for 138.7 deaths per 100,000 per year.[2,20] Nielsen and Hansen have carefully studied the epidemiologic patterns of cancer in Greenland for various periods extending back to 1955.[62,63] An examination of the data for the nine-year period 1975–1983 revealed a total of 574 cancers reported among native Greenlanders, with much the same pattern observed between 1965 and 1974. Between 1965 and 1974 a total of 486 cancers (188 in males; 298 in females) were diagnosed, giving a significantly low observed/expected ratio for males of 0.8. The ratio for females was 1.1 and not significant. Between 1975 and 1983, 574 cancers (233 in males, 341 in females) were observed, for a significantly low observed/expected ratio in males of 0.8 and a nonsignificant ratio in females of 1.0.[62] These ratios, based on expected incidence in Denmark, represent relative risk.[62] Overall, the total cancer incidence in Greenland was significantly lower than in Denmark for males but not for females.[62] When compared to the Danish population, the risks for Greenlanders were higher for a number of anatomical sites, including nasopharynx, salivary glands, and esophagus in both males and females and cervix and lung in females. Significantly lower rates were found in Greenland for male bladder and rectal cancer, prostate cancer, and cancer of the testes. For females the rates were lower compared to Denmark for endometrial and breast cancer.[62] A low incidence of Hodgkin disease makes diseases of the combined lymphatic and hematopoietic systems less frequent compared to rates for Denmark.[62] The risks for the remaining cancers, analyzed by sex, did not significantly differ from those for Denmark.[62] Again, primary liver cancer was not found to be frequent, despite the epidemic nature of hepatitis B infection, yet in Alaska and Canada, where hepatitis B infection is also endemic, the incidence of primary liver cancer is high.[62]

Adjusted rates for two periods studied by Nielsen and Hansen revealed increased rates of lung cancer in both

TABLE XIV. Malignant Neoplasms Reported in Greenland for the Periods 1965–1974 (a) and 1973–1977 (b) by Anatomical Site[62]

Site		Number Males	Females	Total
Lung	a	34	15	49
	b	57	27	84
Cervix	a	—	70	70
	b	—	100	100
Esophagus	a	15	9	24
	b	20	13	33
Nasopharynx	a	14	9	23
	b	13	13	26
Salivary glands	a	5	12	17
	b	4	7	11
Breast	a	—	32	32
	b	—	38	38
Urinary bladder	a	6	3	9
	b	9	6	15
Rectum	a	10	5	15
	b	6	11	17
Prostate	a	1	—	1
	b	2	—	2
Testes	a	1	—	1
	b	5	—	5
Endometrium	a	—	6	6
	b	—	8	8
Lymphatic and hematopoietic systems	a	7	9	16
	b	11	9	20

men and women in the second period (1975–1983).[62,63] This is most pronounced among women, where the rate is 21.4/100,000 compared to a rate of 15.9 in Denmark. Cigarette smoking is widespread among native Greenlandic women at present.[20] The cervical cancer rate was found to be 64.2/100,000, four times that of Denmark which is 16.8/100,000.[62]

The pattern of neoplastic disease has recently changed in the western and central Canadian Arctic.[64] Hildes and Schaefer analyzed data on 239 verified cases of malignant disease diagnosed between January 1950 and December 1980 in 104 male and 135 female Inuit from the western and central Canadian Arctic.[64] They found that tumors of the salivary glands, kidney, and nasopharynx were most frequent between 1950 and 1966, but that their frequency then declined.[64] The commonest tumors in the most recent period were lung, cervical, and colorectal cancers.[64] Breast cancer was absent before 1966 and was found in only two of 107 Canadian Inuit women stricken with cancer from 1967 to 1980.

Between 1965 and 1983, 70 cases of breast cancer were diagnosed among native Greenlandic women.[62] The breast cancer rate for these women was 27.1/100,000 population between 1975 and 1983, having decreased from 31.5/100,000 for the 1970–1974 period.[62] The relative risk of breast cancer for Greenlandic women compared to Danish women has been 0.5 for the period 1965–1983.[62] However, compared to Inuit women in the western and central Canadian Arctic, Greenlandic women show a breast cancer pattern at present that is moving in the direction of that of women in western industrialized countries.[64]

Hildes and Schaefer[64] conclude that the gradual reduction in the relative frequency of tumors typical for traditional Inuit in Canada and their replacement by "modern tumors" appear to reflect various factors in acculturation including nutritional habits, life-style, and the environment.

Despite the westernization of Greenlandic society, with its attendant changes in diet, life-style, and environment, most of the traditional patterns of malignancies have remained unchanged, except for lung and cervical cancer.[62]

The incidence of nasopharyngeal carcinoma (NPC) among native Greenlanders is one of the highest in the world.[65] Nielsen et al[65] found it to be 12.3/100,000 for males and 8.5/100,000 for females in a study conducted in the late 1970s. During the decade 1965–1974, 23 cases were reported in Greenland, and from 1973 to 1977 a total of 26 cases were reported.[62] Salmundsen et al[66] closely studied the tumors of ten patients with NPC who were evacuated to Denmark for medical treatment.[66] Histopathologic examination of all ten tumors revealed them to be undifferentiated carcinomas.[66] Salivary gland carcinoma (SGC) also has a high incidence among native Greenlanders, 3.9/100,000 for males and 7.7/100,000 for females.[67] Most of these tumors (over 90%) are located in the parotid gland and at the histopathologic level are indistinguishable from undifferentiated NPC.[67] Nielsen et al[67] found that over a 20-year period, 92% of these tumors were undifferentiated carcinomas. Salmundsen et al[66] examined biopsy specimens from ten native Greenlanders with nasopharyngeal carcinoma and from three native Greenlanders with salivary gland carcinoma for the presence of Epstein-Barr virus (EBV) DNA and also examined sera from these patients for EBV-specific antibody titers. Among seven patients whose tumors were tested for EBV DNA, six were positive. The tumor of one patient with SGC was also positive for EBV DNA.[66] The EBV-specific antibody spectra and titers of the patients with NPC or SGC conformed to results obtained in other high incidence areas.[66] Salmundsen et al[66] concluded that their results lent further support to an etiological role for EBV in NPC. More recent studies have shown early infection with Epstein-Barr virus in Greenlandic children.[68,69] Merrick et al[70] have reported on familial clustering of salivary gland carcinoma in Greenland.

Hubert et al[71] recently published the results of a field study conducted in Greenland and Tunisia and among Chinese from Canton that attempted to correlate NPC with environmental factors and dietary habits. Using an anthropologic approach, these investigators surveyed 20 Greenlandic families in which NPC had occurred.[71] The sample consisted of families in which NPC had occurred among 11 men and 9 women. The families were widely dispersed over the southern and western coasts of Greenland.[71] Very detailed interviews in which a wide range of information was collected were conducted over a period of two months. This included a detailed family history and genealogy, history of disease, life history, data on past and present environment, housing, professions, etc, observations on diet and foods including descriptions of cooking areas and fuels used, observations on conditions of personal and environmental hygiene, and many other types of information.[71]

Hubert et al found that 15 of the 20 families were from small villages and that ten of them had subsequently moved to towns. Eighteen of the 20 came from hunting/fishing families and family members had had a traditional lifestyle in their youth. These families had all eaten traditional foods including fresh and dried meat of sea mammals, reindeer (in the midwest coast), and sheep (in the south). The consumption of sea mammals was a common factor, as was the consumption of dried fish. Fermented meat and fish were also consumed. As children all had lived in crowded, unventilated buildings in conditions of poor hygiene. These authors concluded on the basis of their studies among all three groups (Chinese, Tunisians, and Greenlanders) that traditional food preservation techniques including drying and fermentation were common to all of them. They recommended a closer examination of these methods in order to search for possible mycotoxins or other oncogenic or EBV-reactivating factors.[71] Compared to the other two groups studied, Greenlanders showed a high proportion of family aggregation for NPC and SGC (7 out of 20).[71] More recent reports have detailed the levels of volatile nitrosamine in foods in these three areas, and reported on space-time clustering of NPC in Greenland.[72]

SUICIDE, ACCIDENTS, VIOLENCE, AND ALCOHOL ABUSE

Greenlanders have one of the highest current suicide rates in the world (62.7/100,000).[2,20] There are regional variations in this rate, one of the highest, 124.9/100,000

(1976–1980), being in Ammassalik in eastern Greenland.[28] This latter figure is truly an astounding one in that it is more than twice the rate reported for any other country in the world.[2] Many detailed ethnographic studies of traditional Eskimo cultures have documented that suicide was well established before the arrival of western influences. While there were local differences in the motivations and methods used, suicide was generally the result of unbearable physical or mental suffering, and the sense of being a useless burden to the rest of the group. Suicide was undertaken only after very careful consideration during which family and group members were fully consulted and gave their assent. Not infrequently they would also give assistance in carrying out the actual act.[12(pp 194–195)] Thus suicide became institutionalized as an acceptable means by which unproductive members could lift a burden from their marginally surviving family and group. Within the context of traditional Greenlandic culture one must also be aware that both homicide and infanticide were socially acceptable under certain circumstances.[12 (pp 149–150)]

Freuchen recounts the story of a north Greenland woman, Itusarssuk, who killed four of her children during a hunger period in order to save them from starvation.[12(pp 149–150)] The oldest daughter helped her hang the three youngest and then put a string around her own neck so that the mother could pull it and fasten it to a hook.[12(p 150)] Freuchen recorded that Itusarssuk was highly respected for killing her four children because she had demonstrated that she loved them so much as to spare them further suffering.[12(p 150)]

Freuchen also vividly describes infanticide and the reasons for it.[12(pp 97–98)] Female infants were often strangled at birth or else left out to die from exposure.[12(p 97)] In a hunting society where men were the providers of food, this was considered a necessity. In old age, when people could no longer hunt, their only means of survival was to have sons who could provide for them. Female infants stood in the way of couples having sons because Eskimos weaned their young at three or four years of age, leading to a long period between pregnancies. Killing female infants at birth facilitated the rapid occurrence of the next pregnancy in the hopes that it would produce a male.[12(p 98)] This practice led to a scarcity of women in some Eskimo societies, and, not surprisingly, polyandry developed.[12(p 99),55(p 144)]

A number of writers described suicide in Greenland in the 19th century. Holm recorded that among the Angmagssalik Eskimo of eastern Greenland a hopelessly ill person suffering from a serious disease was encouraged to commit suicide, usually by jumping into the sea.[73] Knud Rasmussen,[74] the greatest authority on traditional Greenlandic society, recorded on his Fifth Thule Expedition that an old man with an incurable illness had hanged himself. The old man and his wife were Roman Catholics. They dealt with the prohibition against suicide by Roman Catholicism on the one hand and Eskimo tradition that sanctioned it on the other in a unique fashion. He had his wife strangle him with a thong, but just before he died she released it and later claimed he had died a natural death.

The registration of suicides in Greenland during the colonial period was neither systematic nor thorough, leading to underreporting.[75] It is likely that a number of accidents, including falls from high rocks and drownings in kayaks, were suicides.[75] With the spread of Christianity during the 19th century came strong moral condemnation of suicide. This may have led to a decline in rates. However, it is possible that it had either no effect or only a modest one, since many suicides could have been reported as either deaths from natural causes or else as accidents. Thus figures for suicide in the 19th century and the early part of this century are probably artificially low. In a survey published by Rink[76] in 1857, only three suicides were recorded from the early 18th century until then. These three suicides were among 4,770 registered deaths.[76] However, given what has been described above, these figures have little meaning.

In 1936 Borresen[77] published suicide data from the Nuuk area for the period 1738 to 1900 and found only two cases. Nansen[78] in 1891 reported that in 1884 suicide was virtually unknown in the Nuuk area, except among a few old and sick persons. Bertelsen[79] reported in 1935 that the overall frequency for suicide for the period 1891–1930 was 5/100,000, representing a total of only 14 suicides. Half of these occurred during the first three months of the year, and all cases occurred among psychologically disturbed individuals who shot themselves.[79] The first two reports from Nuuk are not necessarily representative of the rest of Greenland for the period. As the capital, Nuuk has been the center of western influence in Greenland. Rates from all three reports must be viewed in light of those factors that favored underreporting. Contrasting with these data is Peter Freuchen's observations that "...suicides are numerous among them."[12(p 194)] Freuchen was considered one of the greatest authorities on the Greenland Eskimos.

More recent studies of suicide in Greenland have been hampered by the fact that suicides have not been separately registered until recently.[75] Thus longitudinal comparisons of rates over time are misleading. In addition, failed suicidal attempts are not regularly registered. Despite these handicaps, Grove and Lynge[75] conducted a controlled study of suicides and suicide attempts in Nuuk in the years 1972 and 1973. There were 73 cases, 37 males and 36 females. In both years there were a total of 61 attempts and 12 completed suicides. At the time Nuuk had a population of 3,273 native-born Greenlandic adults. There were some 1,000 adult Danish males in the population as well at the time.[75] The rates recorded in this study were analyzed by age group (Table XV). The rates per 100,000 population were found to be extremely high among teenagers and young adults, a different age distri-

TABLE XV. Suicides and Suicide Attempts, Nuuk, Greenland, 1972 and 1973[75]

	No. of Cases per 100,000 Inhabitants per Year	
Age Group* (yrs)	Suicide Attempts	Suicides
15–19	1,124	300
20–24	1,993	173
25–39	950	226
40+	108	54
Total	932	183

* Males, N = 37; females, N = 38.

bution from what had usually been observed in traditional Eskimo society.[12(p 194)] A variety of means were used including shooting and jumping from heights (favored by men), drowning, exposure, suffocation, stabbing, and ingestion of pills (favored by women).[75] Of the 12 completed suicides, ten were by men and two by women. Among the 61 attempters, 24 (39%) had previously attempted suicide.[75]

Grove and Lynge found a strong association between suicidal behavior and alcohol ingestion.[75] Among 41 attempters and suicides in 1973, 23 (68%) were affected by alcohol problems.[75] They also looked at meteorologic influences on suicides in 1973 and found no correlation with precipitation, temperature, barometric pressure, and wind velocity.[75]

Grove and Lynge present data on the monthly distribution of suicides and attempts, counting as one instance several attempts made by the same person in one month, otherwise as one instance per month in question.[75] Of the total of 76 suicides and attempts calculated for 1972 and 1973, only 13 (17.10%) occurred during the four months of maximum daylight, May–August. By comparison, 36 (47.36%) occurred between September and December and 27 (35.52%) between January and April, the dark months of the year when there is little daylight.[75] These authors provide no interpretation of these data in their paper. Although they do not comment on it, their data show a significant clustering of cases during the dark months of the year, September through April, when there are few hours of light.[75] Only 13 of the 73 cases in 1972 and 1973 occurred between May and August, the months of maximum daylight.[75] They also looked at such factors as criminality and found that for the 41 cases in 1973, 75% of the men had criminal records.[75] They conclude that suicide in Greenland today occurs among a variegated group as regards motive, intent, and outcome. Greenlanders who attempt suicide come from homes with emotional disturbances and alcohol problems and frequently experience problems such as unemployment, frequent crisis and conflicts, alcohol abuse, and criminality. Neither they nor their support systems have the resources to resolve these problems.[75] The currently observed suicide pattern in Greenland is similar to that seen among Alaskan Eskimos and North American Indians.[75,80]

Robert-Lamblin[28] in studying suicide in Ammassalik in East Greenland found that the rates had risen from 19.7/100,000 in 1961–1965 to 124.9/100,000 in 1976–1980.[28] For the 1976–1980 period, suicides represented 11.4% of all deaths. Male suicide rates were four times higher than those of females and the 20–24-year-old age group was the most affected for both sexes.[28] Robert-Lamblin likens this and the increase in other violent deaths such as drowning, freezing to death, and unintentional homicide to alcohol abuse. She found that young adults in the depressive phase of alcoholic intoxication were prone to these violent acts.[28] Ammassalik in Eastern Greenland is a community of 2,500 Greenlanders and 200 Danes. It is an area of the country with a harsh climate compared to the west coast and one that is relatively isolated and thus more tied to the traditional way of Eskimo life.

Robert-Lamblin found high accident rates in Ammassalik. From 1959 to 1978, 64 deaths (21.3%) among males and 25 (20.4%) among females were due to accidents. In this traditional hunting society, men are at risk from ice covered seas, snow and wind storms, the capsizing of boats while at sea, and ice pack collapses. Men who leave the village to hunt and fish must face all these dangers. Children and teenagers are also frequent accident victims because they are often left unsupervised in their games and activities.[28] They are exposed to fires, guns, and toxic products kept in the home.[28]

Greenland has the highest rate of jaw fractures in the world.[81] A retrospective study of mandibular jaw fractures in Nuuk was published in 1981.[82] Thorn and Hansen undertook a prospective study of the incidence and etiological factors in Greenland from July 1, 1981, to December 31, 1982. They recorded a total of 129 jaw fractures, giving an annual incidence rate of 17/10,000 population, the highest in the world.[81] This is four times the rate reported in Scandinavian countries.[81] Over half of the patients were in the age group 20–29 years, the most commonly affected age group in other countries.[81] Females constituted 36% of patients. It has been found in other studies that where violence plays a role the proportion of women with jaw fractures is high.[83] In 112 cases (90%) violence was the cause.[81] The proportion of women with jaw fractures in Greenland is the highest yet recorded.[81] Only 2% of cases were due to traffic accidents, not surprising since traffic is sparse. In 125 cases (97%) the mandible was fractured, a usual outcome of interpersonal violence. Maxillary fractures, more often seen in traffic accident victims, were few (two cases). In 90 cases (79%) the victim was intoxicated.[81] The association between jaw fracture and intoxication has been previously documented. However, the level of the association in Greenland is the highest yet recorded.[81] Thorn and Hansen attribute this level in part to the deregulation of alcohol in Greenland which occurred on April 1, 1982, midway through the time frame of their prospective study.[81] The result was a doubling of the rate, 80 cases being recorded in the postderegulation period of nine months compared to 49 in the nine-month period before deregulation.[81]

Thorn and Hansen[81] found that jaw fractures most often occurred in towns and among native Greenlanders. Since the initial deregulation period jaw fracture incidence has fallen, but is still higher than in the pre-deregulation period.[81] Mandibular fractures were still a major cause of morbidity in 1987, the victims often being wives of intoxicated husbands.[84] The high level of violence in Greenland has been documented as well by a number of authors.[85-89]

Bjerregaard and Johansen[90] recently undertook a detailed analysis of mortality trends in Greenland from 1967 to 1983 using Potential Years of Life Lost (PYLL) before age 65 years as an indicator of premature death. They found among other things that PYLL from infectious diseases decreased over time while PYLL from violent deaths (suicide, accidents, homicide) increased and currently account for 65% of all PYLL.[90] PYLL rates from all violent causes were much higher in Greenland than in Denmark. Marine accidents are more frequent in Greenlandic settlements than in the towns due to the traditional hunting and fishing culture that dominates in the former. Suicide and homicide, which are three times as high in the towns as in the settlements, increased more than threefold between 1968 and 1983.[90] Because of the scarcity of motor vehicles in Greenland, PYLL from vehicular accidents are few.[90]

As evidenced by the data from studies of suicide, violence, and accidents, alcohol abuse has progressively become a major social and medical problem in Greenland. Per capita consumption of alcohol has significantly increased over time, from about 9 liters per year in 1952 to 18 liters in the 1980s.[75] With the deregulation of alcohol in Greenland in 1982, a wide variety of alcoholic beverages have become available. Grocery stores and supermarkets in most towns and settlements carry a large array of imported alcoholic beverages including wines, beer, and hard liquor.[20] In addition, locally brewed alcoholic beverages are also available.[20]

Although marijuana is used by some it is a relatively minor problem at present.[84] Cocaine and heroin use is practically unknown.[84] Alcohol continues to be the principal substance of abuse in Greenland and is associated with assaults, homicides, and suicides. Danish physicians at the Queen Ingrid Hospital in Nuuk estimate that close to half of all patients brought to the hospital for care come for problems that are in one way or another related to alcohol abuse.[84] Underlying alcohol abuse in Greenland are societal stresses that have emerged as the society has modernized and the traditional hunting and fishing culture has disappeared. Within some towns there are an impressive array of consumer goods available and great disparities in living standards and purchasing power. High unemployment rates among youth no doubt contribute to both alcohol abuse and suicide. Also symptomatic of unemployment is the high rate of burglary that has developed in the capital, Nuuk.[84]

CARDIOVASCULAR DISEASE AND DIABETES MELLITUS

Greenlanders subsisting on a traditional diet rich in marine animal fats have long been known to have virtually no ischemic heart disease.[91] Native Greenlanders do not have either maturity onset or juvenile insulin-dependent diabetes mellitus.[91,92] Harvald[91] makes a cogent observation when he notes that native Greenlanders constitute a population descended through 150–200 generations who have been living in isolation for close to 5,000 years.[91] Among west Greenlanders there is an estimated admixture of Caucasian genes to the population gene pool of about 30%.[91] Yet this level of Caucasian genes has not yet resulted in overt clinical diabetes mellitus.[91] This phenomenon and the low level of myocardial infarctions and the rarity of ischemic heart disease may be due to either genetic determinants, environmental ones, or both.[91] Diet alone cannot explain the absence of these diseases because the traditional diet of Greenlanders has been largely abandoned for many years in large towns such as Nuuk along the west coast.[20] The population of these towns have a life-style and diet almost identical to that of Danes, being high in sugars and dairy products. In addition, the west Greenlanders have high per capita rates of consumption of cigarettes and alcohol.[20] Despite the introduction of these risk factors, myocardial infarctions are very rare.[84,91]

A number of avenues of inquiry have been followed to explain these observations. Bang et al[93] studied the plasma lipid and lipoprotein patterns in west coast Greenlanders. The population studied had at the time a way of life and diet little changed from the late 19th century.[93] Their diet consisted of the meat of whales, seals, birds, and fish, foods extremely rich in protein and fat but poor in carbohydrates. These food sources are also rich in polyunsaturated fats. Bang et al[93] found much lower levels of pre-B-lipoprotein and consequently of plasma triglycerides compared to Danish controls. Greenlanders also had significantly lower cholesterol levels compared to Danish controls. These authors concluded that the low serum cholesterol levels observed were due to the large amounts of polyunsaturated fatty acids found in the fat of the animals eaten.[93] Low serum cholesterol and triglyceride levels were thought to partially explain the very low incidence of ischemic heart disease in Greenlanders.[93] However, Bang et al also allowed for the possibility that genetic determinants in addition to dietary ones played a role.[93,94]

It has long been observed that native Greenlanders exhibit a bleeding tendency and this observation led to another avenue of inquiry with regard to ischemic heart disease. The first recorded description of this bleeding tendency is found in the *Historia Norvegiae*, which dates to the 13th century.[95(p 14)] The author of this early saga wrote:

> On the other side, north of the Greenlanders [ie, the Norsemen of Greenland], hunters have found some very little people, whom they call Skraelings and who, when they are wounded with weapons while alive, die without loss of blood, but whose blood when they are dead, will not cease to flow.[95(pp 14-15)]

Hans Egede, the Danish missionary and trader who settled on the west coast of Greenland in 1721, observed, "They are very plethoric. Therefore they most often bleed from the nose."[96] Explorers, travelers, and physicians who worked in Greenland gave vivid descriptions of this bleeding tendency, describing epistaxis, hemoptysis, hematuria, postpartum bleeding, and submucosal bleeds during measles attacks.[30,97] These descriptions are found in the writings of observers from the 18th century up to the present time. Peter Freuchen, the renowned Danish arctic explorer, not only provided a clear description of the phenomenon, but also pinpointed the probable cause.

> An extremely frequent disorder. . . is the frequent nose-bleeds. There is scarcely one individual of the tribe whose nose will not bleed spontaneously at least every fourth or fifth day. . . The frequency of nose bleeding is probably connected with the nutrition which is exclusively of animal origin, because I myself, when living for longer periods as a hunter or traveling on expeditions, under which circumstances I never carry bread or other European provisions, get just as frequent nose bleeding. . .[98]

As early as 1856, Prosch expressed the view that hemoptysis among Eskimos was in some way ". . . connected with the fact that the Greenlanders food is of animal origin. The consumption of blubber may also be of importance."[97] Tuberculosis had become a major problem by the later 19th century, and was frequently associated with hemoptysis.[99] Hansen said:

> Tuberculosis in Greenland is far less fatal than one should expect. . . Especially in the bigger settlements the area outside

> the trading station is often stained by sanguinous expectoration in a highly grim way; but the Greenlanders walk about for several years in high spirits with their phthisis, and a serious hemoptysis.[99]

Postpartum hemorrhage was recorded as a serious problem as far back as 1896, and was well documented later in the 20th century.[97]

The bleeding diathesis exhibited by Greenlandic Eskimos perplexed early observers, a number of whom—such as Freuchen (1915),[98] Prosch (1856),[97] Rohleder (1923),[97] and Hoygaard (1940)[100]—attributed it to diet. It was tempting later on to ascribe the phenomenon to avitaminosis C. Scurvy had indeed been recorded among both Europeans and Eskimos in Greenland since the 18th century.[97] Yet scurvy was generally only seen during periods of starvation.[97] Hoygaard summed up the situation in 1937 when he observed, "...In the spring...bleeding from the nose was very common. This did not appear to have any connection with C-hypovitaminosis."[100] The clinical picture of hemorrhage diathesis clearly did not fit that of scurvy.

In the late 1970s, Bang and Dyerberg conducted a number of studies which elucidated the bleeding diathesis among native Greenlanders and which provided possible explanations for the observed low rates of ischemic heart disease.[101-105] They found that Greenlanders living on a traditional diet had low levels of arachidonic acid, but high levels of eicosapentaenoic acid, a 20-atom fatty acid found in arctic marine animals.[94,103] They showed that eicosapentaenoic acid cannot act as a precursor for any active proaggregatory thrombaxan.[104] Dyerberg et al demonstrated that an antiaggregatory prostacyclin, probably PGI_3, can be produced from eicosapentaenoic acid and that this prostacyclin is even as active as the antiaggregatory PGI_2 coming from arachidonic acid.[105]

This means that when the blood contains predominately arachidonic acid, as is the case in Europeans, both a proaggregatory prostaglandin (thrombaxan) and an antiaggregatory prostaglandin (prostacyclin) can be produced. Primary hemostasis seems to be controlled by the balance between the effect of the two. In native Greenlanders on a traditional diet, the dominating fatty acid in blood and platelets is eicosapentaenoic acid from which only antiaggregatory prostaglandin can be produced. Thus the balance is toward antiaggregation.[97] Bang and Dyerberg[97] claim that the resulting decreased platelet aggregability explains the bleeding tendency in native Greenlanders as well as the rarity of thrombosis and ischemic heart disease. Among 24 Greenland hunters, Dyerberg and Bang[102] found a marked decrease of adenosine diphosphate-induced secondary platelet aggregation as well as a doubled bleeding time as compared with Danes of the same age and sex.

The demonstrated role of eicosapentaenoic acid in the prevention of acute myocardial infarction has recently been commercialized in the United States with the marketing of tablets and suspensions containing the acid. During 1987 and 1988, commercials on US television advertising such a product showed a native Greenlander in traditional dress (rarely worn today)[20] paddling a kayak (rarely used today) and later presenting a fish to his family.

Some postulate that the traditional Eskimo diet, rich in polyunsaturated fatty acids and low in carbohydrates, is only part of the explanation. An elevated antithrombin III level has been demonstrated in Eskimos, which may be either a genetic trait or a result of polyunsaturated fatty acid intake. The C3F genotype of the complement system associated with a high susceptibility to myocardial infarction is rare in Greenlanders.[106] Thus it appears that the rarity of ischemic heart disease is in part due to the antithrombotic effect of long-chained polyunsaturated fatty acids, especially eicosapentaenoic acid prevalent in diets rich in marine oils.[101] However, this is not the only reason, as genetic and other factors may play a role as well.[91] More recently, Jorgensen et al[107] have demonstrated that antiaggregatory prostamoids predominate in Eskimos compared to Danes.

OTHER DISEASES

As previously described, the traditional Greenland diet was rich in fish and arctic marine protein and fats. This diet is low in calcium and high in magnesium. The low serum calcium levels documented in Greenland are thought to be due to low dietary intake and not to vitamin D deficiency.[108] Similarly, the higher serum magnesium levels seen in Greenlanders are thought to be related to a diet rich in fish and sea mammals.[109] The low calcium levels are thought to be causally related to early onset and rapid age-related bone loss in adult Eskimos.[110,111] Osteoporosis with a high frequency of collapsed and wedge-shaped vertebrae are common in Eskimo populations.[108]

Greenlanders have a low urinary calcium/magnesium ratio which theoretically should lower the risk of stone formation.[112] Using hospital data, Jeppesen and Harvald[112] compared rates of urinary calculi between Danes and Greenlanders for the years 1973–1975 and found that males in Denmark had a rate of 2.0/1,000, double that of males in Greenland. For females the rates were 0.5/1,000 for Greenland and 1.0/1,000 for Denmark.[112] These data must be interpreted in light of the fact that they are hospital based. Differences may exist because of dissimilar admission practices and because the majority of patients with urinary calculi are generally not hospitalized. However, the number of hospital admissions in Greenland per population unit is double that of Denmark for all diagnoses. This implies that the observed differences may be far greater than the actual ones since most Danes with urinary calculi are not hospitalized whereas most Greenlanders are.[107]

A wide variety of other disease and health problems have been reported in Greenland. Among these are exposure to heavy metals such as lead and mercury,[113-118] skin cancer,[119] Behcet disease,[120] demodex folliculorum,[121] inguinal hernia,[122] sucrose deficiency,[123] cholestatic syndrome,[124] spondylolisthesis,[125] and dental problems.[126]

Blindness in Greenland has been extensively studied by Rosenberg.[127,128] In studying epidemiologic data from the Register for the Blind in Greenland, he has found high prevalence rates of blindness due to senile macular degeneration.[128] Prevalence rates varied from 5.7/1,000 for men 65–69 years of age to 180/1,000 in men aged 80–84 years.[128] As of January 1986, there were a total of 102 legally blind persons in Greenland.[128] Of this number, 91 were blind because of senile macular degeneration.[128] Glaucoma was the cause of blindness in eight individuals.[128]

Radioactive pollution at Thule in the north and its impact on cancer rates among Danes exposed there have been studied in detail.[129,130] Radioactive cesium-134 and 137 from the Chernobyl accident in April 1986 has been found as layered deposit in Greenland's polar glaciers.[131] This layer of radioactive cesium has provided a new reference for estimating snow accumulation rates and dating ice core samples.[131]

ENVIRONMENTAL SANITATION

Environmental sanitation poses special problems in the arctic climate. A site inspection of various parts of Greenland revealed that problems related to water supplies, solid waste disposal, and sewage continue to exist, despite some significant progress.[20,21] Because of geological conditions, ground water is generally not available in Greenland. All water must be obtained from surface sources such as reservoirs, fjords, lakes, and rivers. In a dozen towns and settlements year-round water supplies are provided through insulated or heated pipes and pumping stations to large buildings, hospitals, clinics, etc. Individual houses may be supplied all year (in Nuuk, for example) or for part of the year.[20] Water tank trucks make home deliveries of water, and in small settlements people fetch it themselves.[20] In some localities such as Upernavik ice blocks from glaciers are collected. Both glaciers and icebergs are composed of fresh water. Where municipal water supply systems exist, the water is treated by filtration, aeration, neutralization, and chlorination.[20]

Although great strides have been made in Greenland in providing potable water supplies, less success has been made with solid waste disposal and sewage treatment. In July 1987 the town of Nuuk began operation of an incinerator for the disposal of solid waste.[6] Outside visitors to Nuuk and to other towns in Greenland are struck by the amount of solid waste lying about.[6] With modernization and the availability of consumer goods, nonbiodegradable synthetic packaging has become a serious problem. Discarded cans and plastics litter the ground in numerous towns and settlements.[20] Even where municipal garbage collection services exist, people tend to throw litter wherever they happen to be. There may be a residual cultural element in this habit since in traditional Eskimo society discards, which were mainly biodegradable, were left on the floors of houses and igloos for the dogs to eat or else were tossed outside. There they were consumed by dogs and foxes. Similarly, human excrement was left on the floors of shelters and the dogs allowed in to eat it.[12(pp 58-59)] Thus refuse and human excrement were cycled out of sight because either they were eaten by dogs or else they underwent biodegradation. Most garbage today is not biodegradable and its volume is enormous compared to that generated when Greenlanders led a traditional way of life. Some towns such as Paamiut have litter baskets, but these are far too small and are not regularly emptied.[20] Large towns have garbage collection services with the garbage being taken to a surface dump. Despite this, people still tend to throw a great deal of garbage on the streets and around their homes. Haraldson in describing the west coast settlements said, "Enormous numbers of empty beer tins and glass bottles were found all over the west coast settlements surrounding each hamlet. In some places (Sarqaq) the contamination has reached such dimensions that it is doubtful if even very costly cleaning programs can restore nature."[21] Haraldson goes on to say: "A destruction on a larger scale, practically ruining a whole valley, is the iron and other scrap and burned barracks left behind by U.S. military forces in Narssarssuaq."[21] Around Camp Lloyd, a US Air Force facility at Kangerlussuaq, the rocks on otherwise pristine hillsides are painted with defacing graffiti, the vulgar legacy of US airmen who were once stationed there.[20] Since it never rains at this place, "Broadway 64-65," "Boise, Idaho," "Kansas," "Ohio," and all the initials will continue to deface nature for decades to come.

FIGURE 4. The town of Paamiut (Frederikshab), west coast of Greenland.

The disposal of sewage through sewerage systems has proven to be a significant problem because of the climate. In some towns raw sewage is piped out of homes to the ocean.[20,21] Other residences and buildings are equipped with chemical toilets which must be periodically emptied and cleaned. For example, chemical toilets are used at the Museum of Greenland, which is housed in modern buildings.[6] Public collections of the contents of chemical toilets are regularly conducted in towns. The contents are often dumped into open drums and the latter in turn discharged on the outskirts of the settlements.[20,21] In Nuuk closed tank trucks are used for collection and cleaning processes and a bucket change system is well established.[20,21]

On an extensive on-site health survey of Greenland in 1975, Haraldson found that of 656 residences constructed in 1972, 492 had toilets connected to discharge sewerage systems. Forty-eight had chemical toilets and the remainder had no toilets at all.[21]

All local communities in Greenland now have *sundhedskommission* (health boards) to oversee public health activities. These groups have attempted to grapple with the problems of potable water supplies and sewage and solid waste disposal in addition to a host of other issues. A number of these boards in conjunction with municipal authorities annually sponsor spring cleanup campaigns after the snow has melted.

The organization of health services in Greenland is presented in detail by Haraldson and more recently by the Department of Health of Greenland.[21,132]

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Cinchona and its alkaloids: 350 years

LEONARD JAN BRUCE-CHWATT, MD, MPH

The story of cinchona and its alkaloids is one of the most fascinating and romantic in medicine.[1-3] A number of imaginary or uncertain versions of the date and circumstances of the discovery of the therapeutic properties of the Peruvian "fever bark" exist, but not one of them can be confirmed by clear evidence.[3,4] The sentimental story told by Bado in 1663 that sometime in the 1630s the bark cured the Countess of Chinchón, first wife of the Viceroy of Peru, has now been disproved by several writers.[5-12] The most plausible and prosaic guess is that the medicinal effect of the bark of certain species of the Andean trees was discovered by the Spanish missionaries either by following the usual practice of local Amerindian herbalists or by a rational method of trial and error.[9-12]

The year 1638 represents the only exact date of the first printed record of the use of Peruvian or Jesuit's Bark for treatment of intermittent fevers or "agues." It was 350 years ago that a 1633 manuscript completed in Peru by an Augustinian missionary, Fray Antonio de la Calancha, was published in Barcelona. It described "the fever tree that grew in Loja, Peru, the bark of which, when made into a powder . . . and given as a beverage cures the tertian fevers . . . one hears of its miraculous effects in Lima . . ."[13]

EARLY USE IN EUROPE

Malignant fevers were so common in Europe in the 17th and 18th centuries that the powdered bark introduced by the Spanish Jesuits at the request of Cardinal Juan de Lugo became widely used under the names of *Pulvis Cardinalis*, *Pulvis Patrum*, etc. The value of this remedy was fiercely contested for some time, not only because of religious bigotry but because there were many worthless substitutes or adulterants of the genuine product.

The first written record of the use of the bark in England was in 1656 by John Metford of Northampton[14]; Richard Brady, Regius Professor of Physic in Cambridge, was also among the first who prescribed the bark. Thomas Willis, Richard Morton, and Thomas Sydenham used it, not without caution. Sydenham,[15] a staunch Puritan, was skeptical at first but became convinced of its value. The new remedy acquired wide acceptance through the courtesy of Robert Talbor[16] (or Tabor), an apprentice apothecary, who in 1672 cured King Charles II of persistent ague and was awarded a knighthood. He even received the king's protection from the "molestations and disturbance" of the Royal College of Physicians!

In 1677, "*Cortex Peruanus*" was listed in the third edition of the Pharmacopeia Londinensis. In Italy, Torti,[17] the great defender of the Peruvian bark, stressed its efficacy for treatment of true intermittent fevers and advocated its rational use for agues, which were regarded then as "opprobria medicorum."

The rising demand for the new remedy started a series of botanical explorations to the New World, one of the earliest being sponsored by the French Académie des Sciences. The 1753 expedition under Charles Marie de la Condamine[18] collected specimens of leaves, flowers, and seeds of the fever bark tree from Loja. It was Condamine who sent the specimens of the plant to Linnaeus, the great Swedish naturalist; the latter prepared the first botanical description of it. Wishing to immortalize the name of the first Countess of Chinchón, supposedly cured by the miraculous drug, Linnaeus gave the tree the generic name *Cinchona*. However, by a linguistic error, he misspelled her name and in his *Genera Plantarum* of 1742, left out

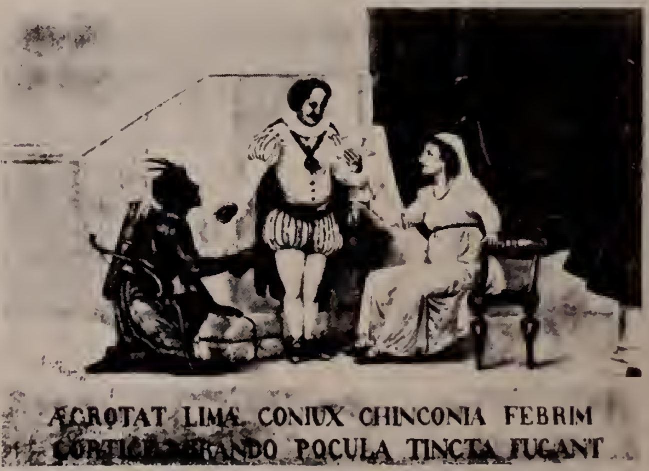
ÆGROTAT LIMÆ CONIUX CHINCONIA FEBRIM
CORTIC[illegible]RANDO POCULA TINCTA FUGANT

FIGURE 1. One of the frescoes at the Hospital Santo Spirito in Rome. It shows the imaginary scene of the Count of Chinchón receiving from an Amerindian chief the potion made of Peruvian bark for the ailing Countess.

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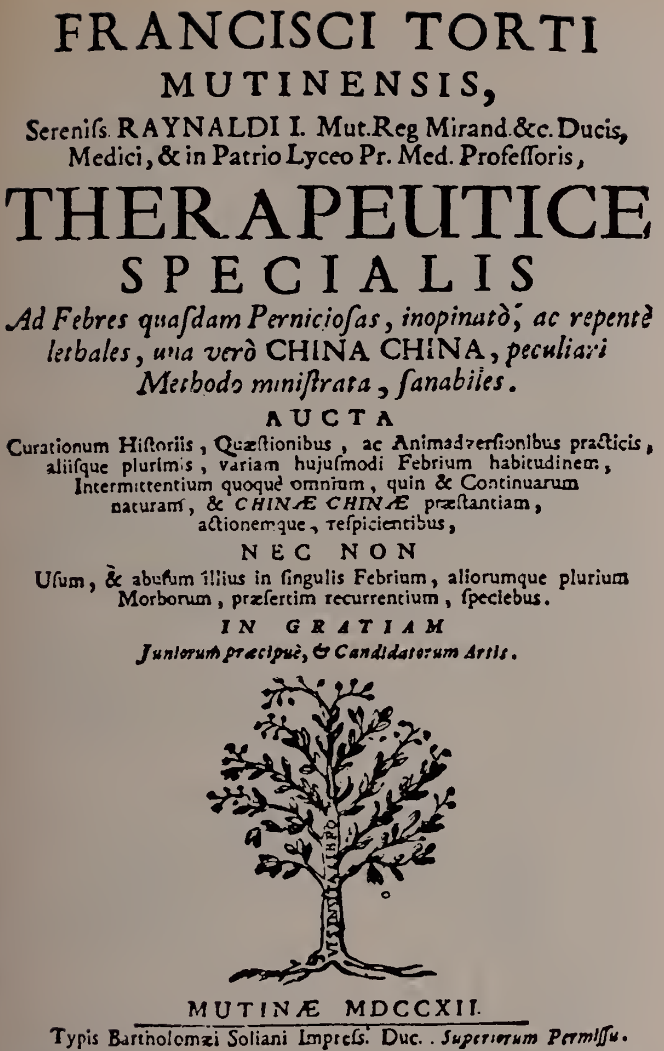
Based on a lecture given at the London School of Hygiene and Tropical Medicine on December 1, 1987, in commemoration of the 350th anniversary of the use of cinchona and quinine.



FRANCISCI TORTI
MUTINENSIS,
Sereniſs. RAYNALDI I. Mut.Reg Mirand.&c. Ducis,
Medici, & in Patrio Lyceo Pr. Med. Profeſſoris,
THERAPEUTICE
SPECIALIS
Ad Febres quaſdam Pernicioſas, inopinatò, ac repentè
lethales, una verò CHINA CHINA, *peculiari*
Methodo miniſtrata, ſanabiles.
AUCTA
Curationum Hiſtoriis, Quæſtionibus, ac Animadverſionibus practicis,
aliiſque plurimis, variam hujuſmodi Febrium habitudinem,
Intermittentium quoquè omnium, quin & Continuarum
naturam, & *CHINÆ CHINÆ* præſtantiam,
actionemque, reſpicientibus,
NEC NON
Uſum, & abuſum illius in ſingulis Febrium, aliorumque plurium
Morborum, præſertim recurrentium, ſpeciebus.
IN GRATIAM
Juniorum præcipuè, & Candidatorum Artis.

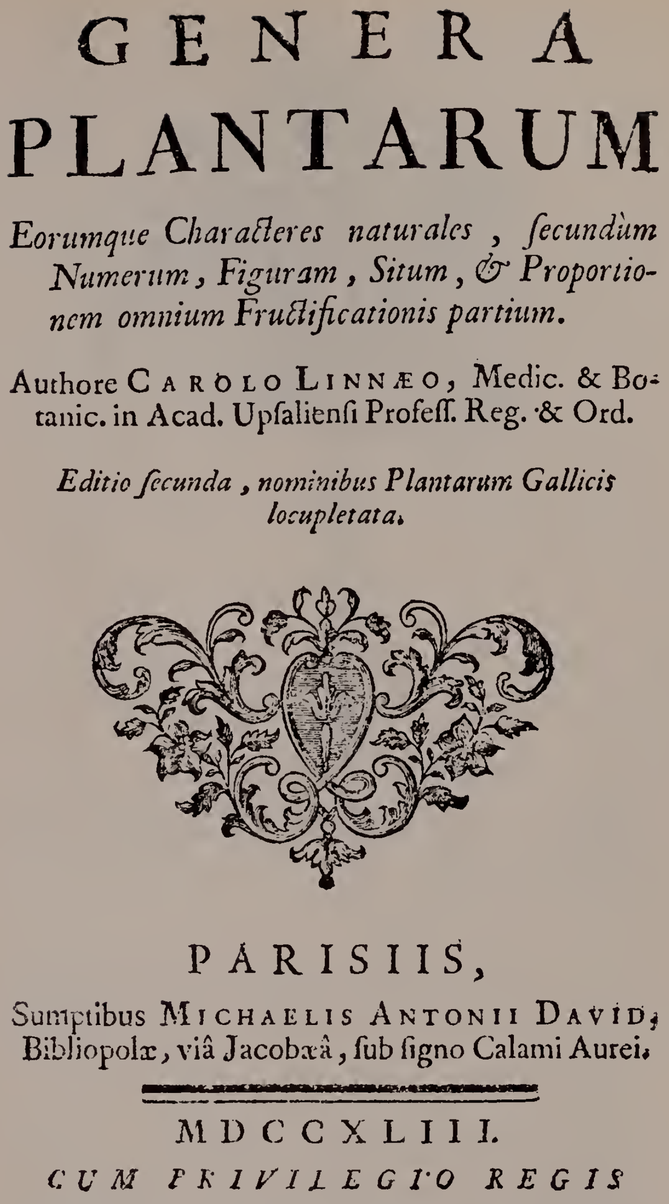
MUTINÆ MDCCXII.
Typis Bartholomæi Soliani Impreſſ. Duc. *Superiorum Permiſſu.*

FIGURE 2. Title page of Francesco Torti's 1712 book on the value and use of Peruvian bark, or Chinae China. Torti was a fervent advocate of the use of this remedy for treatment of genuine intermittent fevers.

the first *h* of the Chinchón family name.[6-8,19] Linnaeus repeated this error in his *Materia Medica* of 1749, but added to the description of the plant a common native name Quinquina, as used (wrongly) by La Condamine. Some attempts made a century later to correct Linnaeus' error were opposed, so the inaccurate name *Cinchona* is enshrined forever in the botanical nomenclature.

EARLY SOURCES OF CINCHONA

In the early 18th century, numerous botanical expeditions were arranged in search of sources of cinchona trees to satisfy the growing demands for the medicinal bark. In defining the characteristics of the best cinchona trees, the reports of many naturalists resulted in endless controversies. Much confusion was created concerning various species and relative values of many different plants.[20,21] The demand for cinchona bark increased still further after 1820, when two French chemists, Pierre Pelletier and Joseph Caventou,[22] isolated from samples of cinchona bark two different alkaloids, quinine and cinchonine; other alkaloids, including quinidine and cinchonidine, were soon separated from the complex mixture.



GENERA
PLANTARUM
Eorumque Characteres naturales, ſecundùm
Numerum, Figuram, Situm, & Proportionem omnium Fructificationis partium.
Authore CAROLO LINNÆO, Medic. & Botanic. in Acad. Upſalienſi Profeſſ. Reg. & Ord.

Editio ſecunda, nominibus Plantarum Gallicis
locupletata.

PARISIIS,
Sumptibus MICHAELIS ANTONII DAVID,
Bibliopolæ, viâ Jacobæâ, ſub ſigno Calami Aurei.

MDCCXLIII.
CUM PRIVILEGIO REGIS

FIGURE 3. Title page of the second edition of Carl Linnaeus' *Genera Plantarum* of 1743, in which the Peruvian bark tree was given the botanical name of *Cinchona*.

The ruthless exploitation of natural forests of cinchona threatened the world with rapid disappearance of the best species. Steps were taken to set up plantations in several parts of the world, particularly in India, Ceylon, and Dutch East Indies. In 1853, in great secrecy, the Dutch government sent Justus Hasskarl, a botanist, to Peru and Bolivia, with the aim of collecting the plants and seeds of *Cinchona calisaya*, which had previously been brought to Europe. However, when cultivated in Indonesia, these plants produced a bark of low quinine content, and in 1860 the attempt to establish vast plantations of these potentially valuable trees was given up.[21]

In 1859, the British government sent a party under Clements Markham, an explorer and geographer, to South America, with the aim of collecting seeds of the best cinchonas from the Andean forests for prospective plantations in Ceylon and India.[1,6-9,20]

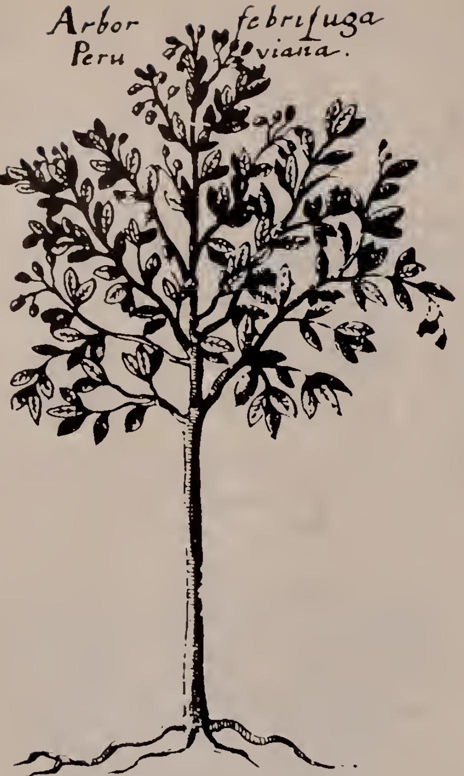


FIGURE 4. Drawing of the Cinchona tree. One of the earliest prints of the fever bark tree, published in Jan Jonston's *Dendrographia sive Historia Naturalis de arboribus et fructibus*, 1662 (Wellcome Institute for the History of Medicine, London).

During the next 30 years, in this and other ventures related to economic exploration of tropical countries, the constant attention and guidance given by the Royal Botanic Gardens at Kew was of utmost importance. Unfortunately, hardly anything is known about it by the general public, and little is remembered by scientists.[22-25]

Thanks to the indomitable courage and extraordinary endurance of Richard Spruce, a botanist of Markham's expedition, the British group collected the seeds of *Cinchona succirubra*, a sturdy and promising species, on a distant Bolivian mountain. When planted in southern India and Ceylon, this species grew well. Unfortunately, within the next few years, as in Indonesia, the quinine content of the bark of these trees was shown to be low, and the project proved to be still another commercial failure.[7,8]

In the 1860s, the Dutch plantations had a remarkable change of fortune thanks to Charles Ledger, an English merchant living in Peru. At Ledger's request, his Amerindian servant, Manuel Icamanahi, collected with great difficulty the seeds of what was believed to be a variety of *Cinchona calisaya*, growing on a faraway mountain site in Bolivia.[26] The precious package was sent to London for sale, but the British India government was not interested. The Dutch bought it for a trial, and soon some 20,000 seedlings were growing on their Indonesian plantation. When the Dutch chemists analyzed the quinine content of the bark of young trees, they were amazed at the 8–10% yield of quinine alkaloid instead of the usual 3% or less found in other barks.[1-10,20,21]

The Dutch decided to destroy over one million low-yielding trees and replace them with seedlings of *Cinchona ledgeriana*, as the new species was named. In 1877, the new bark was selling at a high price. The great technical and commercial success of the Dutch was due not only to a lucky choice of Ledger's seeds, but also to the remarkable scientific effort and tenacity deployed in this venture.[21]

The accomplishment of the Dutch was a grievous blow to the cinchona plantations in India and Ceylon. The government of British India attempted to stimulate the production of a cheap and efficacious antimalaria febrifuge based on a mixture of four alkaloids of the India-grown bark (quinine, cinchonine, quinidine, and cinchonidine). This could be manufactured locally from the bark of *Cinchona succirubra*, of which the supply was plentiful. Such "poor man's quinine" was almost as good as the refined product and could be bought at a mere fraction of the price of quinine. A factory set up in Bengal was actually preparing the cinchona febrifuge priced at one rupee an ounce. Yet such altruistic strategy could not compete in commercial terms with the highly profitable Dutch enterprise, based on producing quinine, which fetched high prices in Europe.[7,8,21,27-29]

Although the mode of curative action of quinine remained unknown before the discovery of the malaria parasites by Laveran in 1880, one can only admire the perspicacity of Binz, a German pharmacologist, who in 1867

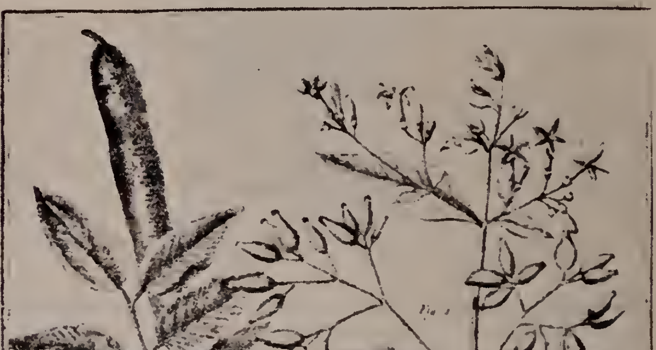


FIGURE 5. Cinchona *sp* (right), and Cassia *sp*. Engraving from Diderot's *Encyclopedie Raisonné des Sciences, des Arts et des Metiers*. Paris, 1751.

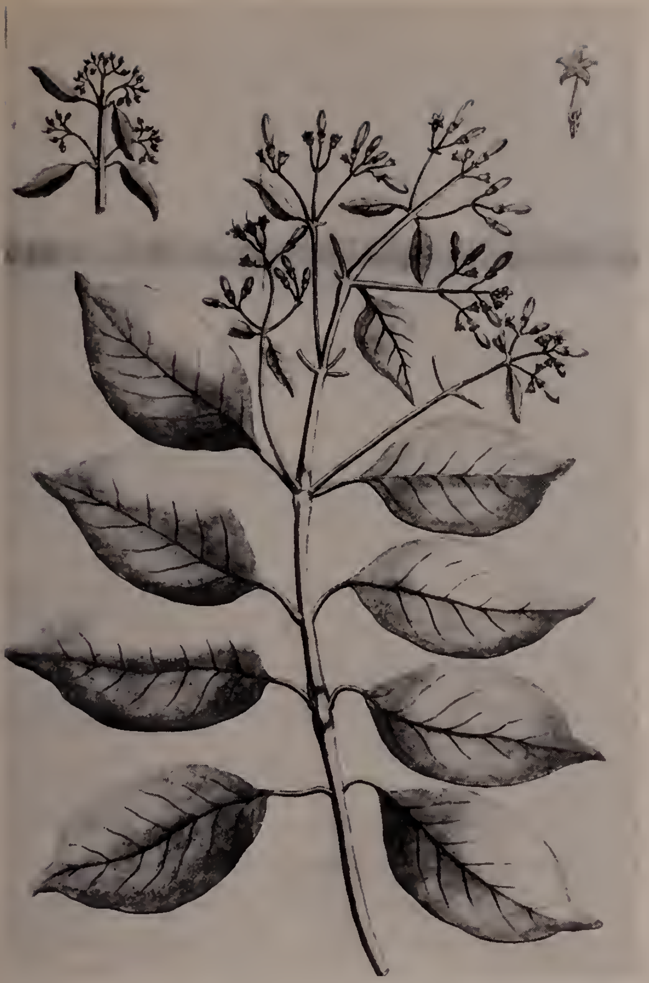


FIGURE 6. Leaves, flowers, and capsules of *Cinchona officinalis*.

reasoned, by analogy with water protozoa killed in solutions of quinine, that some blood parasites must be the cause of malaria.[30]

By 1887, most of the private plantations in the British Indian possessions came to an end; cinchona trees were destroyed and made way for tea. The same fate awaited other plantations in Asia, Africa, or the Caribbean. The story of plantations of high yielding species of cinchona trees in British India is a stirring and sad tale of individual foresight, initiative, courage, and determination marred by a series of unfortunate accidents, by lack of a definite health policy, and by inept social and economic decisions of the high administration.[24-26]

LATER DEVELOPMENTS

At the end of the 19th century, the saga of cinchona entered a new phase. The production of the best bark and the manufacture of quinine passed into the hands of large, profit-making enterprises. Greater supply of the bark and a better method of chemical extraction lowered the price of quinine sulphate from $100/kg in 1880 to $7 in 1893.[28,29] A wider use of quinine as a prophylactic drug now became possible.

An international agreement on the production and sale of quinine had to wait until 1913. The second Quinine Convention of 1918 gave the Dutch complete control of the quinine industry, under the organization known as the Kina Bureau. Some activities of the Kina Bureau were criticized, and not without reason; however, the supremacy of the Dutch in this field was a reward for a far-seeing and determined approach to large scale cultivation of the best cinchona plants.[21,24,25]

No tree, not even rubber, has ever had so much scientific attention, intelligent care, patience, and financial outlay as that of cinchona plantations in the former Dutch East Indies. By the late 1930s, the annual production of dried bark was close to 10,000 tons, most of which came from Indonesia.[29-31]

In 1934, the Health Organization of the League of Nations recommended that a standard mixture of cinchona alkaloids named *Totaquina* be widely used for chemoprophylaxis. With the outbreak of the Second World War, all these plans came to nothing, as the Japanese forces occupied Indonesia in 1942, cutting off the rest of the world from the main supplies of cinchona.[31,32]

Already in the 1930s, German scientists had developed plasmoquine and, soon after that, announced the discovery of Atebrin (quinacrine, or mepacrine) the first synthetic antimalarial drug, valuable for treatment and prophylaxis of malaria. Huge quantities of the new compound were produced in 1943–1944 in the United Kingdom and the United States. Introduced as a suppressive drug in all the malarious theaters of war, mepacrine enabled the Allied forces in southeast Asia and the Pacific to maintain their fighting conditions. There is no exaggeration in saying that this changed the recent course of world history. Other compounds (chloroquine, proguanil, pyrimethamine, mefloquine, etc) were soon developed by American, British, French, and Russian scientists, and this stimulated the postwar programs of malaria control and eradication.[31]

The past 50 years in the history of cinchona were marked by Indonesia's losing its dominant position as the major source of the bark, because of the large scale felling of the trees during the Japanese occupation, and because of the extensive use of synthetic antimalarials to meet the growing threat of drug resistance (especially in *P falciparum*, the main tropical parasite). The discovery in the 1980s of the antiplasmodial action of the Chinese Qinghaosu (*Artemisia annua*) drew attention to other botanical sources of new compounds.[29]

Of about 40 species of the genus *Cinchona*, only four—and certain hybrids—are of commercial value. The species *C calisaya*, *C ledgeriana*, *C succirubra*, and *C officinalis* have provided the rootstocks for the successful cultivation of these trees in India, Indonesia (Java), the Philippines, and more recently in Africa (Burundi, Cameroon, Kenya, Tanzania, Zaire). It is estimated that the present annual production of dried bark varies between 5,000 and 10,000 tons, and the current demand for all cinchona alkaloids is between 30,000 and 50,000 kg/year.[29] The World Health Organization estimated recently (1984) that the annual world demand for quinine for treatment of malaria was about 35,000 to 40,000 kg during the years 1980–1985.[31,32]

Some 30–50% of quinine produced is converted into quinidine for antiarrhythmic treatment. Altogether the medical applications account for 60% of the production, while 40% is used by the food and drug industry as a bittering agent.[23] The danger of overexploitation of commercially valuable species, and the possibility of genetic loss due to a limited range of genotypes of cinchona, is now causing concern. The vagaries of cultivation of cinchona trees have stimulated interest in plant cell culture; the yield of this method is very low, but some interesting scientific results offer a distant promise.[32]

In the long and remarkable saga of cinchona and its alkaloids, many dates could be chosen as historical milestones. The year 1988, as mentioned, marks the 350th anniversary of the first published memoir of the therapeutic virtue of the Jesuit's bark. The previous historical landmark was celebrated in 1920, a century after the isolation of quinine by Pelletier and Caventou. Although the chemical structure of quinine was known since 1908, the total synthesis of it was achieved by Woodward and Doering only in 1945, but industrial production of the drug proved to be too difficult and costly.[31]

During the last four decades of this century, the role of quinine as an antimalarial agent was underrated in competition with synthetic drugs. One might remember that during the past 20 years some 300,000 various chemical compounds were screened in primary animal tests, but only a dozen underwent more stringent studies for possible uses in humans.[31,32]

The revival of our interest in quinine and quinidine for treatment of falciparum malaria, increasingly resistant to many synthetic compounds (but much less to cinchona alkaloids), shows how far we are still from discovering an ideal antimalarial drug. Nevertheless, the present intense scientific activity, ranging from combinations of existing cinchona alkaloids and other compounds to the discovery of new plant species with significant antiplasmodial effect or to field studies in pharmacology and ethnobotany, has opened new perspectives.[33] Many of these were evoked at the recent meeting in Amsterdam, held under the ambitious motto: *Quinine Now!*[33] The future may show whether these "tender leaves of hope" will grow and branch out, but a wave of optimism is now evident.

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SPECIAL ARTICLE

My day on court: Thoughts from a summer vacation

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This story is an object lesson for busy doctors who have forgotten how to have fun because they've never been able to make the time. The old cliche, "If you don't use it, you lose it," is especially true for would-be tennis players, who've given up trying to find the time or a place to play. I'm going to tell you how I built a tennis court in my backyard 12 years ago for $5,400, and the fun it has given many people since. Go and do likewise!

My adventure started shortly after I bought the old 1825 farmhouse in Center Barnstead, New Hampshire, back in 1972. Sandy Tothill, a lively young woman in her mid-20s who lived up the road with her folks, came to visit. Staring out my kitchen window at a forlorn plateau consisting primarily of rocks, hills, half-starved weeds, and mosquitoes, she jumped up suddenly.

"Needs a tennis court," she said.

"Over my dead body," I replied.

I could think of a hundred good reasons not to build a court. First of all, it would probably cost an arm and a leg. Second, it was 300 miles from home and I would only be there on occasional weekends. I loved the country and was unwilling to swap even my mosquitoes for another parking lot with a fence around it. There were concerns about privacy, worries that hordes of neighbors would descend on me en masse. What would happen to my property taxes? And if I sold the house, would it improve the value, or drive away potential buyers who didn't like chasing little orange balls? Besides, what did either of us know about building asphalt tennis courts? A salaried physician and an unemployed guitar player seemed a poor risk for such a venture.

But Sandy's prodding was a force to be reckoned with. I had developed a healthy respect for her formidable talents ever since her parents had bought the old Brewster place up the dirt road. She was a near-pro tennis player, a swim instructor and lifeguard, a song writer, part of a two-sister singing act, a house restorer, a furniture refinisher, a macrame artisan, an auto mechanic, and the best gardener I'd ever hired. And now she was hell-bent on putting a tennis court in the middle of nowhere.

As we paced off potential sites, being bitten alive in the process, she decided that the cost could be halved by putting our hands and neighbors to work and using only inexpensive, "do-it-yourself" materials. She was sure that Barnsteaders would respect my privacy, and as for the house value . . . why, anyone who didn't want a tennis court shouldn't be allowed to buy such a beautiful old house anyway! The clincher came when she volunteered to get estimates, supervise construction, act as consultant and handywoman, and be my resident manager during the long weeks when I would be stuck in the city. I had to forcibly restrain her from beginning to macrame the net, while I went off to clean up the mess at our hospital's Employee Health Service.

A week later, real progress had been made. Turned off by the slickness of the professional court builders she'd interviewed, she had hired a local paving contractor, who to date had done nothing but driveways and parking lots. Smitten with Sandy's charm, he promised us an asphalt court for five thousand dollars instead of the customary ten-plus. It seemed to me like using boric acid instead of Bicillin,™ but there were pluses. He agreed to go along with our wish to do much of the work ourselves, gave realistic time estimates, and dug some test holes which proved promising because of the sand and gravel soil of the Granite State. We came up with a budget any office manager would have approved. The figures looked like this:

Bank the surface	$300
Save topsoil for later use	120
Layer with crushed gravel	450
Hand labor	100
Asphalt surfacing	3,600
Hire local farmer to dig post holes	55
Posts and fencing (4×4s and chicken wire)	225
Creosote for base of posts	9
Nets and poles	155
Practice board (four 4×4s and paint)	58
Two layers acrylic paint, no sealer	190
Measuring and setting lines	?
Total	$5,262

(Our total did not include the cost of landscaping around the 120′ by 60′ court, nor did it include estimates for upkeep.)

The bulldozer arrived in mid-June, and with it came the usual construction headaches; one complication after another subjected our huge, flat patient to more surgery. Despite the thrilling experience of watching a structure being created from raw land, we were disappointed many a time

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by the tardiness of our contractor and by inopportune rains which sprinkle New Hampshire when least expected. We could not pour our paving onto the crushed gravel because the rain, welcome at first, did not let up for weeks at a time. By mid-July, our man was off onto other jobs while we were left fuming, and without new photographs to take. When finally the blacktop went down, we were dismayed to find "puddling" in three areas. Entreaties to patch the area before the second layer went down were ignored because "the second layer will take care of it." It didn't, of course, and to this day, one corner of the court cries for its "squeegee" after each rain.

Meanwhile Sandy did what she could. She coaxed Caspar Hillsgrove, a retired farmer, to come over and dig the post holes, and creosoted the bases of the 38 4×4s which served as our fencing struts. These 20-foot monsters were spaced 14 feet apart, and surrounded with chicken wire. (Sandy climbed on the family car to staple the top layer, then bound the strips together with pieces of fine gauge wire.) When everything was in place, she made a practice backboard, painted it, and fixed it to the fence. She picked up extra rackets and balls at bargain prices. Neighbors and tennis buffs came by to help, one woman actually hacking down tenacious weeds encircling the new fence with a primitive scythe. Even the sidewalk superintendents formed a morale-boosting auxiliary.

It was October before the contractor honored his commitment to give us a surface smooth enough to take the planned two coats of paint. Only then did we pay the $1,200 which we had wisely withheld from him. Nothing more remained but to get the paint and lines down, and hope the neighbor's meadow would grow back before he got the law on us. We decided to have the lines drawn by a professional, the only non-amateur service of the project. His bill came to $70, raising our total to $5,332. We felt great! Our estimates had held very well.

None of my initial fears concerning this project turned out to be realized and I heartily recommend it to any physician ambitious enough to try it. We have had a dozen years of wonderful play. We enjoy rolling out of bed early on a summer morning, to play in sneaks and pajamas, or bundled up before the first snow sends us scurrying to bring in the net. Heaven knows how many balls have gone to meet their maker, sailing over the fence and into the mini-forest alongside the court. We joked about growing "tennis ball trees" until one day we did indeed find a moldy sphere with a whole root system wrapped around it, and we proudly paraded it around as an example of some exotic rotund arboreal *species tennisiana*. We have also enjoyed the berries that grew around the periphery of the court, bending over to snatch a few between serves. One of my favorite photographs of my father, taken during his final illness, shows him sunning himself by the net, while David Tothill, Sandy's brother, painted the raw surface on his hands and knees in the background!

Then, last year, an interesting sequel to the above story developed. My husband and I purchased a rental property, a charming old house two miles up the dirt road, with 52 acres of gorgeous land, and a nine-acre pond so loaded with bass it is virtually the only private pond in the state licensed to breed fish for sale to other ponds. The dam was in need of repair and we called in cement contractors to have a look. Up came guess who! Naturally he asked to see the court he had built for me over a decade before, saying that he now did the professional courts up in North Conway where the Volvo tournament was played annually! He couldn't believe his eyes.

"You mean *I* did this?!" he asked in wonderment. "This was my first court!" (As if we didn't know!) "It's spectacular! Look at how well it lasted! Only one small crack! Great condition! I want to fly up and take an aerial photo. And weren't you smart to insist on all that extra room at the baseline!"

Our contractor nearly went up without a plane when he asked us how many coats of paint we had put on through the years, and was informed that the paint was original. We were happy to produce the remaining cans of Allied Chemical Tech-Tone Top-Kote for tennis courts, which had been sitting in the basement of David's 200-year-old house since the day I took the picture of my dad. Even the net was original, although it had been left frozen to the ground one winter by its neglectful owner. Sandy and I couldn't have been prouder had we been medical students safely delivering triplets in a country kitchen.

We decided to refurbish the court this year. The chicken wire fencing, valued because it did not block our view of the lake, had rusted out so badly that balls were bouncing through the fence and vanishing into the raspberries, the barn, and the road to the beach. We had to do something. Sandy had just finished law school and was barely recovered from the bar exam, but who else would manage all?

"Let's have a tennis court repair party," I said. "Bob and I will supply dinner, you round up the usual suspects."

The idea was less than a week old when the court got its new fencing. We readied ourselves and synchronized efforts as if for a heart transplant. Here's how we did it.

Clark's Grain Store over on Route 28 in Pittsfield gave us a 10% discount on 750 linear feet of green vinyl-coated wire fencing, and deposited it right on the court for us. Bob, Sandy, and I stripped off the old chicken wire, a nasty job, in preparation for the big operation.

We laid in supplies. Bob, on a run to the city to check on an architectural job which could not tolerate his vacation, came back with enough Hebrew National hot dogs to fuel an army. Sandy brought over her new grill and a huge tossed salad. Her folks showed up with coffee in a big thermos, forgoing the evening's fishing. Sandy's hair dresser (her latest tennis student) and her boyfriend appeared with a pasta salad. Sandy's sister-in-law came with Katie, the baby, and husband David, toting a huge pot to cook the corn. This was supplied by Sandy's tennis partner, Luke, and his wife, Barb, who sell lobsters and roses and corn down by the Epsom traffic circle. Diane Crary came with two kids in tow and a carrot cake from the local bakery; her husband Jeff is the caretaker of Bass Pond Cottage, and a local landscape gardener.

We thought our working crew was fully assembled when along ambled the first tenants of Bass Pond Cottage, three darling yuppies who volunteered their services. Among the 15 of us, we had three stepladders, two wire cutters, seven hammers, a thousand hog ties, four kids under the age of four, and one Lhasa apso, who sent our semi-Siamese to the attic for the evening. This impromptu MASH unit of the tennis world descended on the periphery of our court with all the intense dedication of those who have taken vows. Banging, twisting, and pulling with fearless disregard for the laws of gravity, they worked from 4:30 PM to sundown.

By 8:15 we were all congregated on the candlelit screened porch, tired but satisfied, part of a happy country tradition that goes back to the first New England barn raising. Only our tenants had to rush back to the pond to take their little nephew fishing as promised; the next morning we brought them over a "CARE" package of leftovers. We also invited them to come and throw out the first ball, which they did, for two hours. They noted that our eight-foot fencing, as suspected, didn't quite make it. They volunteered to help us put up the last two feet but the Granite State rains got in the way again. We're leaving that for next year.

I urge everyone so mired in work that life can't be enjoyed to get out and build a tennis court. If you pay someone to do it today, it could cost you a good $25,000. If you do it yourself with your friends and neighbors, you'll save money and discover something new about friendship, teamwork, and the fun of being a kid again. We will not soon forget our adventure. We even got an unexpected bonus. Sandy's dad left us his copy of the novel *Malpractice* by Eleazar Lipsky. We are feeling so mellow by now, we may even read it!

FROM THE LIBRARY

THE SUMMER VACATION

It is hot; the dog days are upon us; it is time for rest and recreation. To the complicated, exacting and trying duties which daily befall the professional man, especially the physician, it is essential that he should bring to bear a clear mind, a fresh point of view and a steady hand.

As the writer more and more comprehends and increasingly appreciates the grave responsibilities, the nervous tasks, which are the lot of every busy doctor, the wonder to him is, not that so many of this great profession die in the very fullness of their powers and strength, but that any of them attain serene old age. Quietly making his rounds in the hospitals or his calls upon the sick at their homes, listening to the constant histories of pain and suffering, the worries and the fears of friends, supported only by his own fortitude and strong will, the doctor goes about his work painstakingly applying all the results of his study and his professional knowledge for the alleviation of pain and suffering for the benefit of mankind. Unheralded and unsung, ofttimes bitterly assailed and criticized, frequently misunderstood, the doctor plies his troubled way through life wherein mayhap the only award awaiting him is the possession of a quiet conscience and the consciousness of work well done.

Much has been said in criticism of the medical profession; its virtues go unproclaimed.

It is, therefore, not only a privilege, but it is in a very real sense the duty of every physician to dedicate some part of his year to recreation, rest and peace. Just now the woods, mountains and the pleasant beaches all are calling. We trust that by no member of this great profession will the call go unheard. In the hurried, hectic, troubled life of modern civilization there is all too small a place for contemplation and quiet; and yet it is in those rare moments that man finds himself and attains to those readjustments which the shocks of life so pressingly demand.

To every one of his medical friends, therefore (and the writer now numbers these by the hundreds), a pleasant, recuperative, beneficial summer most heartily is wished. Out in the trout streams, in the forests, in and on the refreshing waters of the Atlantic, along the smooth white ribbons of road that now make the remotest corners of our land accessible, it is hoped that every doctor will seek and find the rest and recreation that he needs and to which he is so abundantly entitled.

Having carried as best he could, in court and in conference, the burden of his responsibilities, the writer himself has decided for a few weeks to get far away from the turmoils of the city, and has planned to spend the month of August upon a ranch in Wyoming. Here it is hoped that he will gain added strength for the duties of the coming year, for the championship and espousal of the rights and the just causes of the medical profession, collectively and individually.

The year has been an exacting and a difficult one, and yet the countless acts of cooperation, sympathy and friendliness have lightened his task and filled him with a new appreciation of those whom he seeks to serve.

LLOYD PAUL STRYKER, ESQ
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Ritodrine-associated pulmonary edema

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Beta-adrenergic agents such as ritodrine are useful in the treatment of premature labor. Pulmonary edema is a rare, potentially life threatening complication associated with the use of these agents.[1-5] This unusual entity may require ventilatory support and hemodynamic monitoring in an intensive care unit setting. Thus, physicians should be aware of this infrequent cause of pulmonary edema.

CASE REPORT

A 29-year-old woman presented in preterm labor at 30 weeks' gestation. Physical examination revealed a blood pressure of 110/60 mm Hg; pulse, 86/min; and normal cardiorespiratory findings. Abdominal and ultrasound evaluations were compatible with the stated gestational age. The cervix was completely effaced and 3 cm dilated.

Intravenous ritodrine therapy was initiated at 6 mg/hr in conjunction with two doses of 12.5 mg of betamethasone (administered 12 hours apart to enhance pulmonary maturity in the fetus). During the next 36 hours the patient required progressively increasing doses of ritodrine up to 24 mg/hr (48 μg/kg/hr) to achieve control of uterine contractions. The intravenous fluid intake was 4,500 cc, and the urine output was 2,330 cc.

As the ritodrine therapy was being reduced, the patient suffered agitation and dyspnea associated with expiratory wheezing. Her blood pressure was 120/70 mm Hg, and her heart rate increased to 120/min. She received treatment with isoetharine by nebulizer and 20 mg of intramuscular furosemide. Ritodrine therapy was withheld. Despite this, the patient rapidly progressed to acute respiratory insufficiency, becoming diaphoretic, markedly dyspneic, and tachypneic, and she started to produce blood-tinged, frothy pulmonary secretions.

Physical examination revealed bilateral rales and wheezing, and a tachycardia of 140/min. Arterial blood gas studies demonstrated hypoxemia with a Po_2 of 56 mm Hg, Pco_2 of 46 mm Hg, and a pH of 7.26 (forced inspiratory oxygen [FIo_2] unknown). An endotracheal tube was inserted, and the patient received 1 mg of morphine sulfate and 20 mg of intravenous furosemide. She was transferred to the intensive care unit. There ventilation and oxygenation proved difficult, requiring 1.0 FIo_2 and 20 cm of positive end-expiratory pressure (PEEP) to obtain a Po_2 of 62 mm Hg, a Pco_2 of 46 mm Hg, and a pH of 7.30. Peak inspiratory pressures were in the 70 cm H_2O range, and her ventilation was greater than 14 L/min. She was paralyzed using 4 mg of pancuronium, and this condition was maintained with 1 mg/hr pancuronium by continuous infusion. A chest film showed normal heart size with bilateral alveolar infiltrates consistent with pulmonary edema (Fig 1).

A pulmonary artery catheter was inserted, and the initial pressures were pulmonary artery pressure (PAP), 38/22 mm Hg; pulmonary capillary wedge pressure (PCWP), 19 mm Hg, CO, 8.9 L/min, and a stroke volume of 64 cc. Mixed venous oxygenation was 32 mm Hg. A nitroglycerin drip was started, and the labor was allowed to continue. The patient delivered a 3-pound baby girl. Hemodynamics at this time showed a PCWP of 14 mm Hg with a CO of 12 L/min. Stroke volume increased to 87 cc. Fentanyl was added immediately after the delivery to sedate the patient while paralyzed. Oxygenation rapidly improved during the next few hours, and the endotracheal tube was removed 36 hours after admission. The remainder of the patient's course was uneventful.

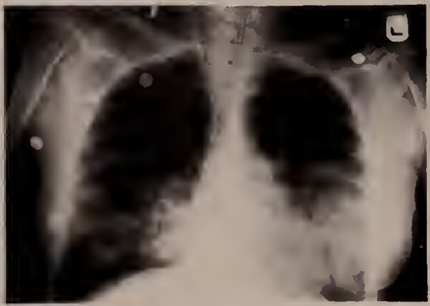

FIGURE 1. Chest film demonstrating bilateral alveolar and interstitial infiltrates in the presence of a normal cardiac silhouette.

DISCUSSION

Ritodrine, a beta-2 agonist, is frequently used for the treatment of premature labor, due to the drug's uterine muscle relaxation properties. Other effects include an increase in cardiac output, tachycardia, and a decrease in systemic vascular resistance due to its smooth muscle vasodilatory action. Common side effects of this drug are similar to those of other beta agonists and include tachycardia, nausea, vomiting, anxiety, tremulousness, and headache.[6] Maternal pulmonary edema has been a rare, but serious, complication of ritodrine therapy.

As demonstrated in the case presented here and in a review of the available literature, the pathophysiology of ritodrine-related pulmonary edema is uncertain and may involve several mechanisms. In the cases reported, patients received large amounts of fluid volume (up to 12 liters).[1,7] It has been shown that ritodrine has antinatriuretic and antidiuretic properties mediated through release of endogenous antidiuretic hormone. Animal studies using ritodrine have demonstrated fluid retention.[8,9] In addition, most of these patients were receiving steroids, and this may add to the sodium retentive state. Whether this increased volume is sufficient to cause hydrostatic pulmonary edema is unclear. Several case reports have demonstrated low pulmonary artery wedge pressures and high cardiac outputs (although catheterization data from these reports were obtained after the patients had been treated).[2,3] Other patients have shown elevated left-sided

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pressures. Studies in pregnant baboons and sheep receiving ritodrine have failed to demonstrate significant changes in extravascular lung water, central venous pressure, or pulmonary artery pressure, despite significant elevation in wedge pressures.[8,10] Experimental attempts to produce left ventricular dysfunction using ritodrine have been unsuccessful. Echocardiographic studies obtained in patients receiving ritodrine also failed to demonstrate significant global or segmental wall motion abnormalities.[7]

As in the case presented, the hypoxemia associated with ritodrine pulmonary edema may appear greater than expected based upon the radiographic findings. To investigate this clinical observation, Conover et al[11] studied the pulmonary blood flow changes induced by ritodrine in the canine model. They demonstrated that ritodrine inhibits normal hypoxic pulmonary vasoconstriction and hypothesize that this may be the mechanism that produces the more severe ventilation-perfusion mismatching. Based on the absence of clear hemodynamic evidence of hydrostatic pulmonary edema in both the experimental and clinical studies of ritodrine pulmonary edema, some investigators have suggested that this may represent a form of noncardiogenic pulmonary edema.[2,3] Despite this, data clearly demonstrating increased pulmonary capillary permeability are lacking.

The patient presented here showed some of the frequently reported features found with this entity: She had been treated for more than 24 hours with high doses of ritodrine and had received 4.5 L of fluid. She experienced nausea, vomiting, tremulousness, and anxiety prior to the sudden onset of respiratory distress. There was no evidence of other causes for her pulmonary edema, including aspiration, pre-eclampsia, or amniotic emboli. Her severe hypoxemia appeared to be out of proportion to the radiographic findings, and she improved rapidly after the ritodrine was discontinued and therapy for volume overload was instituted. The hemodynamic data obtained early in this case revealed an elevated wedge pressure, but a high cardiac output and low systemic vascular resistance state.

In summary, we can only postulate as to the possible cause of beta-2 agonist-produced pulmonary edema. The patients at risk have increased total body volume secondary to pregnancy and then are submitted to a treatment with ritodrine that produces vasodilation and requires more volume replacement. The antidiuretic, antinaturetic effects of ritodrine further increase total body volume. The "third spacing" of fluid could involve the interstitial spaces in the lung. It is unclear whether at this point the patient at risk develops either cardiac or direct pulmonary dysfunction, in that available data do not clearly support either etiology. Further, the possible effect of prostaglandins associated with labor and their interaction with ritodrine has not been evaluated, and conceivably this factor could be involved in the pathophysiology of this entity. It is clear, however, that these patients respond to discontinuation of the ritodrine, institution of ventilatory support, and diuretic therapy. Further studies elucidating the exact mechanism of ritodrine-induced pulmonary edema are warranted.

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Agenesis of the left hepatic lobe with gastric volvulus

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Agenesis of the left hepatic lobe is detectable in adults by radiography,[1] ultrasound, and computed tomographic (CT) scanning.[2] We report a case of agenesis of the left hepatic lobe which was in itself asymptomatic but had important consequences for an adjacent viscus.

CASE REPORT

A 57-year-old man suffering from chronic alcoholism was admitted with a two-month history of intermittent postprandial abdominal pain and vomiting. Physical examination disclosed congenital nystagmus and a periumbilical hernia. An abdominal series revealed a large soft tissue density in the left upper quadrant with displacement of small bowel gas to the right. An unsuccessful attempt was made to visualize the duodenum with endoscopy. No ulcerative lesions were identified in the stomach. An upper gastrointestinal radiographic series revealed an inverted U-shaped stomach and a high position of the duodenal bulb, with slow passage of contrast into the duodenum (Fig 1). A diagnosis of chronic, recurrent organo-axial volvulus of the stomach was made. On surgical exploration, the stomach was found to be folded, with the greater curvature tilted upwards and accommodated in the large area created by the absence of the left hepatic lobe. The volvulus was surgically corrected.

To document the agenesis of the left hepatic lobe, a postoperative Tc-99 radionuclide scan was done (Fig 2).

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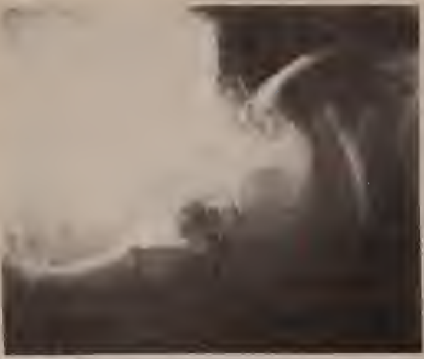

FIGURE 1. Upper gastrointestinal series showing gastric volvulus with high position of duodenal bulb, and passage of contrast into the duodenum.

DISCUSSION

Liver malformations are classified by Battle et al[3] as anomalous lobulation, accessory lobe attached to the liver with a pedicle, ectopic liver tissue without a pedicle, and hypoplasia or absence of a lobe.

Aplasia of the left lobe is rare. Merrill[1] reported only one case in 19,000 necropsies. Emery[4] noted some shrinkage and color change in the left lobe in 22 instances out of 110 consecutive necropsies in children under two months of age. McCurdy[5] reported an aplastic left lobe associated with peptic ulcer, which he attributed to the adhesions of the pylorus to the under surface of the diaphragm. Atrophy of the left lobe has also been described in association with chronic liver disease. It has been hypothesized that malnutrition causes a preferential flow to the right lobe. Benz et al[6] found an aplastic lobe with no evidence of cirrhosis in 32 patients.

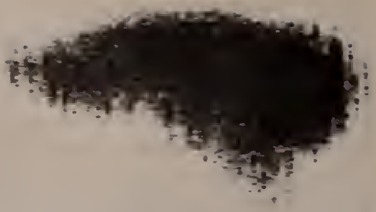

FIGURE 2. Postoperative Tc99 radionuclide scan showing absence of the left hepatic lobe 20 minutes after injection dose (3 mCi).

The most common entity associated with congenital absence of the left lobe of the liver is volvulus of the stomach.[7,8] Other associated anomalies include hiatal hernia and chronic cholelithiasis.

A number of review articles on the subject stress findings on upper gastrointestinal series that include a U-shaped folding of the stomach with high positioning of the duodenal bulb. Marked mobility of the stomach on barium meal examination can be seen with an aplastic hepatic lobe.[9]

Nonvisualization of the left hepatic lobe on scintiscan of the liver can also be secondary to metastatic gastric cancer and replacement by malignancy.[10] As demonstrated by the case reported here and a review of the literature, the association between the anomaly and gastric volvulus is not coincidental. Radiographic findings are specific and call for prompt recognition of the disorder and surgical correction.

Acknowledgment. The authors thank Anil Gulati, MD, for typing the script and Paul Archibald for his assistance in preparing the photographs.

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Single coronary artery originating from the right sinus of Valsalva

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The incidence of single coronary artery is rare, occurring in less than 0.1% of patients undergoing coronary angiography.[1] We present the first angiographic report, to our knowledge, of a patient with a single coronary artery originating from the right sinus of Valsalva. The left circumflex artery shares a cloacal ostium with the right coronary artery. The left anterior descending coronary artery originates from the proximal right coronary artery and courses between the aorta and the pulmonary artery before pursuing a normal distribution. The circumflex course is posterior to the aorta, eventually assuming an obtuse marginal distribution.

CASE REPORT

A 71-year-old woman with hypertension, chronic atrial fibrillation, and chest pain was admitted with an inferior wall myocardial infarction. The hospital course was complicated by recurrent pulmonary edema and postinfarctional angina. She also suffered an embolus to the left femoral artery,

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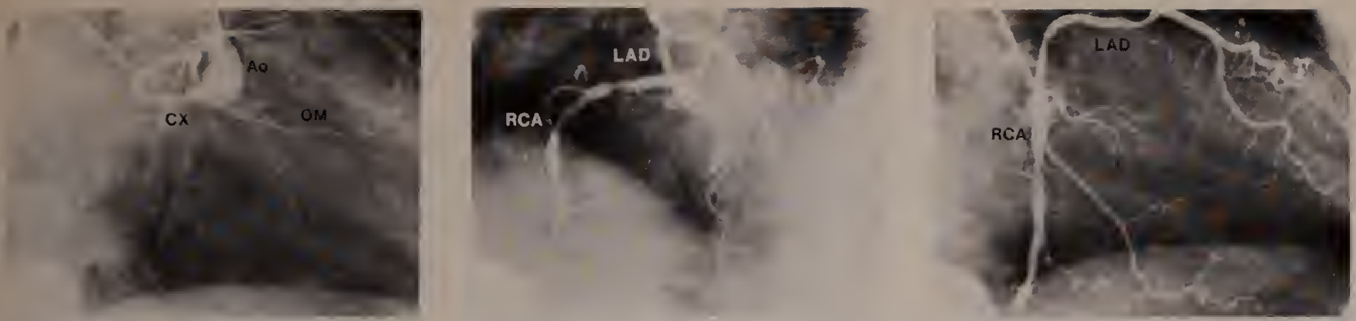

FIGURE 1. **Left,** right anterior oblique angiogram of anomalous circumflex (CX), which courses posterior to the aorta (Ao) and assumes an obtuse marginal (OM) distribution. **Center,** left anterior oblique angiogram of right coronary artery (RCA) and left anterior descending artery (LAD). Left anterior descending artery courses anteriorly, between the pulmonary artery and the aorta. **Right,** right anterior oblique angiogram of right coronary artery and left anterior descending artery. Left anterior descending artery courses anteriorly, between the pulmonary artery and the aorta.

underwent successful embolectomy, and received systemic anticoagulation therapy. Because of recurrent angina and pulmonary edema, she underwent cardiac catheterization and coronary angiography.

Angiography revealed a single cloacal ostium in the right sinus of Valsalva, giving rise to the right coronary artery and the circumflex artery. The distribution of the right coronary artery was normal. The circumflex artery coursed posterior to the aorta and assumed an obtuse marginal distribution (Fig 1). The left anterior descending coronary artery originated from the proximal right coronary artery and coursed anteriorly between the aorta and pulmonary artery (Fig 1). Its final distribution was normal. There was evidence of significant atherosclerotic coronary artery disease involving all of the major coronary arteries. A left ventriculogram revealed moderately severe mitral regurgitation and a left ventricular ejection fraction of 42%. The patient underwent saphenous vein coronary artery bypass grafting to the high left anterior descending coronary artery, the right coronary artery, and the circumflex artery. She also had mitral valve replacement with a porcine heterograft valve. After a long recuperative period, she was discharged from the hospital in satisfactory condition.

Discussion

When all coronary arteries arise from the right sinus of Valsalva, the usual anatomic patterns are a separate right sinus of Valsalva for the left main segment, which then courses between the aorta and the pulmonary artery before bifurcating into the left anterior descending and circumflex branches; and the left main arising from the proximal right coronary artery, coursing posterior to the aorta, and then bifurcating into the left anterior descending and circumflex branches.[1] A rare variant consists of a separate right sinus of Valsalva ostium for the left anterior descending coronary artery, with the right coronary artery and circumflex arteries arising from their normal origin.[1] These variations are represented in a diagram in Figure 2.

There are two reported cases of aberrant origin of a single coronary artery from the right sinus of Valsalva, in which the left anterior descending coronary artery courses between the aorta and pulmonary artery, and the circumflex artery passes posterior to the aorta.[2,3] As diagrammed in Figure 3, the patient reported here differs from these cases in two aspects: the cloacal ostium gives rise to the right coronary artery and circumflex artery; and the left anterior descending artery arises from the proximal right coronary artery, with an anterior course.

A single coronary artery is a rare angiographic finding. It occurs slightly more often in males than in females (1.4:1) and arises with equal frequency from the right and left sinus of Valsalva. There is an increased association of single coronary arteries with other congenital cardiac anomalies.[2]

In most cases, anomalous coronary arteries are incidental findings with minor clinical significance. However, sudden death has been reported as a complication of aberrant origin of the left coronary artery from the right sinus of Valsalva, when the proximal course of this vessel is between the aorta and pulmonary artery.[4] The mechanism of sudden death is unknown. It has been suggested that expansion of the aorta and pulmonary arteries during exercise results in compromised coronary blood flow. The underlying mechanism may be mechanical stretching of the left coronary artery with compression of the orifice.[5] Other mechanisms, such as kinking of the artery, have also been postulated.[4] Indications for early surgical therapy are controversial in this subgroup. Some advocate prophylactic bypass grafting, while others would advocate surgery only in those patients who have suffered a cardiac event.[6] It is doubtful that there is an increased incidence of accelerated atherosclerotic coronary artery disease in patients with single coronary arteries.[3,7]

As demonstrated in this case, a single coronary artery can be compatible with long life, even if the left anterior descending coronary artery courses between the aorta and the pulmonary artery. Identifying anomalous coronary

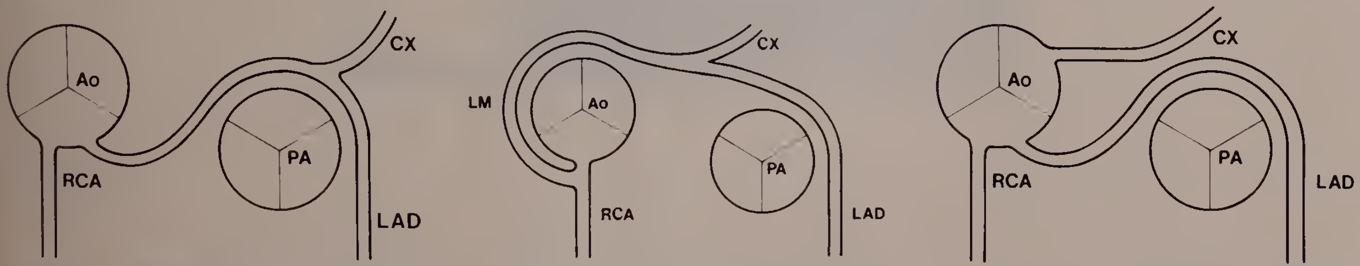

FIGURE 2. **Left,** diagrammatic representation of a separate right sinus of Valsalva ostium for the left main artery, which then courses between the pulmonary artery (PA) and the aorta (Ao) before bifurcating. **Center,** left main artery (LM) arising from the right coronary artery (RCA), coursing posterior to the aorta, and then bifurcating. **Right,** a separate right sinus of Valsalva ostium for the left anterior descending artery (LAD) (CX, circumflex).

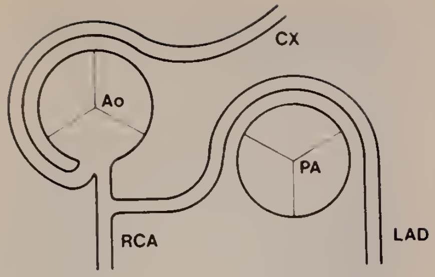

FIGURE 3. Diagrammatic representation of the cloacal ostium giving rise to the right coronary artery (RCA) and circumflex (CX). Left anterior descending artery (LAD) originates from the proximal right coronary artery and courses between the pulmonary artery (PA) and the aorta (Ao).

arteries before coronary bypass grafting is obviously important. If a coronary artery is not visualized, this may be interpreted as a completely occluded or congenitally absent vessel. In this patient, if the circumflex artery had not been identified, a stenotic vessel would have been excluded from bypass grafting.

Acknowledgment. The authors thank Dr Deborah J. Barbour, who independently reviewed the cineangiograms, and Lori Stephenson, for her technical assistance.

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Tufts of grass on wires, Jamaica (Spencer King, MD).

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Sleep deprivation in residents

TO THE EDITOR: I just read Dr Garcia's commentary in the December 1987 issue of the *Journal* regarding sleep deprivation in residents.[1] As a third-year medical student I am concerned about my ability to provide the best medical care possible to patients when I am in a residency program. His article was well written and expressed all of my thoughts and opinions on the subject.

I hope that by the time I apply to residency programs, more will have adopted the night float system. It seems humane and rational.

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[1] Garcia EE: Sleep deprivation in physician training. *NY State J Med* 1987; 87:637-638.

Chinese torture of Tibetans

TO THE EDITOR: From January 20 to February 1, 1988, I had the privilege of running a "tent clinic" for Tibetans at Sarnath, India, on the occasion of a series of public teachings given by His Holiness the Dalai Lama. I was in India working among Tibetan refugees on an elective for fourth-year medical students, "Health Care in Developing Countries," sponsored by the Department of Preventive Medicine and Community Health and the Alumni Fund of the State University of New York Health Science Center at Brooklyn. A large number of Tibetan refugees living in India were present in Sarnath, as well as an estimated 800-1,000 persons from Tibet itself. Many of these persons required medical attention during our 12 days of operation, and as one of two physicians I personally treated about 50 patients per day.

Aside from the usual complaints any newcomer to the Indian plains suffers, such as acute gastroenteritis, skin rashes, cough, colds, and fever, we treated three patients whose cases I feel compelled to report. All three were recent arrivals from Tibet (ie, the Tibetan People's Autonomous Region of China) and all three were Tibetan-speaking. They were two middle-aged women and an elderly Buddhist monk. All had been victims within the previous two months of systematic torture by the Chinese authorities in prisons in or around Lhasa, Tibet.

The first of these patients I encountered was a 50-year-old Tibetan woman who presented to our clinic with a complaint of swollen ankles. I examined both ankles and found them to be deeply bruised and swollen. The patient also had loss of sensation over both feet. There was no evidence of fracture. I inquired about possible injury and at that point in the examination the patient became tearful and explained that she had been manacled about the ankles for 18 days in an unheated cell and beaten about both legs, feet, back, shoulders, arms, and hands at intervals throughout her internment. As her feet began to swell the metal manacles became so tight she'd lost all feeling in both feet. This was slowly resolving since her release. The emotional and psychological effects of her torture were still very much with her.

The second patient was a 41-year-old mother who presented with weakness and abdominal pain. Upon examination I found her to have a cardiac arrhythmia and a heart murmur. After asking her about previous heart problems she proceeded to tell us about her recent arrest, torture, and escape from a Chinese prison. She had been interrogated for three consecutive days with an electric prod which she described as flinging her across her cell each time she was struck with it. This treatment ended when she eventually lost consciousness. She had been having cardiac symptoms ever since. This woman eventually escaped by jumping from a prison window and finding her way on foot to the Nepalese border. She was forced to leave her husband and several young children behind and to avoid any contact with them for fear of reprisals by the Chinese.

The third patient was a Buddhist monk in his 60s who complained of a painful gluteal abscess. As we prepared to drain the abscess he became anxious and tearful, saying he "could not stand any more pain." We stopped the preparations and asked the patient to explain. He then showed us healing rope burns on both forearms and explained that he'd been interrogated after the October 1987 protests in Lhasa. Ropes had been gradually tightened over both of his arms until the skin was torn. He apologized for his tears in the clinic but remained, as did the other victims, tearful and deeply distressed.

As a young physician these were some of the most disturbing patients I have ever encountered. I believe my clinical judgment to be sound when I assert that these people have been victims of systematic physical torture. Such torture while under interrogation is clearly a part of Chinese treatment of ethnic Tibetans.

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The Make-A-Wish Foundation of the Hudson Valley

TO THE EDITOR: We at the Make-A-Wish Foundation are dedicated to granting the wishes of children under age 18 who suffer from life-threatening illnesses. We do our best to bring some happiness to the lives of these stricken youngsters by fulfilling their dreams, whether it is taking a trip to Disney World, visiting a friend or relative, or merely seeing snow for the first time. Once we have the doctor's approval and parental consent, we begin to make dreams come true for children who may have few tomorrows.

We are all volunteers. We have no paid staff. Our reward comes from seeing the smiling faces of the children we serve. We have the resources and the volunteers on hand to help as many children as we can find, but we need your assistance. We are trying to form a network whereby physicians who are aware of these special youngsters will contact us. We do no research, and we seek no cures, but we hope, between the medicine and the Make-A-Wish, that sometimes we can make a miracle. All we need from you are referrals—we will do the rest. We can be contacted at (914) 666-WISH.

STEVE BLAKE
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Protected time

TO THE EDITOR: We continue to be concerned about the high price physicians and their families pay to meet the insatiable demands of our profession. To speak of the health of the physician is perhaps to be too narrowly focused. It neglects the impact of the discipline on our families. As medicine has become more complex and the demands of time and effort even greater, we observe physicians of all ages struggling with the dilemmas of "what to give to whom." How do they allocate their time?

Make no mistake, our families pay dearly for our pursuits. The irony is that often their cries are muted to whispers because we are serving a "good cause." We are the healers. As absentee parents we place a devastating burden on our other halves. They become both mothers and fathers as we occupy half-time roles. We assuage our guilt by showering them with gifts of everything but time, the most precious gift of all. Often while we proceed to grow and develop, our partners are left floundering in the backwash of our drives.

Our children are often channeled, consciously or unconsciously, into professions—especially ours. They learn all too early, "If you can't beat them, join them." Society often seems to expect them to be super achievers and leaves scant room for them to explore and find themselves.

One of the most heartening things is to observe physicians "making time" to be with their families. The wise ones realize that it is better to pay up front rather than pay later. The victims may not recover from our victories. Unless we make time, we become familiar strangers in our homes. Clinical researchers speak of "protected time," that period of quality time that is committed solely to scientific endeavors. Should our families receive less?

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Neurologic complications of metoclopramide therapy

TO THE EDITOR. Metoclopramide centrally antagonizes dopamine[1] and often causes extrapyramidal side effects.[2] A recent report documents multifocal myoclonus without seizures in a patient on dialysis.[3] The following report demonstrates the ability of metoclopramide to induce both in the same patient.

Case Report. A 75-year-old man on hemodialysis underwent successful hemicolectomy for gastrointestinal bleeding. The patient received 75 mg of metoclopramide in divided doses over 48 hours, which relieved postoperative ileus. Prior to the last dose the patient underwent dialysis. Conscious and without hypotension, he exhibited uncontrollable twitching and jerking of his arms and legs. One half hour after dialysis, a witnessed generalized convulsion occurred. Four hours later, a neurologist witnessed a second seizure. Oral phenytoin therapy was begun, and subsequent neurologic evaluation revealed asterixis and hyperreflexia without focal signs. Two hours later, the patient stated that his hiccups and jerking arms prevented him from feeding himself breakfast. His speech and language were normal; gross flinging motions of the arms and legs occurred frequently but irregularly. A total of three generalized convulsions and four myoclonic episodes were witnessed during a 36-hour period before clonazepam relieved symptoms. A computed tomographic (CT) scan of the head revealed diffuse atrophy. An electroencephalogram documented only mild, diffuse slowing. Electrolyte and urea levels were unchanged. Concomitant medications included digoxin, nitroglycerin, and cimetidine (one intravenous dose).

Discussion. Patients on dialysis may exhibit several neurologic syndromes including dialysis dementia, uremic encephalopathy, and dialysis disequilibrium. The absence of speech defects and progressive mental deterioration argues against dialysis dementia in this case. Five hours after dialysis and the first seizure, the blood urea nitrogen level was 64 mg/dL, a typical value for this patient. Such a low value and a clear sensorium do not suggest uremic encephalopathy. The onset of seizures shortly after dialysis implicates disequilibrium, yet years of dialysis prior to metoclopramide therapy had caused no difficulty. No episodes occurred after clonazepam was subsequently tapered off. Thus, metoclopramide appears to have lowered the threshold for both seizures and myoclonus. The central nervous system excitability noted here is not typical of extrapyramidal dystonia alone. Though little therapeutic information is available in the published literature, this patient's response to clonazepam suggests that this drug is preferable to phenytoin. Metoclopramide must be given judiciously in renal failure. Unfortunately, the development of myoclonic jerking appears too late to prevent convulsions.

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1. Ahtee L, Buncombe G: Metoclopramide induces catalepsy and increases striated homovanillic acid content in mice. *Acta Pharmacol Toxicol* (Kbh) 1974; 35:429–432.
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Extrapleural hematoma following infraclavicular subclavian vein catheterization

TO THE EDITOR: The routine use of venous cannulation in the management and monitoring of medical and surgical conditions is not without complications. These complications, which include pneumothorax, hydrothorax, hemothorax, hydromediastinum,[1] mediastinal hematoma,[2,3] and others appear to be more frequent with the subclavian vein than with the internal jugular vein. This report describes a large extrapleural hematoma resulting from infraclavicular left subclavian vein cannulation, an unusual complication which has been reported in the literature only following internal jugular vein catheterization.[4]

Case Report. A 48-year-old man presented to the emergency room of the Bronx-Lebanon Hospital Center with abdominal pain, vomiting, and rectal bleeding. The chest roentgenogram on admission did not reveal any obvious abnormality. An exploratory celiotomy revealed a left Bochdalek hernia with a strangulation obstruction of the transverse colon. Resection was done, and an end colostomy and mucous fistula were created. A chest tube was inserted into the left pleural cavity, and the diaphragmatic defect was repaired.

To provide postoperative fluid management, the left subclavian vein was catheterized by the infraclavicular approach. The catheter was successfully introduced in one attempt. "Intra cath," 8-inch, 16-gauge catheter was used through a 14-gauge needle. Roentgenograms taken immediately after the catheterization did not reveal any abnormality. During the next 72 hours, however, a large, well-circumscribed opacity with rounded lower borders developed in the patient's left upper chest and neck (Fig 1). Thoracentesis revealed sanguinous fluid. A computed tomographic (CT) scan of the chest was obtained for anatomic definition of the opacity. The opacity was found to be an extrapleural fluid collection extending from the mid left chest to the neck (Fig 2). An anterior chest tube was inserted into the patient's second left intercostal space, and serosanguinous fluid was drained. Repeat radiographs after 24 hours showed complete resolution of the mass. Three months later, repeat chest films were within normal limits.

Discussion. Extrapleural hematoma is an uncommon condition. It has been reported following lower or upper sympathectomy,[5,6] and in a case of ruptured intercostal artery.[7] Aguilar[8] reported a case in which a retropleural hematoma complicated spontaneous rupture of a traumatic pseudoaneurysm of the aorta, and Wisheart et al[9] reported a case of fatal retropleural hematoma following damage to the ascending pharyngeal artery.

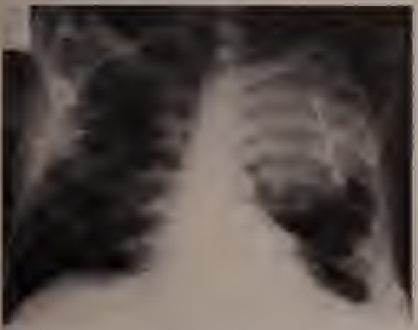

FIGURE 1. Chest film taken two days following insertion of left subclavian catheter shows a well-circumscribed left upper zone opacity. The pleural cavity is free of any fluid collection. Chest tube drainage was minimal at this time.

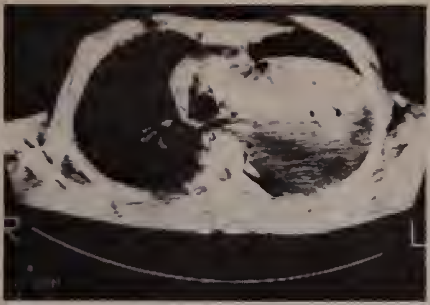

FIGURE 2. CT scan showing fluid collection in the upper left chest; compressed lung parenchyma is seen anteriorly.

In the patient described here, a large, well-circumscribed fluid collection developed in the left upper chest in spite of the presence of a chest tube in the left pleural cavity. A CT scan confirmed that this was an extrapleural collection extending into the neck.

The etiology of the complication is not clear. Although the hematoma could have been caused by inadvertent puncture of the subclavian artery, this does not seem likely in this case, since the puncture was easily done and was successful on the first attempt. The other possibility is extravasation from the subclavian vein at the puncture site.[10] When the needle-over-catheter technique is used, as in this case, the hole in the vein is larger than the diameter of the catheter. Thus, when proximal venous obstruction is present, fluid may extravasate. Although we did not do any further studies to determine whether or not this was the case, there was no clinical evidence of subclavian vein obstruction. Another possibility is that irritation of the endothelium by the infusate could have induced venoconstriction. The proximal pressure then could have enlarged the needle hole in the wall of the vein, leading to extravasation.

Although the case of extrapleural hematoma following catheterization of the internal jugular vein, as reported by Kontozoglou et al,[4] had a fatal outcome, the patient described here did well with simple drainage of the extrapleural collection. This may be because the soft tissue around the subclavian vein sealed the puncture more efficiently compared to the internal jugular vein. Also, no anticoagulation therapy was being used in this patient.

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Immunization of children infected with human immunodeficiency virus—supplementary ACIP statement[*]

The Immunization Practices Advisory Committee (ACIP) recently reviewed data both on the risks and benefits of immunizing children infected with human immunodeficiency virus (HIV)[1] and on severe and fatal measles in HIV-infected children in the United States.[2] Since this review, the committee has revised its previous recommendations for measles vaccination and for mumps and rubella vaccination.

Previously published ACIP statements on immunizing HIV-infected children have recommended vaccinating children with asymptomatic HIV infection, but not those with symptomatic HIV infection.[3] After considering reports of severe measles in symptomatic HIV-infected children, and in the absence of reports of serious or unusual adverse effects of measles, mumps, and rubella (MMR) vaccination in limited studies of symptomatic patients,[4,5] the committee feels that administration of MMR vaccine should be considered for all HIV-infected children, regardless of symptoms. This approach is consistent with the World Health Organization's recommendation for measles vaccination.[6]

If the decision to vaccinate is made, symptomatic HIV-infected children should receive MMR vaccine at 15 months, the age currently recommended for vaccination of children without HIV infection and for those with asymptomatic HIV infection. When there is an increased risk of exposure to measles, such as during an outbreak, these children should receive vaccine at younger ages. At such times, infants six to 11 months of age should receive monovalent measles vaccine and should be revaccinated with MMR at 12 months of age or older. Children 12–14 months of age should receive MMR and do not need revaccination.[7]

The use of high-dose intravenous immune globulin (IGIV) (approximately 5 gm% protein) administered at regular intervals is being studied to determine whether it will prevent a variety of infections in HIV-infected children. It should be recognized that MMR vaccine may be ineffective if administered to a child who has received IGIV during the preceding three months.

Immune globulin (IG) (16.5 gm% protein) can be used to prevent or modify measles infection in HIV-infected children if administered within six days of exposure. IG is indicated for measles-susceptible[†] household contacts of children with asymptomatic HIV infection, particularly for those under one year of age and for measles-susceptible pregnant women. The recommended dose is 0.25 mL/kg intramuscularly (maximum dose, 15 mL).[7]

In contrast, exposed symptomatic HIV-infected patients should receive IG prophylaxis regardless of vaccination status. The standard postexposure measles prophylaxis regimen for such patients is 0.5 mL/kg of IG intramuscularly (maximum dose, 15 mL).[7] This regimen corresponds to a dose of protein of approximately 82.5 mg/kg (maximum dose, 2,475 mg). Intramuscular IG may not be necessary if a patient with HIV infection is receiving 100–400 mg/kg IGIV at regular intervals and received the last dose within three weeks of exposure to measles. Based on the amount of protein that can be administered, high-dose IGIV may be as effective as IG given intramuscularly. However, no data exist on the efficacy of IGIV administered postexposure in preventing measles.

Although postexposure administration of globulins to symptomatic HIV-infected patients is recommended regardless of measles vaccine status, vaccination prior to exposure is desirable. Measles exposures are often unrecognized, and postexposure prophylaxis is not always possible.

While recommendations for MMR vaccine have changed, those for other vaccines have not.[3] A summary of the current ACIP recommendations for HIV-infected persons follows (Table I). These recommendations apply to adolescents and adults with HIV infection as well as to HIV-infected children.

TABLE I. Recommendations for Routine Immunization of HIV-Infected Children—United States, 1988[*]

	HIV Infection	
Vaccine	Known Asymptomatic	Symptomatic
DTP[†]	yes	yes
OPV[§]	no	no
IPV[¶]	yes	yes
MMR[**]	yes	yes[††]
HbCV[§§]	yes	yes
Pneumococcal	no	yes
Influenza	no	yes

[*] See accompanying text and previous ACIP statement[3] for details.
[†] DTP = Diphtheria and tetanus toxoids and pertussis vaccine.
[§] OPV = Oral, attenuated poliovirus vaccine; contains poliovirus types 1, 2, and 3.
[¶] IPV = Inactivated poliovirus vaccine; contains poliovirus types 1, 2, and 3.
[**] MMR = Live measles, mumps, and rubella viruses in a combined vaccine.
[††] Should be considered.
[§§] HbCV = *Haemophilus influenzae* type b conjugate vaccine.

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[*] These guidelines are reprinted from *Morbidity and Mortality Weekly Reports* 1988; 37:181–183. The prior Immunization Practices Advisory Committee recommendations for immunization of HIV-infected children were published in the May 1988 issue of the *Journal*.

[†] Persons who are unvaccinated or do not have laboratory evidence or physician documentation of previous measles disease.[7]

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Campylobacter enteritis

Introduction

Campylobacter enteritis is an acute disease of variable severity characterized by diarrhea, abdominal pain, malaise, fever, nausea and vomiting. It is frequently self-limited within one to four days and usually lasts no more than ten days. A prolonged illness may occur in up to 20% of cases, especially in adults, and relapses can occur. The usual incubation period is three–five days with a range of one–ten days. Individuals not treated with antibiotics may excrete the organism for two–seven weeks. A chronic carrier state is unusual except among animals and poultry. Gross or occult blood in association with mucus and white blood cells is usually present in liquid, foul-smelling stools. A typhoidal-type syndrome, reactive arthritis and, rarely, febrile convulsions and meningitis have been described.

Campylobacter fetus has long been recognized as an occasional cause of systemic infection in debilitated hosts. However, *C jejuni* remained unrecognized as a human pathogen until sophisticated isolation techniques were developed in the mid 1970s. Since then, *C jejuni* has been recognized as possibly the most prevalent bacterial pathogen causing diarrheal disease worldwide, including temperate United States.

Epidemiology

Campylobacter causes approximately 5–14% of diarrhea worldwide and may be responsible for a greater proportion of enteritis than either Salmonellae or Shigellae. Cases have been linked to contact with animals including swine, cattle, sheep, cats, dogs and other domestic animals, rodents and birds including poultry. Common source outbreaks have been associated with food, unpasteurized milk and unchlorinated water.[1,2,3] In 1978, 2,000 of 10,000 people living in a Vermont town became ill after exposure to a contaminated water supply.[4]

Campylobacter was added to the list of reportable diseases in New York State on January 1, 1986. During 1986, 1,713 cases were reported among residents of upstate New York compared to 2,031 and 277 cases of salmonellosis and shigellosis respectively. Most cases were sporadic in nature and only one significant outbreak affecting 120 people at the Hudson Valley Correctional Facility in Columbia County was reported. This outbreak was associated with tuna fish salad which was apparently cross-contaminated by poultry. Reported cases were most prevalent during the late spring and summer months with a peak in June. Monroe, Erie, Suffolk and Nassau counties reported the greatest number of cases. As of week 40 for 1987, 1,366 cases have been reported compared to 1,594 salmonella and 843 shigella cases. Although a similar distribution has been noted, there have been no significant outbreaks.

In a review of 8,837 isolates reported from 42 states to the Centers for Disease Control (CDC) in 1984, Tauxe et al[5] reported a nationwide isolation rate of 4.9/100,000 population in the U.S. The age-specific isolation rate was highest among infants (11/100,000) particularly in the second month of life (17/100,000). A broad peak in the isolation rate was observed in people 15 to 34 years of age with the highest rate in those aged 25 to 29 years (8/100,000).

Marked seasonality was noted, with reporting showing a sharp increase in the summer months for all age groups and all parts of the country. This similarity of seasonal distribution suggested that a single route of transmission could be a major source of Campylobacter. This source is likely to be retail poultry since sporadic infections have been frequently associated with eating undercooked or raw chicken. Campylobacter contamination of retail poultry has been shown to increase in the summer months.

Most cases are sporadic and outbreaks account for only a small proportion of reported cases. Eight outbreaks of Campylobacter infections were reported in the 1984 study; all were due to *C jejuni*. Three were traced to raw milk, four to drinking water and one to foreign travel. The paucity of isolates from children aged two to four from day care centers was thought to argue against person-to-person spread as a significant factor in disease transmission. The relatively high isolation rates among infants and young adults was thought to reflect two major periods of "weaning." In developing countries, Campylobacter infections have been shown to follow infant weaning. The infections in young adults may relate to leaving their family home, a time when they are dependent on foods prepared by themselves or by inexperienced cooks.

Diagnosis

The diagnosis of *Campylobacter enteritis* should be suspected in any individual who has acute onset of watery diarrhea, fever and periumbilical pain. Gross bleeding is seen in a large proportion of adults and children.

Microscopic examination of the stool reveals polymorphonuclear leukocytes in approximately 30% of cases. Approximately 65% will have a peripheral leukocytosis. Examination of fresh stool by phase-contrast microscopy usually demonstrates the characteristic motile, comma-shaped organisms. Gram stain may reveal typical "gull-winged" vibrios in up to two-thirds of cases. Definitive diagnosis depends on culture using selective media which is commercially available. Although most patients mount a humoral antibody response to the autologous organism, no single group antigen has been identified for use in a serologic study.

Treatment

Many cases are self-limited and specific antibiotic therapy is often unnecessary. In more severe cases, oral erythromycin 50 mg/kg/day in four divided doses *may* shorten the duration of clinical disease. Tetracycline may be useful in cases resistant to erythromycin.

Prevention and Control Measures

All food derived from animal sources, particularly poultry, should be thoroughly cooked. All milk should be pasteurized and drinking water chlorinated. Care must be taken during food preparation to prevent cross contamination from raw poultry to other food items. Infections among domestic animals must be recognized and controlled and handwashing after animal contact should be emphasized.

Despite the lack of evidence documenting significant person-to-person transmission, the usual control measures employed for enteric disease are recommended including isolation of hospitalized patients, exclusion of symptomatic individuals from custodial institutions, day care centers, food handling and patient care.[6] In these sensitive situations, treatment may be considered to reduce carriage time and risk of transmission.

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Regional poison control centers

From 1981 to 1985, there were an average of 650 poisoning deaths (3.71/100,000 population) annually in New York State. Rates ranging from 2.41 to 5.41 were observed among the state's eight Health Service Areas (HSAs). (See Table I.)

The statewide average annual rate for suicide deaths by poisoning during the same period was 3.08 per 100,000 population, with a range among the HSAs of 1.19 to 3.08 (see Table II).

Also, during the same period, the statewide average annual rate for unintentional poisoning deaths was 1.02 per 100,000 population, ranging from 0.26 to 2.20 among the HSAs. In addition, during 1983, there were 17,787 hospitalizations for poisoning from drugs, medicinal and biological substances, and another 3,902 hospitalizations from toxic effects from nonmedicinal substances which together accounted for over 143,000 bed-days of acute hospital care.

From 1980 to 1985, 965,125 telephone calls to poison control centers were reported statewide. In addition to providing treatment for people who have ingested harmful substances, poison control centers disseminate information to professionals and the public and participate in the collection of uniform data and research activities to enhance poison control management.

TABLE I. Regional Poison Control Centers

There are 8 regional poison control centers in New York State. In an emergency, contact the regional poison control center serving your county.

Health Service Areas Counties		Poison Control Center
Area 1		
Allegany	Genesee	Western N.Y. Poison Control
Cattaraugus	Niagara	Center at Buffalo
Chautauqua	Orleans	Buffalo Children's Hospital
Erie	Wyoming	219 Bryant Street
		Buffalo, NY 14222
		(716) 878-7654
Area 2		
Chemung	Seneca	Finger Lakes Poison Control
Livingston	Steuben	Center, University of Rochester
Monroe	Wayne	601 Elmwood Avenue, Box 321
Ontario	Yates	Rochester, NY 14642
Schuyler		(716) 275-5151
Area 3		
Cayuga	Oneida	Central N.Y. Poison Control Center
Cortland	Onondaga	University Hospital
Herkimer	Oswego	SUNY Health Science Center
Jefferson	St. Lawrence	750 East Adams Street
Lewis	Tompkins	Syracuse, NY 13210
Madison		(315) 476-4766
Area 4		
Broome		Southern Tier Poison Control Center
Chenango		Binghamton General Hospital
Tioga		Mitchell Street
		Binghamton, NY 13905
		(607) 723-8929
Area 5		
Albany	Montgomery	Ellis Hospital Poison Control Center
Clinton	Otsego	1101 Nott Street
Columbia	Rensselaer	Schenectady, NY 12308
Delaware	Saratoga	(518) 382-4039
Essex	Schenectady	(518) 382-4309
Franklin	Schoharie	
Fulton	Warren	
Greene	Washington	
Hamilton		
Area 6		
Dutchess	Sullivan	Hudson Valley Poison Control Center
Orange	Westchester	Nyack Hospital
Putnam	Ulster	North Midland Avenue
Rockland		Nyack, NY 10960
		(914) 353-1000
Area 7		
Bronx	Richmond	New York City Regional Poison
Kings	Queens	Control Center
New York		New York City Department of Health
		455 First Avenue, Room 123
		New York, NY 10016
		(212) POISONS
		(212) 340-4494
Area 8		
Nassau		Long Island Regional Poison
Suffolk		Control Center
		Nassau County Medical Center
		2201 Hempstead Turnpike
		East Meadow, NY 11554
		(516) 542-2323

TABLE II. Deaths by Poisoning, Rates* Within Health Service Area, New York State 1981–1985

HSA	Suicide	Homicide	Unintentional	Undetermined	Total
1	2.38	0	1.18	1.74	5.31
2	3.08	.02	1.16	.32	4.58
3	2.14	.01	1.42	.32	3.89
4	2.05	0	1.03	.19	3.27
5	1.92	.01	.87	.13	2.93
6	2.09	.02	2.20	.48	4.78
7	1.19	0	.26	.95	2.41
8	2.70	.01	1.90	.80	5.41
Entire State	1.90	.01	1.02	.78	3.71

* Average annual rates per 100,000 population.
Source: New York State Department of Health.

In 1986, Governor Cuomo signed legislation enacting the Poison Control Network Law. The law provides for the New York State Department of Health to designate qualified poison control centers, to establish standards of operation for the centers and to oversee the operation of a statewide poison control network. Under terms of the law, appropriate costs of operation of the centers are reimbursed by the department through the hospital reimbursement process.

Contact your regional poison control center for further information.

Breast cancer treatment alternatives

Breast cancer can take many forms and follow different metastatic paths. There is not a single treatment approach suitable for all breast cancer patients. Rather, the treatment of breast cancer today encompasses many options—surgery, varying from local removal of the tumor to mastectomy, radiation therapy, chemotherapy or hormone therapy. Treatment for each patient with breast cancer must be individualized and based on a number of considerations which include medical history, general health, type and location of the cancer(s), the age and preference of the patient and the considered judgment of the physician.

In order to reach a decision on the treatment method to be used, it is important for the patient to understand the nature of the disease, the extent of the problem, the treatment needed and the method(s) of providing that treatment. Physicians should discuss with their patients the options available, the details of the recommended approach and the reasons for the specific procedure being recommended.

To ensure that patients are being adequately informed about breast cancer treatment options, several states including California, Wisconsin, New York and Massachusetts have enacted legislation known as the Breast Cancer Informed Consent laws which require physicians to inform patients of alternative methods of effectively treating breast cancer.

The New York State law, which went into effect on January 1, 1986, requires physicians in New York State to provide breast cancer patients with a standardized written summary of treatment methods. This summary is to be provided by a physician to each person under his/her care who has been diagnosed with breast cancer as soon after diagnosis as possible.

Prior to the law's implementation, the New York State Department of Health sent an informational packet to 17,500 physicians in the state. In addition to a letter explaining physician obligations to breast cancer patients under the new law, the packet included a booklet entitled *Breast Cancer* which was reviewed by national experts on breast cancer treatment, and a reprinted report on *Limited Surgery and Radiotherapy for Early Breast Cancer.* Of the more than 200 unsolicited comments received from physicians, 82% were favorable.

For additional information on breast cancer, and/or copies of publications on this subject, contact Ms Helen Bzduch, Cancer Prevention Services Program, at (518) 474-1222.

MEDICINE: PRESERVING THE PASSION

By Phil R. Manning, MD, and Lois DeBakey, PHD, 297 pp, illustrated. New York, Springer-Verlag, 1987. $35.00 (hardcover)

This inspiringly written book describes how truly gifted physicians have preserved passion for their profession, maintaining intellectual curiosity, pride in performance, compassion, and enthusiasm while demonstrating the self-discipline needed to set up systematic methods of achieving the lifelong process of continuing their medical education. Today, when so much is being said about competence, we tend to overlook compassionate physicians who care enough about their patients to ask the right questions, and are self-disciplined enough, despite demands on their time and energy, to seek the answers.

Phil Manning, a pioneer and visionary in continuing medical education, along with Lois DeBakey, a scholar and professor of scientific communication, have surveyed 621 physicians, past and present, from a wide range of environments. Topics are discussed via essays and quotes gathered from these medical practitioners and academicians. The book demonstrates how in the medical profession, continuing education is most successful when linked to experience, and shows how these leading clinicians have developed personal, systematic methods of enhancing the linking of education to practice.

Chapters cover those techniques for continuing medical education (CME) that are most practiced and broadly applicable in medical practice. The successful methods described include the most popular styles of reading, and attending formal courses and conferences, as well as the bonuses of learning derived from writing and teaching. Distinguished teachers interviewed explain how they benefit from acquiring, reviewing, and organizing the material to be taught or written. They demonstrate how good teachers make learning a lively affair through the exchange of ideas, resulting in intellectual stimulation and excitement which ultimately turns them into learners. The development of various forms of personal information centers is explained, along with the effective use of the institutional as well as the personal medical library. The art of learning from formal and informal consultations and the collegial network are also well covered. The uses of technology for the analysis of practice, as a guide in diagnosis and therapy, and as an aid to learning are covered in chapters that stimulate excitement by describing learning methods increasingly becoming available to the clinician/learner.

The hope of the authors is that all clinicians will one day adopt simple methods of practice analysis that will allow them to make corrective changes in their practices. Their emphasis is on methods that link education to specific patients' problems. By elegant example we are shown how master physicians continue learning throughout their lives, using written material, lectures, consultation, and, most importantly, experiences with patients to successfully improve their knowledge, skills, and ultimately patient care. Inspiration derived from these great teachers, clinicians, and scientists confirms that lifelong learning is a central focus in the professional lives of successful physicians. The two chapters on women physicians and how families can help seemed out of place, however, by only superficially touching upon important issues.

This is the first book to so thoroughly address methods for implementing individualized learning directly related to physicians' practices. For those involved in today's important issues of quality assurance, relicensure, cost containment, and ethics, it provides a basis of information as to what CME can and cannot do. Sensitive to the clinician's responsibilities and emotions, the book shows MDs as the dedicated humanitarians they have been treated as in the past. Although the traditional lecture will continue to serve a purpose in CME, if the passion of the authors is contagious, we will see a shift of emphasis to individualized education that will meet the specific practice needs of each physician. Reading this book will be an uplifting and inspiring experience and should result in the preservation of the passion kindled by its authors.

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HOME HEALTH CARE

Second Edition. By Allen D. Spiegel, PHD. 442 pp, illustrated. Owings Mills, Md, Rynd Communications, 1987. $49.00 (hardcover)

Home health care is an issue of major concern in the United States today. It is a field that is going to have increasing importance as an element of health care services, for a variety of reasons. It is a modality that appeals to many—particularly for long term care—because it permits a patient to remain in familiar and comfortable surroundings and to avoid the biological hazards, physical discomforts, and personal indignities that are so often associated with care in hospitals and nursing homes. Nevertheless, as noted by Dr Allen Spiegel in the second edition of *Home Health Care*, it remains the focus of continuing debate and unresolved dilemma. Dr Spiegel also notes that there is likely to be continued growth in the home care industry and, despite resistance, increased governmental funding and insurance coverage.

Dr Spiegel has produced a second and very greatly revised edition of the book he first completed in 1983. Considering how extraordinarily current the discussions and the citations are in this new volume, one can accept at face value Dr Spiegel's assertion that the book has been substantially rewritten. As Professor of Preventive Medicine and Community Health at the State University of New York's Health Science Center at Brooklyn, a former Research Fellow at Brandeis University, and a consultant and author on wide-ranging topics in health and medicine, Dr Spiegel is unusually well equipped to write about home health care. He has sought to produce a work that will be of value to professionals, educators, and practitioners as well as to patients, family members, volunteers, and the general public. This is an ambitious undertaking. Although the book leaves some expectations unfulfilled, Dr Spiegel has made a valuable contribution to the literature.

The 12 chapters of this volume cover historical background, definitions, descriptions, advantages and disadvantages, the impact of prospective payment (DRGs), the growing importance of high technology and durable medical equipment, assessment of the need for home care, long term care and services to the elderly, funding of home care, cost analyses and cost-saving strategies, quality of home care, professional and support personnel, volunteers and family members, the growth of home care services, evaluation of home care, and forecasting of future trends, issues and dilemmas.

Dr Spiegel has packed a great deal of information into this volume, which runs well over 400 pages, with 47 tables and charts. Particularly impressive is the excellent organization of a very large amount of material, so that the reader may absorb the information in a logical fashion and, by consulting the table of contents, may quickly locate subjects of particular interest. Every chapter is divided into sections and subsections, all very thoughtfully organized and clearly listed. The index is adequate but somewhat less detailed than one might wish; however, by combined use of the index and the table of contents, the reader should have no difficulty locating a subject. Indeed, almost every topic of interest within the field of home health care is touched upon in this volume.

Dr Spiegel has done a masterful job of studying the literature, organizing it for the reader, and presenting it with an extensive series of bibliographic references which will enable any interested reader to go back to original sources and study them in depth. The material is written with clarity and, at least to this reviewer's eye, is generally very accurate. Typographical errors occur, but they are infrequent and well within acceptable limits.

In one instance Dr Spiegel's interpretation of a table (Exhibit 6.4) seems to miss a point, where he observes that "Medicare expenditures. . .have remained fairly constant over the years, ranging between 1 and 3 percent of total Medicare outputs." To my eye Exhibit 6.4 indicates that reimbursement by Medicare to home health agencies rose steadily from 0.96% of total Medicare reimbursement in 1973 to 3.1% in 1984. I perceive this pattern not as a "fairly constant" one, but rather as an upward trend. Indeed, I believe that this steady rise through the calendar year 1984 accounts, at least in part, for HCFA's subsequent efforts to reduce Medicare home health expenditures by means of denials, eligibility limitations, and reduced reimbursement levels. However, this is a minor cavil and, taken as a whole, Dr Spiegel's statistical analyses seem very much on target.

A more significant problem has to do with the author's basic approach, which may be characterized as reportorial. In places, the book reads almost like a monthly newsletter—reflecting the author's determination to provide the reader with up-to-date information about every aspect of this rapidly moving field. Inevitably, however, such a book will tend to become outdated as today's information is overtaken by tomorrow's events.

Apart from the question of information becoming obsolete or irrelevant, there is also the question of how much detail the reader wants. In the effort to be encyclopedic, Dr Spiegel includes a tremendous amount of detail that is of limited value. For example, it is of only peripheral interest, at best, to read a summary (about half a dozen lines) of the agenda of the 1986 meeting of the American Association for Continuity of Care. The book teeters precariously between being a newsletter and being a more serious work that examines fundamental issues of policy. Indeed, between these two extremes, there is a middle ground which this work also seeks to occupy—that of a handbook dealing with the myriad practical problems with which home care administrators must cope. To this reviewer, it seems that Dr Spiegel has attempted a work that will serve all three purposes—policy analysis, practical handbook, and newsletter of current developments.

Dr Spiegel has met with mixed success in his effort to ride these three horses at once. Although he has done an outstanding job of collating, organizing, and analyzing material from a great number of sources, this encyclopedic approach is a mixed blessing. The reader has the opportunity to read the views of dozens—indeed, hundreds—of different authorities (with meticulous citation of references), but must wade through a good deal of material that is duplicative. For example, in citing the advantages of home care, Dr Spiegel uses three pages to quote four different sources, each listing a number of important advantages. Inevitably, there is great duplication among the four. Given Dr Spiegel's extensive experience and thorough understanding of the field, it would have been preferable for him to produce a single list that would consume much less space and avoid duplication, and would represent Dr Spiegel's balanced view on the basis of his own judgment.

Given Dr Spiegel's very real expertise and well-deserved reputation as an authority, one has a right to expect him to address the serious issues concerning home health care more directly and to share his convictions with the reader. One would like to read much more of Dr Spiegel's judgment and conclusions about fundamental issues in home care—if necessary, at the expense of an encyclopedic approach that depends so heavily upon attribution and citation.

All of that notwithstanding, this is a very useful book. There is almost no aspect of home health care that is overlooked. The reader who is seeking an overview of the field of home health care will have difficulty finding a more comprehensive or more current treatment in any other single volume.

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A PRIMER ON MANAGEMENT FOR REHABILITATION MEDICINE: PHYSICAL MEDICINE AND REHABILITATION

Edited by F. Patrick Maloney, MD. 338 pp, illustrated. Philadelphia, Hanley & Belfus, Inc, 1987. $26.00 (hardcover)

This is a book about the business management aspects of rehabilitation medicine. However, from the title one might misinterpret its intent as meaning clinical management of rehabilitation patients. It is an edited book in which the chief author has written two sections and selected distinguished authorities from administration, management, and medicine for the others.

A second subtitle is *State of the Art Reviews*, and this most clearly describes the contents. The book comprises reviews of the current research and concepts that relate to each section. The sections are relatively brief, being ten to 20 pages in length, and succinctly describe what we know about the subject: "Management," "The Role of the Physician as Manager," "Leadership Styles," "Groups and Decision Making," "Accounting and Finance Basics," "Marketing: Its Meaning and Application in Rehabilitation Medicine," "Organization Structures," "Recruitment and Compensation," "Structure of Service Delivery in Physical Rehabilitation," "Legislation and Regulation of Rehabilitation," "Productivity Issues in Rehabilitation," "Joint Commission on Accreditation of

Hospitals," "Commission on Accreditation of Rehabilitation Facilities," "The Future of Rehabilitation: Delivery of Rehabilitation Services in the 1990's," and a conclusion by the editor. The reviews are of very high quality and the information in them can well be applied to other fields of medicine as well, for these business management elements would vary little in other specialties. The book holds together well and the chapters are well written and accurate.

The book has a place on the physician's bookshelf because of the growing importance of business management elements in medical care. Although there is an increasing number of such publications, this one should stand up well because of its comprehensiveness, clarity, and quality. It clearly meets the objectives as described in the first chapter. However, some physicians may be initially misled by the title into believing that it is a book on clinical management in rehabilitation medicine.

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STARTING IN MEDICAL PRACTICE

By Morton Walker, DPM, with John Parks Trowbridge, MD. 177 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $22.95 (paperback)

Beginning the private practice of medicine has become a greater challenge to the young physician than ever before. The days of hanging out your shingle and waiting for patients to come to your door are over. Government intervention, third-party insurers, and new advances in medical care have made it more difficult to obtain economic security. According to the author of *Starting in Medical Practice*, the obtaining of this economic security is the prerequisite path to a happy life in medicine.

Morton Walker, DPM, has written extensively on medical subjects and is a consultant to two physicians' business newsletters. Walker uses his 17 years of experience as a practicing podiatrist to aid the reader in adopting certain principles of management, thereby obtaining financial security. This book is useful in helping today's doctors know what practice management is all about.

The author details four main concepts of medical practice management which he believes to be the building blocks of a successful practice. Business preparedness, efficiency, solvency, and stability are reviewed and explained. Organized into 14 chapters, the book first guides the reader through the how and where of opening a new medical practice. The options of solo versus group, fee-for-service versus HMO, and academic and research opportunities are discussed. After these decisions are made, organizational topics are reviewed. Chapters cover buying equipment, designing a successful office, and hiring or joining an associate. In addition, hiring employees, setting and collecting fees, and minimizing malpractice risks are reviewed.

The author has made liberal use of comments by physicians, business managers, and consultants regarding the operation of a successful medical practice. A very useful aspect of this text is its numerous sample forms and checklists. Some helpful examples include questions to ask a prospective employee or partner, letters to patients who are delinquent in payment, and evaluation forms for selecting a practice location. An up-to-date bibliography for each chapter is also included.

Unfortunately there are very few good resources for the physician about to enter the world of private practice. *Medical Economics* and *Physician's Management* are two excellent business magazines, but they are not sufficient. Although not mentioned in the book, I found the two-day practice management seminar sponsored by the Medical Society of the State of New York to be an excellent review. In addition, the Medicare carriers in many states also provide a one-day seminar on filing claims. Overall, I feel Walker's book will be very helpful to the majority of physicians just starting out in private practice.

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A GUIDE TO THE UNDERSTANDING OF ALZHEIMER'S DISEASE AND RELATED DISORDERS

Edited by Anthony F. Jorm. 158 pp, illustrated. New York, New York University Press, 1987. $32.00 (hardcover)

Because of the rapidly changing demographics of the world's older adult population, the number of demented patients is increasing to the point where most health care systems will be unable to provide the necessary hospital and institutional care at some time in the future. Dr Jorm, in the book under review here, quotes a projection for the United States that in the year 2030 as much as $30 billion in 1978-value dollars will be spent for the care of demented patients. This amount is many times the current annual expenditure for any health care problem. Besides the tremendous financial costs, there is an incalculable emotional cost to families and health care professionals who care for the victims of Alzheimer disease and other dementing diseases.

A Guide to the Understanding of Alzheimer's Disease and Related Disorders summarizes the current information and theories on these disorders. Besides pathological and biochemical changes observed in Alzheimer disease, the author discusses risk factors, cognitive deficits, and current theories on the etiologies of other dementing diseases. The author wrote the book for students and medical practitioners who are nonspecialists in this area. This reviewer found the book to be clearly written and well organized. The information is up to date and reflects current knowledge on Alzheimer's. The author included several useful chapters on assessment and management of the demented patient. Many of the concepts presented were written for the student and are not of sufficient depth for clinical application. There is little emphasis on differentiating between conditions that cause delirium and permanent dementias.

This book will appeal to health-related and medical professionals who deal with persons with dementing diseases. Because of the author's emphasis on the family's role in therapy, this book could act as a resource for home care and nursing home administrators and health care planners in setting up future "nursing homes without walls" and institutional programs. For the practitioner, this book can be used as a quick reference in the office setting.

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HEALTH PROBLEMS OF HEALTH CARE WORKERS. OCCUPATIONAL MEDICINE

Edited by Edward A. Emmett, MB. 225 pp, illustrated. Philadelphia, Hanley & Belfus, Inc, 1987. $28.00 (hardcover)

This book constitutes the July/September 1987 issue of the *State of the Art Review in Occupational Medicine.* It offers the reader a synopsis of major occupational illnesses affecting health care workers through a format featuring 20 contributing editors who discuss their areas of expertise.

Statistics on the health care sector reveal that practitioners care for health care workers in many different settings. Health care is one of the largest service sectors, and will be employing an estimated 11 million workers by 1990. Nonhospital institutions employ most of these workers. Although accurate data are lacking, the overall injury and illness rates among these workers appear to be significantly higher than for other service industries. Since few health care institutions have employee safety and health programs, local practitioners are the primary source of health care for the large number of workers in these institutions.

The contents of the book include chapters on infectious diseases as well as occupational subjects. The chapters on infectious diseases present human immunodeficiency virus (HIV), hepatitis, and teratogenic virus infections from the perspective of exposures within the health care setting. Extensive references to the *Morbidity and Mortality Weekly Report* (*MMWR*) support specific recommendations for surveillance programs for these diseases. Other chapters address occupational exposure to antineoplastic drugs, laboratory animal allergy, and stress resulting in chemical dependency. These chapters integrate current findings from the recent literature with specific recommendations for preventive measures. There are chapters devoted to indoor air quality, back pain, and the organization and conduct of a hospital occupational health service. All of the chapters present a balance between theoretical insights and practical recommendations.

How current is this state of the art review? To answer this question, I compared the book's recommendations for preventing the transmission of HIV in the health care setting with the *MMWR* suppplement published in September 1987. The book's recommendations matched those given by the more recent *MMWR*. In some instances the supplement presented more details with its recommendations. Nevertheless, a practitioner will perform creditably by following the book's recommendations. Recent references found in all the chapters allow me to assume that the book is indeed current to 1987. This timely presentation of occupational exposures makes this book a useful reference for those practitioners whose patient population includes health care workers.

In addition to practitioners, I recommend that health care administrators review the book, giving special emphasis to the chapter on organization and conduct of a hospital's occupational health service. Executives in the asbestos industry did not initiate worker safety programs until litigation forced them to. Perhaps concerns about HIV, well represented in nearly every chapter, will shape safety standards in the health care industry just as asbestos litigation has done for industrial safety. By reviewing this book health care administrators can become aware of acceptable standards for their employees' health. Active leadership may prevent retroactive litigation.

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OCCUPATIONAL PULMONARY DISEASE. OCCUPATIONAL MEDICINE

Edited by Linda Rosenstock, MD, MPH. 215 pp, illustrated. Philadelphia, Hanley & Belfus Inc, 1987. $28.00 (hardcover)

This volume is the sixth offering on occupational medicine from State of the Art Reviews. The series promises to be a valuable educational and informational medical resource and this particular issue plays a key role. Its intended audience is "practicing clinicians who are involved in the care of working patients." The audience is well served by the book's purpose of providing "knowledge about the nature and extent of occupational pulmonary disease and about optimal approaches to their recognition, treatment and prevention."

Edited by Linda Rosenstock, MD, MPH, the book has brought together the teaching and research expertise of 16 recognized experts in occupational lung disease. The 12 chapters nicely cover the most curent, clinically interesting areas of work-related pulmonary disease in a style that provides the reader with essential information in a concise and easily readable format.

Topics reviewed range from such fundamentals as immunologic evaluation, impairment evaluation, use of spirometry, and a particularly well-written overview of occupational asthma, to specific common entities such as acute inhalation injuries, silicosis, hard metal dust exposure, agricultural exposures, and the variety of concerns involving asbestos. Controversial topics dealing with occupational lung cancer and asbestos-related lung cancer are handled in an objective manner by providing the reader with the most current epidemiologic data and scientifically supported information in these areas. All of the chapters are extensively referenced, allowing for further study if desired. The index allows easy access to specific information when needed.

I found the book easiest to read when I took each chapter as an individual topic, for there was generally no easy flow between chapters. "Evaluation of Impairment and Disability in Occupational Lung Disease" flowed nicely into "Cardiopulmonary Exercise Testing to Assess Respiratory Impairment in Occupational Lung Disease." Also, "Immunologic Evaluation of Occupational Lung Disease" and "Occupational Asthma: Natural History, Evaluation and Management" fit well together. On the other hand, "The Pleural Manifestations of Asbestos Exposure" might have been better placed in a series dealing with asbestos-related lung cancer and occupational lung cancer. "Agricultural Exposure to Organic Dusts," the last article in the book, could have been placed with the chapter on asthma and immunologic evaluation, which appeared much earlier. If it is assumed that the reader has specific narrow interests in the broad field of occupational pulmonary disease and will limit his or her reading to only those chapters of interest, the order in which they appear is of no particular importance.

This book, like its five predecessors in the series, provides the practitioner of occupational medicine with well written, easily understandable, concise, and clinically relevant information which is of immediate use in caring for working patients. This type of ongoing series should be welcomed by the occupational medicine specialist. This particular volume achieves the objectives stated by the editor and also, I am sure, meets the needs of the practitioner. I would recommend it to anyone who deals with patients who work and are exposed to job-related pulmonary hazards.

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HIV-2 associated AIDS

December 1987 marked the first reported case of AIDS in the US caused by human immunodeficiency virus type 2 (HIV-2) (*MMWR* 1988; 37:33–35). The patient had come to the US from West Africa in 1987, but he reported participating in no activities that would have exposed others in this country to HIV-2.

AIDS antibody tests were ordered following a diagnosis of *Toxoplasma gondii* infection of the brain, with no other underlying cause of immunodeficiency—this disease state fits the Centers for Disease Control's (CDC) surveillance definition of AIDS. The patient's serum tested negative for antibody to HIV-1 on enzyme immunoassay (EIA), but was repeatedly reactive on EIA for HIV-2 antibodies. A Western blot test demonstrated bands for antibodies to HIV-2 proteins. Final confirmation of AIDS stemmed from the detection of HIV-2 DNA, but not HIV-1 DNA, in the patient's lymphocytes.

AIDS-associated HIV-2 was first reported in 1986 in West Africa. HIV-2, its associated disease states, and its modes of transmission appear to be similar to those of HIV-1. EIA tests currently used in the US to detect HIV-1 antibodies in donated blood also detect an estimated 42–92% of HIV-2 infections. Because of the presence of HIV-2 in other countries, in January 1987 the CDC and the Food and Drug Administration initiated a surveillance program for HIV-2 in the US.

No consensus found for hospital HIV antibody testing policies

In a rapid response to the first recognition of AIDS in 1981 and the identification of the causative viral agent (human immunodeficiency virus, HIV) in 1983, an HIV antibody test became available for clinical use in 1985. HIV antibody testing quickly provoked controversial legal and ethical issues.

In 1986, Bayer et al (*JAMA* 1986; 256:1768–1774) published recommendations to govern HIV screening; these included the need for patient consent and for counseling of subjects both before and after the test about HIV transmission and infection. A study performed at one hospital from 1985 to 1986 revealed that only 10% of HIV testing met the criteria of Bayer and colleagues. Based on this finding, authors Henry, Willenbring, and Crossley (*JAMA* 1988; 259:1819–1822) evaluated the policies of a group of leading US teaching hospitals and short-term care hospitals in Minnesota regarding the practice of HIV antibody testing.

The authors surveyed 200 US hospitals that offer fellowship training in infectious diseases and 171 short-term care hospitals in Minnesota. They attribute the excellent response rate (94.5% of US hospitals and 93.6% of Minnesota hospitals) to the importance of and "deep interest" in these issues.

The data convey "considerable uncertainty. . .on practical issues germane to the use of HIV antibody testing" at both the national and state levels. Of the participants who responded, 49% of the US teaching hospitals and 37% of the Minnesota hospitals had a policy for ordering HIV antibody tests. A hospital policy recommending but not requiring prior patient consent and patient counseling and educational information existed in 50% of responding US hospitals and 37% of Minnesota hospitals. Forty-seven percent of US hospitals and 39% of Minnesota hospitals acknowledged having a specific educational program for house staff and physicians concerning AIDS and HIV antibody testing. Varying responses were obtained to the question concerning how test results were handled.

The authors noted a lack of uniformity to the responses to most questions on the survey. They stress the need for a consensus on these issues to help hospitals develop policies for HIV antibody testing. "If hospitals are going to contribute significantly to the diminution of HIV transmission, then equal emphasis should be placed on education, as well as the more traditional institutional focus on the care of patients with AIDS."

Identifying the risk factors for ectopic pregnancy

Despite the increasing incidence of ectopic pregnancy in the US—with the frequency at least tripling since 1970—there has been little published research on the risk factors for this disorder. Marchbanks et al (*JAMA* 1988; 259:1823–1827) evaluated 22 potential risk factors and assessed their role in ectopic pregnancy.

This population-based study focused on the residents of Rochester, Minnesota, and, specifically, those recidents who had a diagnosis suggestive of ectopic pregnancy during the 48-year period of 1935 to 1982. Screening the medical records of these individuals led to a final study population of 274 cases. The authors then matched each case to two control women who had delivered live-born infants. The methods of statistical analysis used included univariate matched-triplet, stratified, and multivariable modeling techniques. These yielded odds ratios for the relative risk of ectopic pregnancy.

Using multivariable modeling, the authors identified four statistically significant risk factors: current use of an intrauterine device (IUD), infertility, pelvic inflammatory disease, and tubal surgery. "These risk factors were strong and independent." Based on univariate analyses, five other risk factors would join this list: abdominal/pelvic surgery, acute appendicitis, adhesions, clomiphene use, and induced abortion. Elevated relative risk, although not statistically significant, was found for women with a history of anovulation, in utero exposure to diethylstilbestrol, myomas, ovarian cysts, or past IUD use.

This study produced other interesting findings. For example, a history of oral contraceptive use was not associated with an increased relative risk of ectopic pregnancy. A history of endometriosis was also not considered to be a risk factor.

In a related editorial (*JAMA* 1988; 259:1862–1864), Robert N. Taylor, MD, PHD, from the University of California—San Francisco, commented on the "apparent epi-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

demic of ectopic pregnancy." He attributes it to three general factors, all of which arose due to technological advances in the field of gynecology: "(1) increased prevalence of tubal infection, (2) iatrogenic causes associated with fertility control, and (3) perceived increases in incidence due to improved diagnostic methodology."

The increased incidence of tubal infection, if viewed in association with the increasing prevalence of *Chlamydia trachomatis*, could lead to the conclusion that "chlamydial salpingitis may be particularly likely to cause ectopic pregnancy." Persistent and recurrent infection with this pathogen, often with no symptoms, can cause chronic tubal inflammation and impaired tubal function. With respect to the second factor, iatrogenic causes related to therapies for infertility, Taylor associates the increasing use of ovulation induction and tubal surgery with the increasing prevalence of ectopic pregnancy.

Taylor finds some fault with the study population chosen by Marchbanks et al, stating that the residents of Rochester, Minnesota, "may not represent the modern, urban patient with whom we associate the greatest likelihood for the development of this condition. Moreover, this era antedates current state-of-the-art diagnostic and therapeutic methods."

FDA approves alteplase and streptokinase for intravenous use

The Food and Drug Administration (FDA) has granted marketing approval for alteplase (trade name Activase; Genentech Inc, South San Francisco, Calif) in the treatment of acute myocardial infarction in adults. Alteplase is a tissue plasminogen activator produced by recombinant DNA technology.

The drug, approved for intravenous administration, is effective for lysing thrombi that obstruct coronary arteries, improving ventricular function, and reducing the incidence of congestive heart failure following acute myocardial infarction. Alteplase produces conversion of plasminogen to plasmin in the presence of fibrin. In a thrombus, this process stimulates local fibrinolysis, while producing limited systemic proteolysis.

In addition, the FDA approved relabeling of streptokinase for intravenous administration. It was previously indicated for intracoronary use in lysing thrombi. The FDA recommends rapid institution of alteplase or streptokinase therapy following onset of symptoms of acute myocardial infarction.

FDA issues new warnings for isotretinoin and nonprescription ocular products

The FDA's earlier warnings that isotretinoin (trade name Accutane), a drug used to treat severe cases of cystic acne, should not be prescribed for women of childbearing age who are or could become pregnant while taking the drug, were reinforced by a recent article in *Morbidity and Mortality Weekly Report* (March 25, 1988). Multiple, serious birth defects involving the brain, cardiovascular system, and face occurred in four infants born to mothers who had taken isotretinoin during the first trimester of pregnancy. Despite these risks, the FDA reports use of the drug by thousands of women of childbearing age who do not even require such intensive treatment. Isotretinoin is indicated only for severe recalcitrant cystic acne that does not respond to conventional therapy.

As of March 4, 1989, the FDA will also require warnings against long term use, except under a doctor's supervision, of nonprescription products currently on the market for treatment of dry, red eyes. To date, the FDA has found safe and effective four ingredients to treat redness, more than 20 ingredients to protect the eye from minor irritations or to relieve dryness, and one astringent ingredient to relieve minor eye irritations.

The FDA will require an additional warning for eyecare products that contain mercury compounds as preservatives. These additives can cause allergic reactions in some people.

Reducing the physical and financial toll of softball injuries

A previous, retrospective study determined that 71% of recreational softball injuries occurred as a result of base sliding. These injuries, which included abrasions, sprains, ligament strains, and fractures, produced physical suffering and costly insurance claims. With an estimated 40 million persons nationwide participating in organized softball leagues, according to the American Softball Association, the frequency of base sliding injuries becomes quite substantial.

Janda et al (*JAMA* 1988; 259:1848–1850) outlined the proposed methods for reducing base sliding injuries: "abolition of sliding, better instruction of sliding techniques, the use of recessed bases, and the use of quick-release bases." Based on choosing the method they would expect to be most readily accepted by softball players, officials, and fans, and because most sliding injuries are caused by rapid deceleration impact against stationary bases, the authors believe that quick-release bases might effectively reduce the incidence of sliding injuries.

They conducted a prospective study comparing standard and breakaway bases. The breakaway base is attached to a rubber mat, which is flush with the infield surface and anchored to the ground with a metal post. "One fifth of the force needed to dislodge a stationary base from its mooring is required for the breakaway portion of the base to release."

Base sliding injuries were recorded and evaluated for 633 games played using breakaway bases and 627 games played with stationary bases. Of the total 47 sliding injuries recorded, 45 occurred on fields with stationary bases, and two occurred on fields with breakaway bases—a statistically significant ($p<0.001$) difference. For the latter two injuries, the bases did not break away.

Comparing the medical charges for these two groups, the authors calculated approximate total charges of $55,050 ($1,223/injury) for the group of 45 injured players, and about $700 ($35/injury) for the two players who suffered injuries on fields with breakaway bases. The authors concluded that the use of breakaway bases could produce a "significant reduction in the quantity and the resultant morbidity of softball injuries."

Visit of second-grade students from Shelter Rock Elementary School, Manhasset, NY

On April 13, Mrs Margaret Griffith-Miles and her class of second graders from Manhasset's Shelter Rock Elementary School visited the Journal's *offices. The students' visit was undertaken as part of their study of the production of goods and services. After a tour of the* Journal *offices, members of the staff gave the students a description of the various phases of the editing and production processes. On returning to school the students wrote reports on what they learned during their visit.*

The following are extracts from their reports:

"I enjoyed the trip to *New York State Journal of Medicine.* I learned a lot about editing. . . . There are 17 articles in a month that you choose and goes in a magazine. Before that you send the articles to two people and they don't know the people who wrote the article. The two people give them grades. They are A, B, C, D, and E. A and B are the two best grades. C, D, and E are not good grades. . . . Thank you for the tour, cookies and milk, and the magazine. I found out that you are a producer of a good because you make magazines for people to read. Thank you."

Kevin Connolly

"I really liked the tour at the *New York State Journal of Medicine.* I really want to read your magazine. We met Mrs Abney (the Librarian), Mrs Burns (the Assistant Librarian), Ms Moore (the Managing Editor), and Dr Imperato. They make 29,000 copies of the *New York State Journal of Medicine* each month. The editor reads all the articles first, corrects English. Dr Imperato sends articles to two people, one in New York and one outside New York. The two people gives the grades A, B, C, D, or E. They make dummies (graphs, pictures, and words) of the articles. In the first page of the magazine there is a table of contents. The doctors write the articles. They keep good articles that they get. And they don't keep the ones that are bad. . . . Thank you."

Sharif Fayache

Managing Editor Carol Moore describes the editing/production process. Mrs Griffith-Miles is seated at the far left.

Journal Secretary Elizabeth Somers demonstrates the use of the word processor and printer.

"I learned so much and I'll tell you what I learned. I learned that you produce 29,000 magazines a month. I learned that you get articles from doctors. I learned that you send articles to two people. . . . Also I learned that an editor reads all the articles. Sometimes you don't feel like putting a doctor's informational article in the magazine because it's not good information. And on dummies you find pictures, words, and graphs. . . . I met Mrs Burns the assistant librarian. I met Ms Moore who is the managing editor and she reads articles. There's advertisements, table of contents. I loved meeting you and your helpers. . . . Oh, I can't forget, thanks for the cookies and milk. I had a wonderful time. Oh, and all of you people out there, you should go to the *New York State Journal of Medicine.* Thank you."

Brooke Gordon

"Thank you for letting us come to your job. . . . I found out that you have to write not just a first draft but sometimes you have to revise ten times and on one you only had to revise three times. . . . Thank you for the magazine. I also found out that you are a producer of a service and goods. Thank you."

Maura Hauk

"I really enjoyed coming to your office. I found it very interesting. . . . I never knew that you produce nearly 25,000 or 30,000 magazines a month. . . . It's also interesting to know most offices have a Fax machine. I think you're a producer of both a service and goods because you make magazines and sell them. Love,"

Gavin Imperato

"I got a lot of information. The library was pretty. I didn't know that the magazine is called the *New York Journal of Medicine.* I didn't know that the medical journal was all over the world. Ms Moore told us that in the medical journal there are dummies. . . . Thank you for the cookies and milk. Love, your friend,"

Sandra Jimenez

"I had a good time. I liked when you showed us the pictures of the people in the magazine. I like the printer. . . . There was a woman that would check the books to see a long time ago what doctors would say. We saw two receptionists. One of the receptionists answered the telephone. At the end we got cookies and milk. Thank you."

Jennifer Kassl

"Thank you for letting us have the opportunity to eat milk and cookies because you said that you're not allowed to eat milk and cookies [in the library]. I liked when I got to push a button to make the printer work. Thank you for letting me and my classmates have an opportunity to go to your business. And letting us have your magazine. . . . Thank you."

Gary LaMarca

"Thank you for the milk and cookies. I had fun! Thanks for the information! My favorite part was when we ate milk and cookies! I liked the trip! My favorite thing was the computer! . . . Your friend,"

Michael Lapore

"I really liked all the information that you gave us about the magazine. And how you should send in an article. When you get the article you read it and then Ms Moore reads it. Then she sends it to two people that the person does not know. And they will read it. They will grade it ABCDE. An "E" is not good. A "D" is not good either. And then when the grading is over they send it back to the person and the person will recopy it. And I also liked the milk and cookies. And I also think you're a producer of goods and a service. Thank you."

Carla Laur

"Thank you for the milk and cookies. I really learned a very lot of things. I hope I go again. . . . My best thing was the computer because I really like computers and because I love writing on computers too. I think you are a producer of a good."

Chip McManus

"Thank you for all the information you gave us. Thank you for the cookies and milk. I learned a lot from you. I would be glad to see you again. I liked the printer because when it printed it printed back and forth. I want to read the medical journal. . . . I would love to be a doctor so I could read the medical journal. I think that if you're going be a editor you should know how to make a first draft, revise, edit, and make a final copy. Thank you for the tour. Thank you."

Livia Mello

"It all began when we got there. I liked the garden. And I think that the rest of the class did too. Dr Imperato, you really told the class a lot about the *New York State Journal of Medicine*. I think I might even come and work there when I get older! It's complicated, so what! You really treated us nice. I really liked it. Thank you for the milk and cookies. Thank you again."

Matthew Mitchell

"I didn't know what a dummy is. You taught me a lot of things about medicine and the magazine. I heard you saying most people prefer the contents on the cover of the magazine. If I become a neurosurgeon then I have to know about medicine. And I do want to become a neurosurgeon. I didn't know that doctors have to revise at least ten times. . . . I learned it's pretty hard to be an editor. I never saw such a fast printer in my life. I liked hearing your information about being an editor. Good Luck! I liked everybody there! You are a producer because you sell magazines and you make them. P.S. Thank you for the milk and cookies! Love,"

Michele Nelson

"I liked the big printer and the cookies and milk. Thank you for the magazine. Thank you. Your friend,"

Jill Pond

"Thank you for your magnificent tour of the Society. I learned why you have advertising in the magazine, you get to have thirty-thousand a month, what a dummy is, what overrun means. . . . that you store all your book reviews on a computer. . . . Today when I get home, I'm gonna tell all I can about my appreciation about this trip. If I can find an order form for subscribing to your journals I'll fill it out. I thank you for all the enjoyment you let me have. . . . I also found out that most companies use fax machines. Thanks for all the things that I learned. This was about the best trip I ever had. Ever!!!! Thank you,"

Christopher Shin

"I really liked you and your people a lot. I've never been to a publisher before. . . . I liked the way you changed your magazine from the colorful front to the black and white front. I think that was a smart thing you did. Thank you,"

Graham Siener

"I liked the cookies and milk. I learned that you sometimes have to read articles over eight times, sometimes ten. The lady on the computer told us how to work it. . . . Thank you."

Salvatore Simeone

"I liked the librarian, assistant librarian, and Ms Moore. I had fun watching the computer. I liked the cookies and milk. I learned about editing. I didn't know that the content was on the cover. . . . Thank you for teaching us about editing and publishing. You are both a kind of producer—a producer of a good and a service. Thank you."

Christina Tenaglia

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Charles Abler, MD, Bronx. Died February 2, 1988; age 78. Dr Abler was a 1935 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. Dr Abler was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Arthur Abramson, MD, New York. Died January 26, 1988; age 86. Dr Abramson was a 1929 graduate of New York University School of Medicine, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Rogelio Jose Barata, MD, Union City, New Jersey. Died September 26, 1987; age 75. He was a 1940 graduate of Facultad de Medecina de la Universidad de La Habana, La Habana, Cuba. Dr Barata was a Fellow of the American College of Surgeons and the American College of Chest Physicians and a Diplomate of the American Board of Surgery and the American Board of Thoracic Surgery (affiliated with the American Board of Surgery). He was a member of the New York County Medical Society and the Medical Society of the State of New York.

James French Benedict, MD, Penn Yan. Died February 21, 1988; age 70. Dr Benedict was a 1944 graduate of State University of New York Health Science Center at Syracuse. He was a member of the Academy of Medicine, the Medical Society of the County of Yates, and the Medical Society of the State of New York.

Edwin Benjamin Bilchick, MD, New York. Died July 27, 1987; age 86. He was a 1924 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Bilchick was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the Academy of Medicine, the American Academy of Otolaryngology, the American Academy of Facial Plastic and Reconstructive Surgery, the Nassau County Medical Society, and the Medical Society of the State of New York.

Julius Sidney Blier, MD, Bronx. Died January 16, 1988; age 77. Dr Blier was a 1937 graduate of the University of Vermont College of Medicine, Burlington. He was a Diplomate of the American Board of Internal Medicine and a member of the Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Edward Joseph Bonavilla, MD, Rochester. Died March 6, 1988; age 46. Dr Bonavilla was a 1967 graduate of the State University of New York Health Science Center at Syracuse. He was a Diplomate of the American Board of Ophthalmology and a member of the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

John J. Buckley, MD, Stamford, Connecticut. Died November 24, 1987; age 71. Dr Buckley was a 1944 graduate of Medizinische Fakultaet der Universitaet Zurich, Zurich, Switzerland. He was a Diplomate of the American Board of Pathology and a member of the Medical Society of the County of St Lawrence and the Medical Society of the State of New York.

Evelyn T. Clerico, MD, New York. Died December 1987; age 79. Dr Clerico was a 1932 graduate of Middlesex University School of Medicine, Waltham. She was a Fellow of the American College of Anesthesiologists. Her memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

George Eaton Daniels, MD, New York, Died August 11, 1987; age 91. He was a 1922 graduate of Harvard Medical School, Boston. Dr Daniels was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Academy of Medicine, the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society, and the Medical Society of the State of New York.

Arthur Maxwell Davids, MD, Bridgehampton. Died January 28, 1988; age 81. He was a 1934 graduate of New York University School of Medicine, New York. Dr Davids was a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Academy of Medicine, the New York Obstetrical Society, the New York County Medical Society, and the Medical Society of the State of New York.

David Murray Davidson, MD, Bay Harbor Islands, Florida. Died October 26, 1987; age 87. Dr Davidson was a 1925 graduate of Rijksuniversiteit te Gent, Faculte der Geneeskunde, Gent, Belgium. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

John M. Doyle, MD, Binghamton. Died March 10, 1988; age 61. Dr Doyle was a 1951 graduate of the University of London Faculty of Medicine, London, England. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Joseph Eisenstein, MD, Long Beach. Died February 8, 1988; age 67. He was a 1965 graduate of Middlesex University School of Medicine, Waltham. Dr Eisenstein was a Diplomate of the American Board of Family Practice and a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Sigmund Falk, MD, Clearwater, Florida. Died January 12, 1988; age 85. Dr Falk was a 1935 graduate of Faculty of Medicine, University of Edinburgh, Edinburgh, Scotland. He was a member of the Clinical Society, New York Diabetes Association, the American Academy of Family Practice, the New York County Medical Society, and the Medical Society of the State of New York.

Naomi Fortgang, MD, New York. Died November 15, 1987; age 66. Dr Fortgang was a 1944 graduate of the Medical College of Pennsylvania, Philadelphia. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Max Fratkin, MD, Brookline, Massachusetts. Died December 16, 1987; age 76. He was a 1936 graduate of Faculte de Medicine de l'Universite de Geneve, Geneva, Switzerland. Dr Fratkin was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. He was a member of the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Oscar Samuel Glatt, MD, Lido Beach. Died October 22, 1987; age 83. Dr Glatt was a 1933 graduate of Faculte de Medicina de l'Universite de Geneve, Geneva, Switzerland. He was a member of the New York State Dermatological Society, the American Academy of Dermatology, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Hans Hirschfeld, MD, Sea Cliff. Died May 1987; age 88. He was a 1923 graduate of Medizinische Fakultaet der Martin Luther Universitaet, Halle, Germany. Dr Hirschfeld was a Qualified Fellow of the International College of Surgeons and a member of the New York County Medical Society and the Medical Society of the State of New York.

Robert John Hubbard, MD, Brooklyn. Died February 9, 1988; age 69. Dr Hubbard was a 1943 graduate of the State University of New York Health Science Center at Brooklyn. Dr Hubbard was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the New York Gastroenterological Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Henry W. Kaessler, MD, Mount Vernon. Died February 8, 1988; age 83. He was a 1930 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Kaessler was a Fellow of the American Academy of Pediatrics, a Diplomate of the American Board of Pediatrics, and a member of the Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Louis T. Kirshenbaum, MD, New York. Died November 22, 1987; age 90. Dr Kirschenbaum was a 1921 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Motilal M. Khubchandani, MD, Tonawanda. Died February 3, 1988; age 54. He was a 1962 graduate of B. J. Medical College, Gujarat University, Ahmedabad, Gujarat, India. Dr Khubchandani was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Colon and Rectal Surgery. His memberships included the American Society of Colon and Rectal Surgeons, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Salem Kooby, MD, Syosset. Died January 7, 1987; age 83. Dr Kooby was a 1933 graduate of Faculte Francaise de Medicine et de Pharmacie de l'Universite, St Joseph, Beyrouth, Lebanon. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Rudolf Kurcz, MD, New York. Died December 27, 1987; age 83. Dr Kurcz was a 1929 graduate of Universitaet Wien, Medizinische Fakultaet Wien, Austria. He was a member of the American Academy of Family Practice, the New York County Medical Society, and the Medical Society of the State of New York.

Anthony J. LaSala, MD, Brooklyn. Died March 7, 1988; age 61. Dr LaSala was a 1956 graduate of Facolta di Medicina e Chirurgia dell' Universita di Bologna, Bologna, Italy. He was a Diplomate of the American Board of Obstetrics and Gynecology and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Nathan Leifer, MD, Dix Hills. Died October 17, 1987; age 87. Dr Leifer was a 1924 graduate of New York Medical College, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Charles Kantor Levy, MD, Kew Gardens Hills. Died November 18, 1987; age 87. He was a 1924 graduate of the State University of New York Health Science Center at Brooklyn. Dr Levy was a Fellow of the American College of Obstetricians and Gynecologists and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the American Society of Abdominal Surgeons, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Robert Samuel Liebert, MD, New York. Died March 9, 1988; age 58. Dr Liebert was a 1960 graduate of New York University School of Medicine, New York. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Margaret Mary Loder, MD, Rye. Died February 8, 1988; age 94. She was a 1925 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. Dr Loder was a Fellow of the American College of Pathologists and a Diplomate of the American Board of Pathology. Her memberships included the New York Pathological Society, the American Association for Cancer Research, the American Society of Clinical Pathologists, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Rafael A. Marin, MD, New York. Died May 30, 1987; age 87. Dr Marin was a 1932 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine and a member of the New York County

Medical Society and the Medical Society of the State of New York.

Robert George Mensing, MD, Massapequa. Died March 5, 1988; age 61. Dr Mensing was a 1958 graduate of Tulane University School of Medicine, New Orleans. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Norbert Menuhin, MD, Newark. Died February 1988; age 77. Dr Menuhin was a 1936 graduate of Universitaet Leipzig Medizinische Fakultaet, Saxony, Germany. He was a member of the American Academy of Family Practice, the Medical Society of the County of Wayne, and the Medical Society of the State of New York.

Peter John Milazzo, MD, Corona. Died October 3, 1987; age 86. He was a 1928 graduate of George Washington University School of Medicine, Washington, DC. Dr Milazzo was a Fellow of the American Academy of Family Practice and a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Amelia M. Milukas, MD, Richmond Hill. Died February 6, 1988; age 71. Dr Milukas was a 1941 graduate of Vytauto Didziojo University Medical Fakelteto, Kaunas (Extinct) Lithuania. She was a member of the American Occupational Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Richmond L. Moore, MD, Lynchburg, Virginia. Died March 7, 1988; age 92. Dr Richmond was a 1922 graduate of Harvard Medical School, Boston. Dr Moore was a member of the New York County Medical Society and the Medical Society of the State of New York.

Claudio F. Norambuena, MD, Brooklyn. Died September 1, 1987; age 47. Dr Norambuena was a 1967 graduate of Universidad de Chile, Escuela de Medicina de Santiago, Santiago, Chile. He was a Fellow of the American College of Anesthesiologists. His memberships included the New York Ophthalmology Society, the American Ophthalmological Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Richard E. Passenger, MD, Newburgh. Died January 25, 1988; age 72. He was a 1941 graduate of Albany Medical College of Union University, Albany. Dr Passenger was a member of the American Academy of Allergy, the Medical Society of the County of Orange, and the Medical Society of the State of New York.

Armando Patrizio, MD, Brooklyn. Died February 23, 1988; age 57. Dr Patrizio was a 1955 graduate of Facolta di Medicina e Chirurgia dell' Universita di Napoli, Naples, Italy. He was a Diplomate of the American Board of Family Practice. His memberships included the American Academy of Family Practice, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Irving Sydney Pearlman, MD, Longboat Key, Florida. Died February 1, 1988; age 93. Dr Pearlman was a 1921 graduate of Northwestern University Medical School, Chicago. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Sidney I. Rabinowitz, MD, Flushing. Died February 7, 1988; age 54. Dr Rabinowitz was a 1959 graduate of Faculte de Medecine de l'Universite de Geneve, Geneva, Switzerland. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Milton Joseph Raisbeck, MD, San Francisco, California. Died February 1, 1988; age 99. Dr Raisbeck was a 1916 graduate of New York Medical College, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Arnold Orson Riley, MD, Brockport. Died February 3, 1988; age 72. He was a 1942 graduate of Albany Medical College of Union University, Albany. Dr Riley was a member of the American Academy of Family Practice, the Medical Society of the County of Orleans, and the Medical Society of the State of New York.

Joseph Rizzo, MD, Forest Hills. Died November 1987; age 91. Dr Rizzo was a 1927 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. He was a Fellow of the American College of Surgeons and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Marc Robbins, MD, Rockaway Park. Died April 5, 1987; age 89. He was a 1920 graduate of New York University School of Medicine, New York. Dr Robbins was a member of the New York Cardiological Society, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Felix Joseph Rotoli, MD, Rochester. Died January 30, 1988; age 77. Dr Rotoli was a 1938 graduate of Loyola University Stritch School of Medicine, Maywood. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

John Lester Schultz, MD, Penn Yan. Died March 10, 1988; age 70. Dr Shultz was a 1944 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the Medical Society of the County of Yates and the Medical Society of the State of New York.

Philip J. Schultze, MD, Brooklyn. Died March 7, 1988; age 79. He received Registrable Qualification Granted by the English Conjoint Board in 1937. Dr Schultze was a member of the New York Cardiological Society, the American Society of Compensation Medicine, Inc, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Beverly Chew Smith, MD, New York. Died March 2, 1988; age 92. Dr Smith was a 1919 graduate of the University of Virginia School of Medicine, Charlottesville. She was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. Her memberships included the Academy of Medicine, the New York State Surgical Society, the Clinical Society of the New York Diabetes Association, the New York County Medical Society, and the Medical Society of the State of New York.

Charles Francis Snopek, MD, Conway, South Carolina. Died January 26, 1988; age 88. Dr Snopek was a 1925 graduate of the State University of Iowa College of Medicine, Iowa City.

He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

David Stockton Speer, MD, New York. Died January 9, 1988; age 72. He was a 1943 graduate of Harvard Medical School, Boston. Dr Speer was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the Academy of Medicine, the American Geriatrics Society, the American Medical Society for Alcoholism, the New York County Medical Society, and the Medical Society of the State of New York.

Mario E. Stella, MD, Lido Beach. Died February 27, 1988; age 81. He was a 1930 graduate of New York University School of Medicine, New York. Dr Stella was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the Academy of Medicine, the American Academy of Orthopaedic Surgeons, the American Academy of Compensation Medicine, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

James Wendell Watson, MD, New York. Died December 28, 1987; age 73. Dr Watson was a 1943 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

George Weiss, MD, Rockville, Maryland. Died September 21, 1987; age 86. He was a 1925 graduate of New York University School of Medicine, New York. Dr Weiss was a Fellow of the American Academy of Pediatrics, a Diplomate of the American Board of Pediatrics, and a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Norman J. Wolf, MD, Buffalo. Died February 1, 1988; age 86. Dr Wolf was a 1927 graduate of State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the American Society of Abdominal Surgeons, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Henry Young, MD, Bloomingburg. Died November 2, 1987; age 74. He was a 1936 graduate of New York University School of Medicine, New York. Dr Young was a Fellow of the American College of Chest Physicians, the American College of Cardiology, and the American College of Physicians, and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the New York Society for Thoracic Surgery, the American Public Health Association, the American Thoracic Society, the American Occupational Medical Association, the American Academy of Occupational Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Robert Landesman, MD
1916–1987

Dr Robert Landesman, Professor Emeritus of Obstetrics and Gynecology at the Cornell University Medical College, Honorary Attending at the New York Hospital, and member of the *Journal's* Associate Editorial Board, died Sunday, December 6, 1987. He was 71 years old.

Dr Landesman was internationally known for having done some of the earliest, most significant research on the treatment of toxemia of pregnancy, the arrest of premature labor, and the early diagnosis of pregnancy using radioimmune assays. He also had a long and distinguished career as a practitioner and teacher.

Born in New York City on May 6, 1916, Dr Landesman graduated from Columbia College in 1936 and from Cornell University Medical College in 1939. From 1939 to 1940 he was a resident in pathology and from 1940 to 1942 an intern and assistant resident in surgery at Mount Sinai Hospital in New York City. In 1942 he joined the United States Air Force and served as a flight surgeon in the Pacific Theater, being discharged as a major. Upon his return from the military he did his residency training in obstetrics and gynecology at New York Hospital and Mount Sinai Hospital from 1946 to 1949. He then embarked on his long and distinguished career as a physician and scholar. He was a major contributor to medical journals and texts, having published 80 papers. Dr Landesman was a Fellow of both the American College of Obstetrics and Gynecology and the American College of Surgery. He also was a Diplomate of the American Board of Obstetrics and Gynecology and a member of the New York Obstetrical Society.

Dr Landesman served as a member of the Associate Editorial Board of the *New York State Journal of Medicine* since 1984. He provided dedicated service to the *Journal* as a peer reviewer and contributor. His conscientious efforts greatly enhanced the continued scientific quality of the *Journal*. On April 21, 1988, Dr Landesman was posthumously awarded the William Hammond Citation for Distinguished Service to the *Journal* by the Medical Society of the State of New York.

Dr Landesman is survived by his three children, Lucy Halperin and Peter and Paul Landesman, two grandchildren, and Ernestine Libros.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83: 925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Your Professional Advantage

Membership in your county medical society and the Medical Society of the State of New York (MSSNY) means that you have the pooled resources of the medical profession throughout New York State working in your interest. You benefit from society activities in the following areas:

Advocacy: Each year, hundreds of bills are filed in the legislature that affect medical practice, teaching, research, public health or medical economics. The MSSNY monitors and responds to these bills on behalf of physicians and their patients, and sponsors legislation of its own concerning medicine and health in the State of New York. Your county medical society is your advocate on local issues and gives you direct input in establishing State Society policy.

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C I B A

References: 1. Data on file, CIBA Pharmaceutical Company **2.** Skoutakis VA, Acchiardo SR, Wojciechowski NJ, et al. Liquid and solid potassium chloride: Bioavailability and safety. *Pharmacotherapy* 1980;4(6):392-397. **3.** Skoutakis VA, Carter CA, Acchiardo SR: Therapeutic assessment of Slow-K and K-Tab potassium chloride formulations in hypertensive patients treated with thiazide diuretics. *Drug Intell Clin Pharm* 1987;21:436-440

Slow-K®
potassium chloride USP
Slow-Release Tablets
8 mEq (600 mg)

BRIEF SUMMARY (FOR FULL PRESCRIBING INFORMATION SEE PACKAGE INSERT)

INDICATIONS AND USAGE

BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS.

1 For therapeutic use in patients with hypokalemia with or without metabolic alkalosis; in digitalis intoxication and in patients with hypokalemic familial periodic paralysis.

2. For prevention of potassium depletion when the dietary intake of potassium is inadequate in the following conditions: patients receiving digitalis and diuretics for congestive heart failure; hepatic cirrhosis with ascites; states of aldosterone excess with normal renal function; potassium-losing nephropathy; and certain diarrheal states

3. The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases supplementation with potassium salts may be indicated.

CONTRAINDICATIONS

Potassium supplements are contraindicated in patients with hyperkalemia, since a further increase in serum potassium concentration in such patients can produce cardiac arrest. Hyperkalemia may complicate any of the following conditions: chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e.g., spironolactone, triamterene) (see OVERDOSAGE).

All solid dosage forms of potassium supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract. In these instances, potassium supplementation should be with a liquid preparation. Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to an enlarged left atrium.

WARNINGS

Hyperkalemia (See OVERDOSAGE)

In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally. Potentially fatal hyperkalemia can develop rapidly and be asymptomatic

The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment.

Interaction With Potassium-Sparing Diuretics

Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e.g., spironolactone or triamterene), since the simultaneous administration of these agents can produce severe hyperkalemia.

Gastrointestinal Lesions

Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths. These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage, or perforation. Slow-K is a wax-matrix tablet formulated to provide a controlled rate of release of potassium chloride and thus to minimize the possibility of a high local concentration of potassium ion near the bowel wall. While the reported frequency of small-bowel lesions is much less with wax-matrix tablets (less than one per 100,000 patient-years) than with enteric-coated potassium chloride tablets (40-50 per 100,000 patient-years) cases associated with wax-matrix tablets have been reported both in foreign countries and in the United States. In addition, perhaps because the wax-matrix preparations are not enteric-coated and release potassium in the stomach, there have been reports of upper gastrointestinal bleeding associated with these products. The total number of gastrointestinal lesions remains approximately one per 100,000 patient-years. Slow-K should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs.

Metabolic Acidosis

Hypokalemia in patients with metabolic *acidosis* should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, or potassium acetate.

PRECAUTIONS

General:

The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis *per* se can produce hypokalemia in the absence of a deficit in total body potassium, while acute acidosis *per* se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium.

Information for Patients

Physicians should consider reminding the patient of the following

To take each dose without crushing, chewing, or sucking the tablets.

To take this medicine only as directed. This is especially important if the patient is also taking both diuretics and digitalis preparations

To check with the physician if there is trouble swallowing tablets or if the tablets seem to stick in the throat.

To check with the doctor at once if tarry stools or other evidence of gastrointestinal bleeding is noticed

Laboratory Tests

Regular serum potassium determinations are recommended. In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis

Drug Interactions

Potassium-sparing diuretics: see WARNINGS.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Long-term carcinogenicity studies in animals have not been performed.

Pregnancy Category C

Animal reproduction studies have not been conducted with Slow-K. It is also not known whether Slow-K can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Slow-K should be given to a pregnant woman only if clearly needed.

Nursing Mothers

The normal potassium ion content of human milk is about 13 mEq/L. It is not known if Slow-K has an effect on this content Caution should be exercised when Slow-K is administered to a nursing woman.

Pediatric Use

Safety and effectiveness in children have not been established

ADVERSE REACTIONS

One of the most severe adverse effects is hyperkalemia (see CONTRAINDICATIONS, WARNINGS, and OVERDOSAGE). There also have been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see CONTRAINDICATIONS and WARNINGS); other factors known to be associated with such conditions were present in many of these patients.

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose.

Skin rash has been reported rarely.

OVERDOSAGE

The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see CONTRAINDICATIONS and WARNINGS). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration (6.5-8.0 mEq/L) and characteristic electrocardiographic changes (peaking of T waves, loss of P wave, depression of S-T segment, and prolongation of the Q-T interval) Late manifestations include muscle paralysis and cardiovascular collapse from cardiac arrest (9-12 mEq/L).

Treatment measures for hyperkalemia include the following: (1) elimination of foods and medications containing potassium and of potassium-sparing diuretics; (2) intravenous administration of 300-500 ml/hr of 10% dextrose solution containing 10-20 units of insulin per 1,000 ml; (3) correction of acidosis, if present, with intravenous sodium bicarbonate; (4) use of exchange resins, hemodialysis, or peritoneal dialysis.

In treating hyperkalemia in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity.

DOSAGE AND ADMINISTRATION

The usual dietary intake of potassium by the average adult is 40-80 mEq per day. Potassium depletion sufficient to cause hypokalemia usually requires the loss of 200 or more mEq of potassium from the total body store. Dosage must be adjusted to the individual needs of each patient but is typically in the range of 20 mEq per day for the prevention of hypokalemia to 40-100 mEq or more per day for the treatment of potassium depletion. Large numbers of tablets should be given in divided doses.

Note: Slow-K slow-release tablets must be swallowed whole and never crushed, chewed, or sucked.

HOW SUPPLIED

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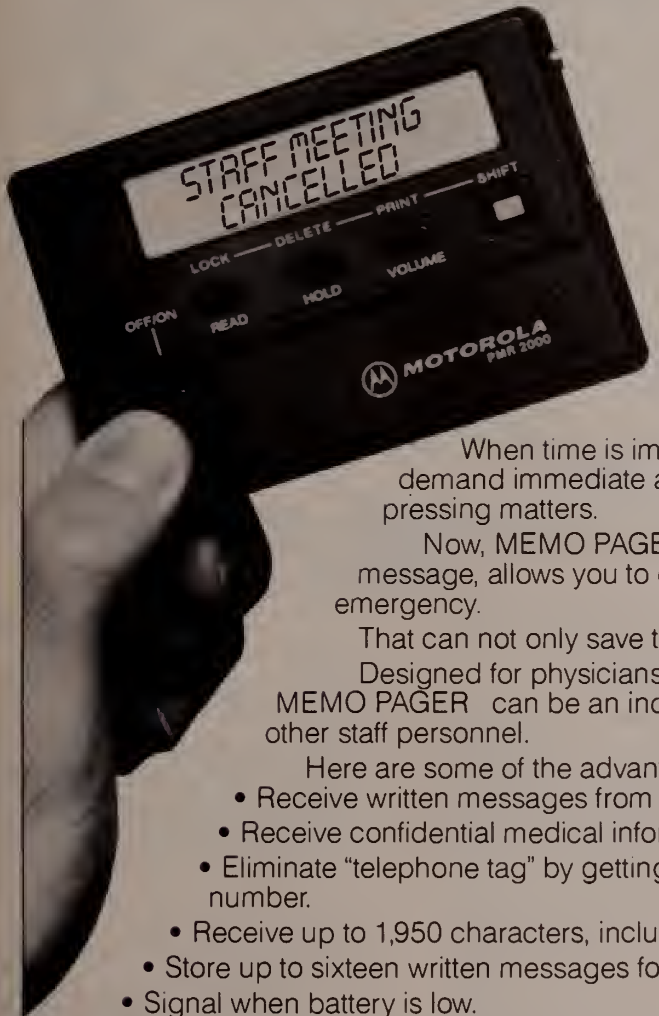

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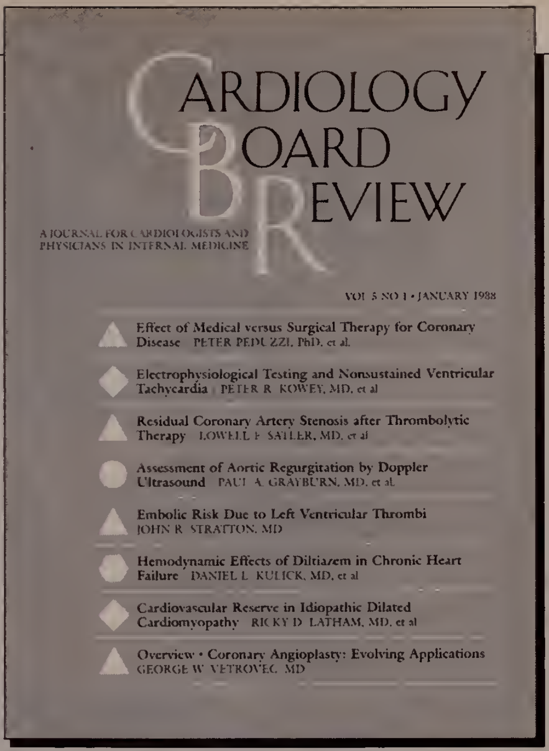

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Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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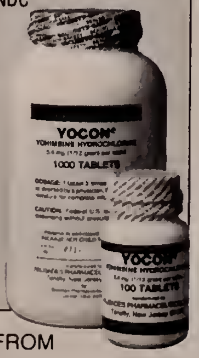

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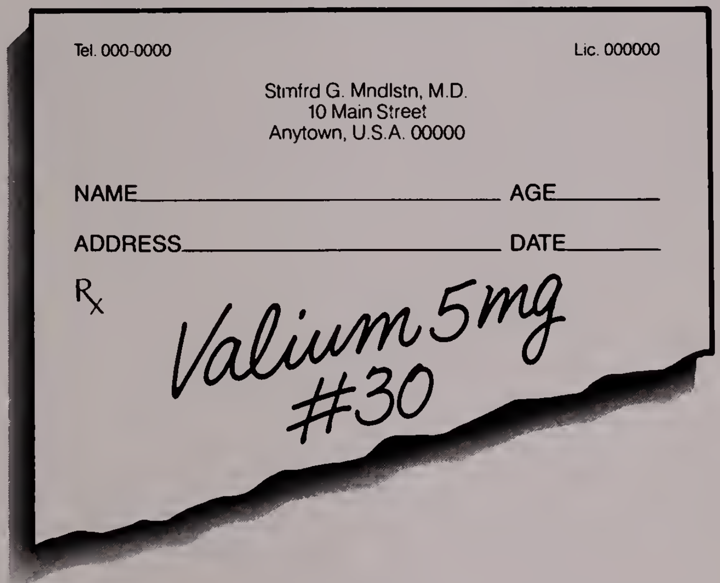

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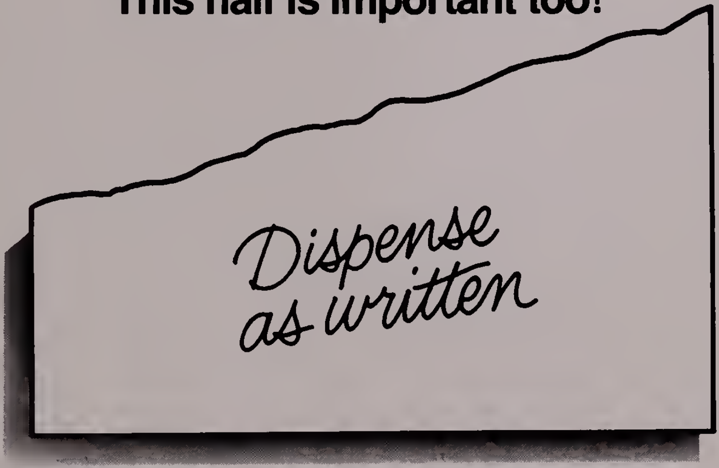

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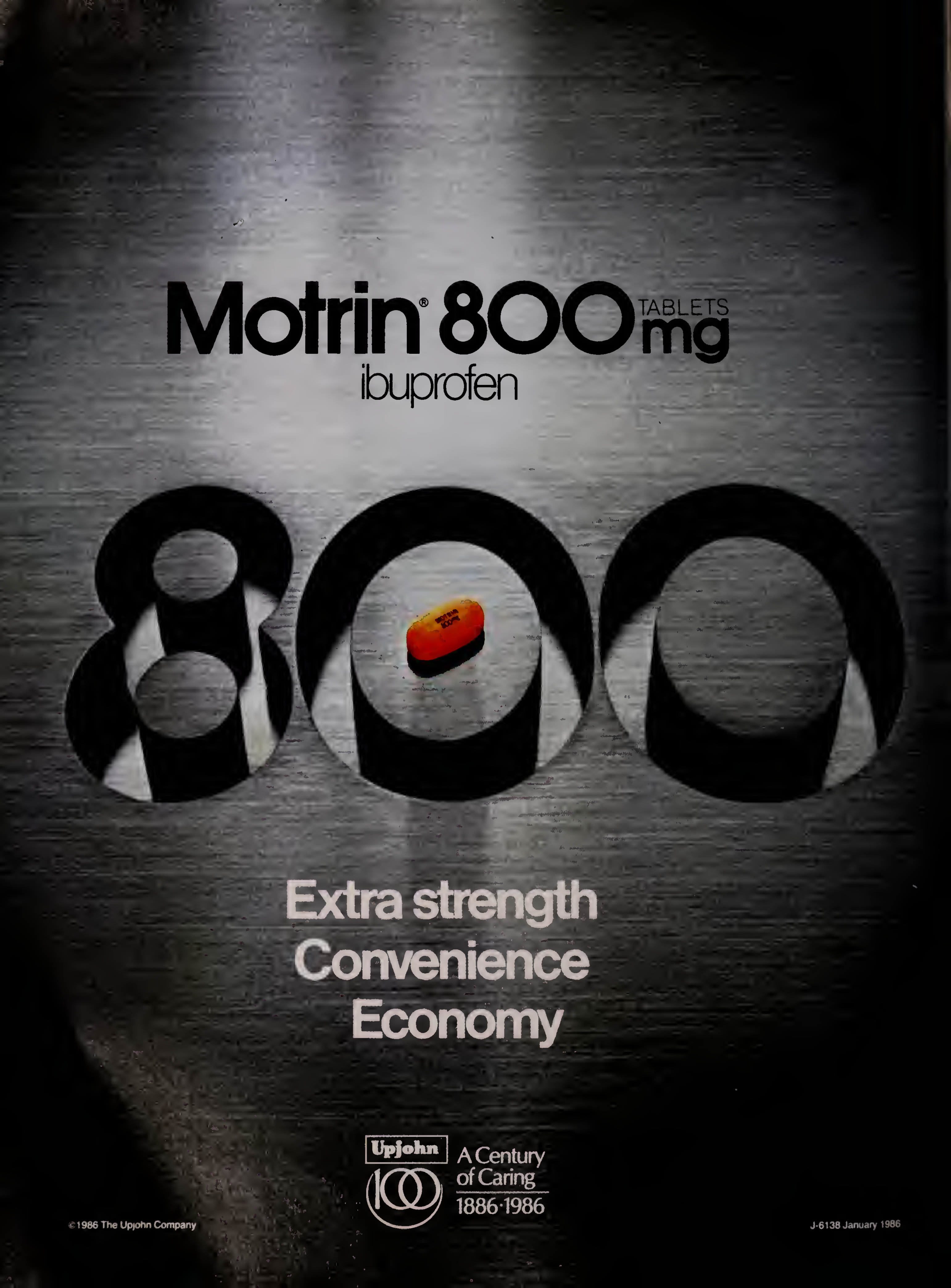

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ALLAN J. HAMILTON, M.D.

Neurosurgical Resident and Research Fellow,
Massachusetts General Hospital, Boston, Massachusetts.
Captain, U.S. Army Reserve.

EDUCATION Ithaca College, B.A. (Magna Cum Laude); Hamilton College (Pre-med); Harvard Medical School.

RESIDENCY General Surgical Internship. Neurosurgical Residency, Massachusetts General Hospital.

CONTINUING EDUCATION Neurology and Neurosurgery Research Fellowship Training, National Institutes of Health.

OUTSTANDING ACHIEVEMENTS Olsen Memorial Fellowship, National Masonic Medical Research Foundation; Albert Schweitzer Fellowship, International Albert Schweitzer Foundation; Harvard Medical School Cabot Prize for Best Senior Thesis; recently published article, "Who Shall Live and Who Shall Die" in Newsweek Magazine.

"The work I'm doing in the Army Reserve fits perfectly with my academic research interests in civilian life. The Army is very concerned with the effects of high-altitude cerebral edema, which is a mirror model of cerebral hypoxia, something I deal with every day in our neurosurgical intensive care unit. I couldn't ask for a smoother transition. And that's true for a lot of Reserve physicians. All we really do is change our clothes, not our mindset.

"Some of the projects the Army is undertaking are on the cutting edge of research. For example, I'm currently involved in developing for the Army a prototype of a non-invasive intracranial pressure-monitoring device that we hope will allow us to measure pressure changes as the brain swells—without drilling holes in the skull. If we can get our design to work, such a device could revolutionize high-altitude medicine as well as civilian neurosurgical care.

"The quality of medicine and the caliber of people I've been associated with in the Army Reserve are, without question, equal to civilian hospitals. In fact, I'm giving serious consideration to applying for an active duty academic position in Army Medicine when my residency ends at Massachusetts General."

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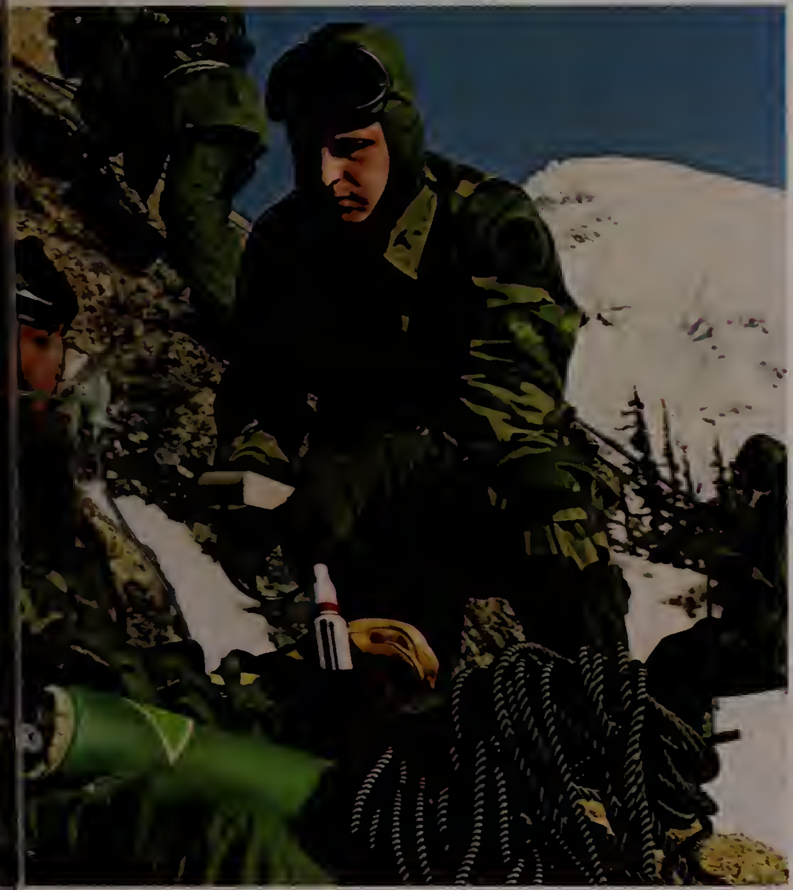

Soldier being examined for effects of high-altitude cerebral edema.

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*Please see last page of this advertisement for
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Coping with genital herpes is rarely easy. For some, the worst part is the pain and discomfort of frequent attacks — month after month, year after year. For others, the emotional burden presents a more difficult problem, leading to social isolation, anxiety, and diminished self-esteem.

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Although your patients have to live with herpes, they shouldn't have to suffer. Daily therapy with ZOVIRAX CAPSULES can help free them from the cycle of recurrent genital herpes. For many, one capsule three times a day can suppress recurrences completely while on therapy.

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Daily therapy with ZOVIRAX CAPSULES is generally well tolerated. The most frequent adverse reactions reported during clinical trials were headache, diarrhea, nausea/vomiting, vertigo, and arthralgia.

The physical and emotional difficulties posed by genital herpes are unique for each patient. The frequency and severity of recurrent episodes, as well as the emotional impact of the disease, should be considered when selecting daily therapy with ZOVIRAX CAPSULES.

Please see brief summary of prescribing information on next page.

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Brief Summary

INDICATIONS AND USAGE: Zovirax Capsules are indicated for the treatment of initial episodes and the management of recurrent episodes of genital herpes in certain patients.

The severity of disease is variable depending upon the immune status of the patient, the frequency and duration of episodes, and the degree of cutaneous or systemic involvement. These factors should determine patient management, which may include symptomatic support and counseling only, or the institution of specific therapy. The physical, emotional and psycho-social difficulties posed by herpes infections as well as the degree of debilitation, particularly in immunocompromised patients, are unique for each patient, and the physician should determine therapeutic alternatives based on his or her understanding of the individual patient's needs. Thus Zovirax Capsules are not appropriate in treating all genital herpes infections. The following guidelines may be useful in weighing the benefit/risk considerations in specific disease categories:

First Episodes (primary and nonprimary infections — commonly known as initial genital herpes):

Double-blind, placebo-controlled studies have demonstrated that orally administered Zovirax significantly reduced the duration of acute infection (detection of virus in lesions by tissue culture) and lesion healing. The duration of pain and new lesion formation was decreased in some patient groups. The promptness of initiation of therapy and/or the patient's prior exposure to Herpes simplex virus may influence the degree of benefit from therapy. Patients with mild disease may derive less benefit than those with more severe episodes. In patients with extremely severe episodes, in which prostration, central nervous system involvement, urinary retention or inability to take oral medication require hospitalization and more aggressive management, therapy may be best initiated with intravenous Zovirax.

Recurrent Episodes:

Double-blind, placebo-controlled studies in patients with frequent recurrences (6 or more episodes per year) have shown that Zovirax Capsules given for 4 to 6 months prevented or reduced the frequency and/or severity of recurrences in greater than 95% of patients. Clinical recurrences were prevented in 40 to 75% of patients. Some patients experienced increased severity of the first episode following cessation of therapy; the severity of subsequent episodes and the effect on the natural history of the disease are still under study.

The safety and efficacy of orally administered acyclovir in the suppression of frequent episodes of genital herpes have been established only for up to 6 months. Chronic suppressive therapy is most appropriate when, in the judgement of the physician, the benefits of such a regimen outweigh known or potential adverse effects. In general, Zovirax Capsules should not be used for the suppression of recurrent disease in mildly affected patients. Unanswered questions concerning the human relevance of *in vitro* mutagenicity studies and reproductive toxicity studies in animals given very high doses of acyclovir for short periods (see Carcinogenesis, Mutagenesis, Impairment of Fertility) should be borne in mind when designing long-term management for individual patients. Discussion of these issues with patients will provide them the opportunity to weigh the potential for toxicity against the severity of their disease. Thus, this regimen should be considered only for appropriate patients and only for six months until the results of ongoing studies allow a more precise evaluation of the benefit/risk assessment of prolonged therapy.

Limited studies have shown that there are certain patients for whom intermittent short-term treatment of recurrent episodes is effective. This approach may be more appropriate than a suppressive regimen in patients with infrequent recurrences.

Immunocompromised patients with recurrent herpes infections can be treated with either intermittent or chronic suppressive therapy. Clinically significant resistance, although rare, is more likely to be seen with prolonged or repeated therapy in severely immunocompromised patients with active lesions.

CONTRAINDICATIONS: Zovirax Capsules are contraindicated for patients who develop hypersensitivity or intolerance to the components of the formulation.

WARNINGS: Zovirax Capsules are intended for oral ingestion only.

PRECAUTIONS: General: Zovirax has caused decreased spermatogenesis at high doses in some animals and mutagenesis in some acute studies at high concentrations of drug (see PRECAUTIONS—Carcinogenesis, Mutagenesis, Impairment of Fertility). The recommended dosage and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION).

Exposure of Herpes simplex isolates to acyclovir *in vitro* can lead to the emergence of less sensitive viruses. The possibility of the appearance of less sensitive viruses in man must be borne in mind when treating patients. The relationship between the *in vitro* sensitivity of Herpes simplex virus to acyclovir and clinical response to therapy has yet to be established.

Because of the possibility that less sensitive virus may be selected in patients who are receiving acyclovir, all patients should be advised to take particular care to avoid potential transmission of virus if active lesions are present while they are on therapy. In severely immunocompromised patients, the physician should be aware that prolonged or repeated courses of acyclovir may result in selection of resistant viruses which may not fully respond to continued acyclovir therapy.

Drug Interactions: Co-administration of probenecid with intravenous acyclovir has been shown to increase the mean half-life and the area under the concentration-time curve. Urinary excretion and renal clearance were correspondingly reduced.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg given by gavage. There was no statistically significant difference in the incidence of tumors between treated and control animals, nor did acyclovir shorten the latency of tumors. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive life-time bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system considered less sensitive.

In acute studies, there was an increase, not statistically significant, in the incidence of chromosomal damage at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats but not Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found after 5 days dosing in a dominant lethal study in mice. In 6 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. At 3 loci in a Chinese hamster ovary cell line, the results were inconclusive. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive responses for mutagenicity and chromosomal damage occurred, but only at concentrations at least 400 times the acyclovir plasma levels achieved in man.

Acyclovir has not been shown to impair fertility or reproduction in mice (450 mg/kg/day, p.o.) or in rats (25 mg/kg/day, s.c.). At 50 mg/kg/day s.c. in the rat, there was a statistically significant increase in post-implantation loss, but no concomitant decrease in litter size. In female rabbits treated subcutaneously with acyclovir subsequent to mating, there was a statistically significant decrease in implantation efficiency but no concomitant decrease in litter size at a dose of 50 mg/kg/day. No effect upon implantation efficiency was observed when the same dose was administered intravenously. In a rat peri- and postnatal study at 50 mg/kg/day s.c., there was a statistically significant decrease in the group mean numbers of corpora lutea, total implantation sites and live fetuses in the F_1 generation. Although not statistically significant, there was also a dose related decrease in group mean numbers of live fetuses and implantation sites at 12.5 mg/kg/day and 25 mg/kg/day, s.c. The intravenous administration of 100 mg/kg/day, a dose known to cause obstructive nephropathy in rabbits, caused a significant increase in fetal resorptions and a corresponding decrease in litter size. However, at a maximum tolerated intravenous dose of 50 mg/kg/day in rabbits, there were no drug-related reproductive effects.

Intraperitoneal doses of 320 or 80 mg/kg/day acyclovir given to rats for 1 and 6 months, respectively, caused testicular atrophy. Testicular atrophy was persistent through the 4-week postdose recovery phase after 320 mg/kg/day; some evidence of recovery of sperm production was evident 30 days postdose. Intravenous doses of 100 and 200 mg/kg/day acyclovir given to dogs for 31 days caused aspermatogenesis. Testicles were normal in dogs given 50 mg/kg/day, i.v. for one month.

Pregnancy: ***Teratogenic Effects:*** Pregnancy Category C. Acyclovir was not teratogenic in the mouse (450 mg/kg/day, p.o.), rat (50 mg/kg/day, s.c.) or rabbit (50 mg/kg/day, s.c. and i.v.). There are no adequate and well-controlled studies in pregnant women. Acyclovir should not be used during pregnancy unless the potential benefit justifies the potential risk to the fetus. Although acyclovir was not teratogenic in animal studies, the drug's potential for causing chromosome breaks at high concentration should be taken into consideration in making this determination.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zovirax is administered to a nursing woman. In nursing mothers, consideration should be given to not using acyclovir treatment or discontinuing breastfeeding.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS—Short-Term Administration: The most frequent adverse reactions reported during clinical trials were nausea and/or vomiting in 8 of 298 patient treatments (2.7%) and headache in 2 of 298 (0.6%). Less frequent adverse reactions, each of which occurred in 1 of 298 patient treatments (0.3%), included diarrhea, dizziness, anorexia, fatigue, edema, skin rash, leg pain, inguinal adenopathy, medication taste and sore throat.

Long-Term Administration: The most frequent adverse reactions reported in studies of daily therapy for 3 to 6 months were headache in 33 of 251 patients (13.1%), diarrhea in 22 of 251 (8.8%), nausea and/or vomiting in 20 of 251 (8.0%), vertigo in 9 of 251 (3.6%), and arthralgia in 9 of 251 (3.6%). Less frequent adverse reactions, each of which occurred in less than 3% of the 251 patients (see number of patients in parentheses), included skin rash (7), insomnia (4), fatigue (7), fever (4), palpitations (1), sore throat (2), superficial thrombophlebitis (1), muscle cramps (2), pars planitis (1), menstrual abnormality (4), acne (3), lymphadenopathy (2), irritability (1), accelerated hair loss (1), and depression (1).

DOSAGE AND ADMINISTRATION: Treatment of initial genital herpes: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 10 days (total 50 capsules).

Chronic suppressive therapy for recurrent disease: One 200 mg capsule 3 times daily for up to 6 months. Some patients may require more drug, up to one 200 mg capsule 5 times daily for up to 6 months.

Intermittent Therapy: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 5 days (total 25 capsules). Therapy should be initiated at the earliest sign or symptom (prodrome) of recurrence.

Patients With Acute or Chronic Renal Impairment: One 200 mg capsule every 12 hours is recommended for patients with creatinine clearance ≤10 ml/min/1.73/m².

HOW SUPPLIED: Zovirax Capsules (blue, opaque) containing 200 mg acyclovir and printed with "Wellcome ZOVIRAX 200" - Bottles of 100 (NDC-0081-0991-55) and unit dose pack of 100 (NDC-0081-0991-56).

Store at 15°-30°C (59°-86°F) and protect from light.

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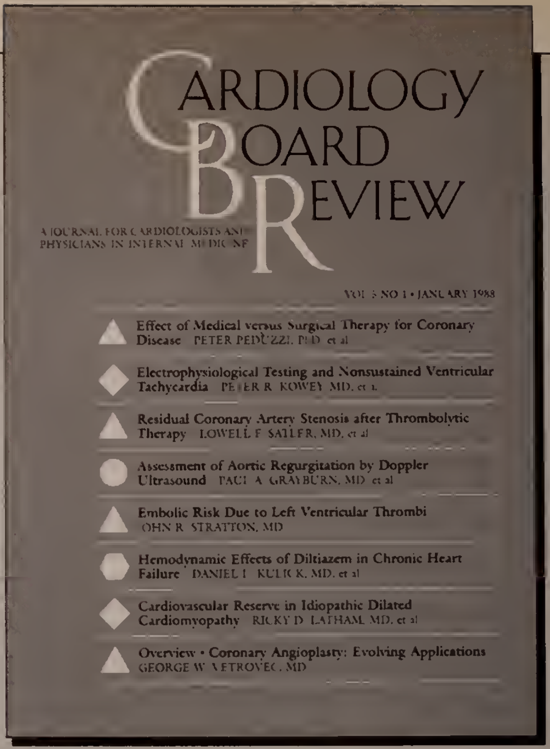

CARDIOLOGY BOARD REVIEW

A JOURNAL FOR CARDIOLOGISTS AND PHYSICIANS IN INTERNAL MEDICINE

VOL 5 NO 1 • JANUARY 1988

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◆ Electrophysiological Testing and Nonsustained Ventricular Tachycardia PETER R KOWEY MD, et al

▲ Residual Coronary Artery Stenosis after Thrombolytic Therapy LOWELL F SATLER, MD, et al

● Assessment of Aortic Regurgitation by Doppler Ultrasound PAUL A GRAYBURN, MD et al

▲ Embolic Risk Due to Left Ventricular Thrombi JOHN R STRATTON, MD

⬢ Hemodynamic Effects of Diltiazem in Chronic Heart Failure DANIEL I KULICK, MD, et al

◆ Cardiovascular Reserve in Idiopathic Dilated Cardiomyopathy RICKY D LATHAM, MD, et al

▲ Overview • Coronary Angioplasty: Evolving Applications GEORGE W VETROVEC, MD

*Journals reviewed include: *Circulation*, *American Heart Journal*, *Journal of the American College of Cardiology*, *British Heart Journal*, *Chest*, *The American Journal of Cardiology*, *The New England Journal of Medicine*, *Annals of Internal Medicine*, *American Journal of Medicine*, and *The Journal of the American Medical Association*.

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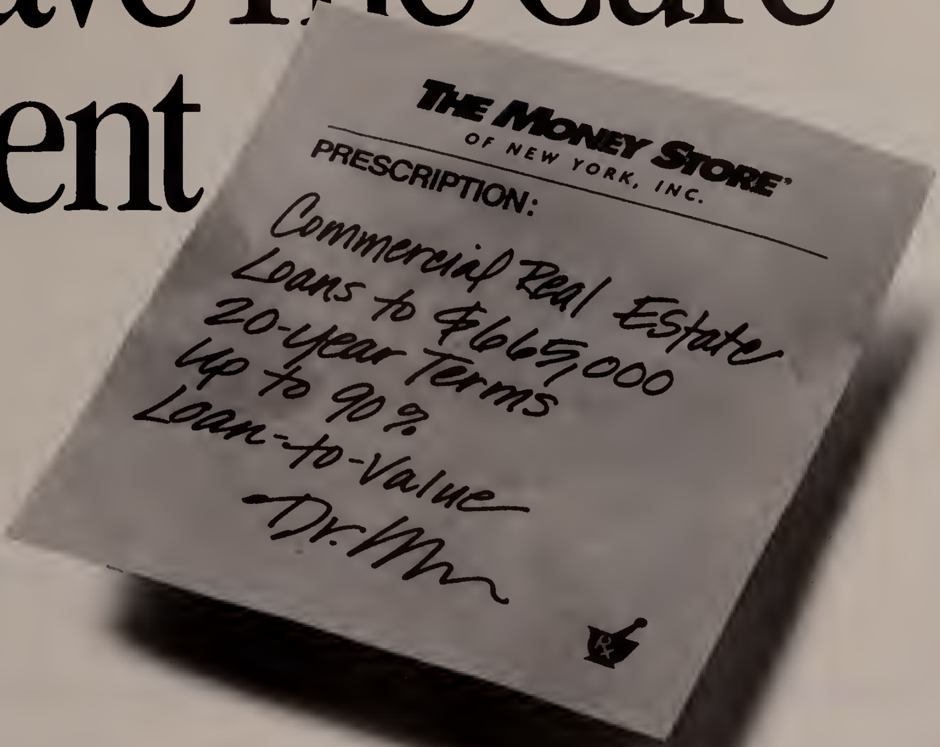

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OTESAGA

August 5-7. **Summer Pediatric Refresher Course.** 11 Cat 1 Credits. Otesaga. Contact: Charlotte Hoag, Medical Education, The Mary Imogene Bassett Hospital, One Atwell Road, Cooperstown, NY 13326. Tel: (607) 547-3926.

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CALIFORNIA

August 1–5. **Modern Radiology Practice.** 30 Cat 1 Credits. Fess Parker Red Lion Resort in Santa Barbara. *August 25–28.* Fourth Annual Physics and Biology of Radiology. 25 Cat 1 Credits. La Jolla Village Inn, La Jolla. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

HAWAII

August 13–20. **Eighth Annual Symposium on Fine Needle Aspiration.** 25 Cat 1 Credits. Westin Hotel, Kaanapali Beach, Maui. Contact: Extended Programs in Medical Education, Registration Office, Room 575-U, University of California, San Francisco, CA 94143-0766. Tel: (415) 476-4251.

MASSACHUSETTS

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August 4–5. **Advanced Trauma Life Support.** 17 Cat 1 Credits. University of Nebraska Medical Center. Contact: Cindy S. Hanssen, University of Nebraska Medical Center, Center for Continuing Education, 42nd and Dewey Avenue, NE 68105-1065. Tel: (402) 559-4152.

NEW JERSEY

August 1–8. **Anesthesiology—Liability Law and Ethics.** 6 Cat 1 Credits. Atlantic City. *August 8–9.* **Adolescent Medicine.** 30 Cat 1 Credits. Atlantic City. *August 29–30.* **Pressing Issues—Neurology Spinal Disorders/Lyme Disease.** Atlantic City. 30 Cat 1 Credits. Contact: Albert Einstein College of Medicine/Montefiore Medical Center, Of-

(*continued on p 16A*)

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Brief Summary. Consult the package insert for prescribing information.

Indications and Usage: Axid is indicated for up to eight weeks for the treatment of active duodenal ulcer. In most patients, the ulcer will heal within four weeks.

Axid is indicated for maintenance therapy for duodenal ulcer patients, at a reduced dosage of 150 mg h.s. after healing of an active duodenal ulcer. The consequences of continuous therapy with Axid for longer than one year are not known.

Contraindication: Axid is contraindicated in patients with known hypersensitivity to the drug and should be used with caution in patients with hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Because nizatidine is excreted primarily by the kidney, dosage should be reduced in patients with moderate to severe renal insufficiency.

3. Pharmacokinetic studies in patients with hepatorenal syndrome have not been done. Part of the dose of nizatidine is metabolized in the liver. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests—False-positive tests for urobilinogen with Multistix® may occur during therapy with nizatidine.

Drug Interactions—No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high dose males compared to placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement compared to concurrent controls, and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive, and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery is not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, and the mouse lymphoma assay.

In a two-generation, perinatal and postnatal, fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose, and in Dutch Belted rabbits at doses up to 55 times the human dose, revealed no evidence of impaired fertility or teratogenic effect, but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Nizatidine is secreted and concentrated in the milk of lactating rats. Pups reared by treated lactating rats had depressed growth rates. Although no studies have been conducted in lactating women, nizatidine is assumed to be secreted in human milk, and caution should be exercised when nizatidine is administered to nursing mothers.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Ulcer healing rates in elderly patients are similar to those in younger age groups. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations. Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo. Among the more common adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common in the nizatidine group. A variety of less common events was also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic—Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients possibly or probably related to nizatidine. In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L), and in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid. Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo. Rare reports of gynecomastia occurred.

Hematologic—Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist. On previous occasions, this patient had experienced thrombocytopenia while taking other drugs.

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine than in placebo patients. Rash and exfoliative dermatitis were also reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported.

Overdosage: There is little clinical experience with overdosage of Axid in humans. If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea. Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal. Intravenous LD_{50} values in the rat and mouse were 301 mg/kg and 232 mg/kg respectively.

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NEW YORK STATE
JOURNAL OF MEDICINE

July 1988 • Volume 88, Number 7

COMMENTARIES

The aging of caretakers of those with chronic mental illness

What becomes of those adults with chronic mental illness as their parent(s) or other family member caretakers begin to reach an age at which they can no longer maintain the patients at home? The answers will have import for the quality of life of the individuals involved, and should command the attention of those formulating public policy for the mentally ill.

An important consequence of deinstitutionalization was the nationwide release of hundreds of thousands of vulnerable patients with chronic mental illness from state hospitals into communities. The change in public policy that resulted in the release of these patients from state hospitals also had a profound effect on a younger generation of mental patients who came to be known as the "young adult chronically mentally ill" by observers of the demographics of the mentally ill such as Pepper, Bachrach, Prevost, and Schwartz.[1-4] These younger mentally ill would have spent much of their lives in state hospitals had it not been for the alteration in philosophy with respect to admissions and continuing treatment.

The case management approach to assisting deinstitutionalized patients emerged in 1977.[5,6] Its goal was to facilitate the survival and quality of life of patients outside institutions by linking them to mental health and general medical services, as well as housing, entitlements, and other services in their communities. New York initiated its Community Support System (CSS) program in 1978. CSS was to be a "network of caring and responsible people committed to assisting a vulnerable population to meet their needs and develop their potentials without being unnecessarily isolated or excluded from the community."[5] Perlman, Melnick, and Kentera have documented the concrete benefits received by clients enrolled in a CSS program.[7] Entrance criteria were highly exclusionary. An experimental CSS-expansion (CSS-X) program was subsequently initiated to reach out to the young adult patients with chronic mental illness who had not experienced prolonged periods of hospitalization. The two components of the CSS program were merged in New York as of January 1, 1988.

Many of the deinstitutionalized patients returned to live with their families, while many of the young adult chronic patients remained at home. Goldman[8] has provided insight into the vast numbers of such patients residing at home. He estimates that 0.8–1.5 million chronically mentally ill reside in the community, with 110,000 being discharged from hospitals each year. He states that in 1975, 73% of patients admitted to psychiatric units of general hospitals had been living with their families, and that 65%, 25% of whom were chronically ill, returned home following discharge. Most of these families were willing to have their relatives at home, despite threatening and embarrassing behavior, social stigma, financial strain, and marital and family disruption. Goldman's conclusions are supported by the findings of a report issued by the New York State Council on Children and Families.[9] The council found that the return of chronically ill mental patients to their families results in a perceived burden and substantial lifestyle change for approximately half the families. Members of such families believed that the availability of social and psychological services to help them deal with the stress resulting from their relatives' return would be helpful, especially if coupled with other concrete services.

The persistence of caretaking families in maintaining chronically mentally ill relatives at home is both humanitarian and psychosocially stabilizing. It averts the cost of providing residence for a large number of individuals who otherwise would need shelter. Even when patients receive housing subsidies, which they contribute to a family living arrangement, the taxpayers save, as no capital funds need be raised to subsidize the construction of community residences. Planners must be concerned with the implications for public policy as persons who maintain patients with chronic mental illness at home reach an age at which they are no longer able to cope.

A review of the roster of one CSS and CSS-X program providing care in an inner-city setting revealed the following data. Forty-four (29%) of 151 CSS clients resided with close relatives who provided some degree of caretaking. Nineteen (43%) of these clients lived with parents. Ten of the clients, with a mean age of 27.3 years and a range of 23 to 34 years, had caretaking relatives aged in their 50s. Seven clients, with a mean age of 36.8 years, ranging from 27 to 49 years, were residing with parents in their 60s. Two clients aged 50 and 53 lived with a parent whose age was in the 70s. One client, age 40, lived with a grandmother who was in her 80s. Eight CSS clients resided with siblings, while 13 lived with spouses. In these cases, the ages of clients and caretakers were approximately the same. The CSS-X program had 23 clients on its rolls. Eight (35%) lived with parents. Three (13%) were dependent on adult children and came to the program's attention when their children left home for lives of their own.

The following case report is a representative sample. JF's parents are in their late 60s and have managed to maintain their 36-year-old son, who suffers from chronic schizophrenia, at home. The CSS program has assisted this family in caring for their son since 1979, at which time the parents had begun to find it increasingly difficult to deal with his bizarre behavior, which often occurred late at night and which was marked by threats and verbal abusiveness. He spoke aloud to himself, laughed inappropriately, and played music loudly at night, thus drawing the complaints of neighbors. His behavior had become more frenetic, and efforts to stabilize him with medication had not succeeded, despite several hospital stays. Attempts to provide structure for the client and respite for the family by enrolling JF in a day treatment program or sheltered workshop failed. The parents now are requesting that a long-term residence be found for the son they so long struggled to keep with them at home.

This illustrative case report and the data gleaned from the records of case management programs suggest that there will be an "aging out" of caretakers, beginning within the next five years and proceeding incrementally into the future. During the next 15 years, the majority of clients residing with families will lose the support of caretaking relatives and the home now available to them. These losses will come from the inability of caretakers to cope with difficult-to-manage patients, as supportive family members grow older and their energy diminishes. It is anticipated that relationships in which parents care for children will have the greatest economic impact, and those in which a spouse cares for a mate the least. The reason for this differentiation lies in the obvious fact that spousal life expectancy is longer than that of children.

The extent to which projections can be made from the limited existing experience will depend on the degree to which these findings can be generalized. State agencies must gather and analyze similar data if they are to prepare for future pressures on their mental health and social service systems. Governmental planners, interested as they seem to be in continuing the maintenance of patients with chronic mental illness in the community, must anticipate future housing needs for those that now reside with families. It will also be necessary to allocate funds for the hiring of custodians and the provision of supervised living situations to replace family members who now offer primary, day-to-day, caretaking. Failure to prepare for this would inflict undue hardships on the vulnerable, and might swell the ranks of the homeless.

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Resurgence of acute rheumatic fever

In this issue of the *Journal*, Giardina and Heaton[1] describe six children with acute rheumatic fever. While this is not a large number of patients, it may represent a possible local resurgence of the disease. One should view the recent outbreaks of rheumatic fever being reported in the United States with some concern.[2-5] While the disease remains a major health problem in third world countries,[6] it has been a disappearing disease during the past two decades in the United States.[7]

The demographic data from recently reported outbreaks differ from those in the past in that the majority of recent patients come from middle income families in suburban or rural areas in contrast to patients seen in the past who primarily came from low-income, crowded, inner-city areas. Medical care has also been readily available to patients seen during the recent outbreaks. Why, then, are we seeing a return of rheumatic fever? The answer to this question is not yet known, but various hypotheses have been suggested.

Since rheumatic fever is caused by a nonsuppurative sequel to group A streptococcal infection, there may have been an increase in the incidence of streptococcal infections. While it is difficult to determine the prevalence of streptococcal infections in the general population, Gordes[7] has stated that unpublished observations from Hall and Breese in Rochester, NY, show that the number of positive throat cultures for group A streptococci did not decline during the period of time rheumatic fever was declining in the country.

Could there be an increase in the virulence of the group A streptococcus? The mucoid strains of this organism have classically been considered extremely virulent, and large numbers of serotypes M-3 and M-18 streptococci have been isolated in areas where recent outbreaks have occurred, albeit in only a small number of patients with rheumatic fever. To date, however, the group A streptococcus remains extremely sensitive to penicillin.

Rheumatic fever occurs in approximately 3% of individuals with an untreated group A streptococcal infection. The possibility that there has been a change in host susceptibility might account for the recent upsurge in rheumatic fever. Recent studies show that B-cell alloantigens are present in a majority of patients with rheumatic fever.[8] A major genetic change in the population in this short period of time would not seem possible.

The appearance or reappearance of a cofactor that could enhance the rheumatogenic properties of streptococcus has been suggested. The fact that a viral infection can enhance a bacterial infection has been demonstrated.[9] No cofactor has ever been found in patients with rheumatic fever.

Finally, due to the decline in rheumatic fever in recent years, has there been complacency on the part of medical services in searching for and treating streptococcal infections? The fact that significant numbers of recent patients have not had symptoms of pharyngitis prior to the onset of acute rheumatic fever makes this aspect of prevention more difficult. In view of this resurgence of rheumatic fever, a throat culture should be taken on any patient with a sore throat or upper respiratory tract infection, regardless of other symptoms. If one of the well established, rapid tests for group A streptococcus antigen is used and is negative, a second conventional culture should be taken. While the direct test is highly specific, the sensitivity is not reliable.

There can be no compromise in the treatment of group A streptococcal pharyngitis. The drug of choice is penicillin given orally for ten days or, if compliance is in doubt, injectable, long-acting benzathine penicillin should be administered. In case of penicillin allergy, oral erythromycin may be given for ten days. Prevention of recurrent attacks with a recommended antibiotic must be instituted following the acute phase of rheumatic fever. The use of these measures will help in preventing rheumatic fever from again becoming a major health problem in the United States.

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Measuring cancer patients' quality of life

A look at physician attitudes

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ABSTRACT. In recent years, the focus of the majority of research in the area of measuring quality of life has involved the development of new measurement instruments. This study, however, examined physicians' attitudes about measuring their cancer patients' quality of life, and determined the current methods of doing so. Six hundred and seventy-five physicians were surveyed, and 257 evaluable questionnaires were returned. The majority of physicians indicated that they think it is possible to measure quality of life; however, few have a specific method for doing so. Only 107 had ever used one of the patient evaluation instruments listed in the questionnaire. Although these physicians expressed positive opinions about measuring quality of life, few were aware of the instruments currently available to help them evaluate their cancer patients. These findings indicate an underutilization of existing measures and the future need for a well-developed and publicized instrument for measuring cancer patients' quality of life.

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The importance of measuring the quality of life of persons with cancer has been recognized by cancer researchers and physicians in recent years. This has led to numerous requests for the development of new, well-developed quality of life indices. The European Organization for Research on Treatment of Cancer set up a Study Group on Quality of Life[1,2] and a 1979 Department of Health, Education and Welfare (DHEW) publication called for the inclusion of health status or quality of life indices in all National Institutes of Health (NIH) sponsored clinical trials.[3] As a result of this impetus, some researchers are now using quality of life measures in their cancer clinical trials.[4-6] The need for refined measures of quality of life is especially critical for the many advanced cancers for which treatment is primarily palliative, and for those cancers for which an uncontroversial "objective" measure of response is not available.

Several studies have been carried out in recent years to develop a valid, reliable quality of life measurement instrument for persons with cancer. Padilla et al[7] studied a 14-item self-assessment questionnaire which evaluated the physical condition, daily activities, and attitudes about quality of life of oncology patients receiving chemotherapy or radiation treatments. Selby et al[8] evaluated a 31-item self-assessment questionnaire using patients attending a clinic for the management of breast cancer. Spitzer et al[9] developed a five-item Quality of Life Index to be used by physicians to rate their patients' quality of life. Finally, Schipper et al,[10] in the most impressive study made to date involving measurement of cancer patients' quality of life, developed a 22-item self-assessment questionnaire called the Functional Living Index—Cancer (FLIC). However, none of these instruments is completely satisfactory, and additional research is still needed in this important area.

If any of the existing or to-be-developed quality of life questionnaires are to be used on a routine basis and become an integral part of cancer treatment decisions, they must be accepted by the medical community. To date, however, little attention has been focused on determining physicians' knowledge of and attitudes about this important issue. The purpose of the present study, therefore, was to gain a better understanding of physicians' opinions about measuring cancer patients' quality of life, as well as to determine their current methods of doing so.

A questionnaire was developed which asked physicians whether they thought that it is possible to measure quality of life, whether quality of life influences cancer patients' length of survival, whether treatment alternatives should be discussed with cancer patients when deciding their treatment, and whether they would ever use a quality of life measurement instrument if a good one were devised. The survey included a list of 16 well-known patient evaluation instruments. The physicians were asked to indicate which ones they had heard about, and which they had ever used. They were also asked to indicate their medical specialties, how many adult cancer patients they had treated during the previous year, and how many they had referred elsewhere for treatment. Questions regarding their type of practice and the year they were graduated from medical school were also asked. The survey concluded with an open-ended section in which the physicians were asked to describe the methods they currently use to assess their

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cancer patients' quality of life, and to make any additional comments on this topic.

METHODS

A registry of all participants in the Continuing Medical Education (CME) programs coordinated through Roswell Park Memorial Institute (RPMI) was used to identify physicians for the survey. Selected from this registry were the names and addresses of 675 physicians who had attended CME programs between January 1, 1982, and October 31, 1986. This sample included physicians residing in Pennsylvania, Ohio, and New York. The majority of the sample was from New York State. (Physicians from RPMI were not included in the sample, since the primary focus of the study was to determine how physicians in the community feel about and are dealing with measuring cancer patients' quality of life. In general, RPMI physicians are involved with cancer research as well as patient treatment and are limited to practicing medicine at RPMI. Many of these physicians are following research protocols and have limited flexibility in deciding on treatments for their patients. Given the unique nature of their practices, these physicians were not considered appropriate for this study.)

Two mailings were conducted. Sixty-six questionnaires were returned because they were undeliverable, five physicians were deceased, two physicians did not wish to participate, and one person was eliminated from the sample because he was not a physician. Completed surveys were returned by 308 physicians, which resulted in an overall response rate, based on the 601 eligible respondents, of 51%. Of those who responded, 51 indicated that the survey was not relevant to their current medical practice. These physicians were not directly involved with treating patients; for example, 15 were pathologists, nine were radiologists, and 11 were retired.

Information on the nonrespondents was limited. However, they were compared to the respondents with respect to location of residence, medical specialties, and number of CME programs attended, to determine any marked differences.

As mentioned previously, the majority of physicians surveyed (80%) resided in New York State. The percentage of respondents and nonrespondents did not differ with respect to the state in which they resided (Table I). Approximately 80% of both the respondents and nonrespondents resided in New York State, 17% of the respondents and 19% of the nonrespondents resided in Pennsylvania, and only about 1% of both groups were from Ohio.

Geographic location within the states was also examined to determine any differences between respondents and nonrespondents. Few differences in response rates were observed among the ten geographic areas into which New York was divided. Pennsylvania was broken into two areas, the western region and the eastern region. Approximately the same percentage of respondents and nonrespondents resided in each of these areas. The number of physicians from Ohio (nine) was too small to merit categorizing into smaller geographic locations.

Medical specialties of respondents and nonrespondents were compared, and again few differences were observed. Of the 35 specialties represented in this sample, a 1% or greater difference in the percentage of respondents and nonrespondents could be found for only seven specialties, with the largest difference being 3%.

It was hypothesized that physicians who had attended a large number of CME programs would be more likely to return the survey than those who had attended only a few. The interest in cancer they demonstrated through their attendance at CME programs was expected to be reflected in their willingness to participate in a survey related to treating cancer patients. This was not found to be true. The majority (62%) of physicians surveyed had attended only one program coordinated through RPMI between January 1, 1982, and October 31, 1986. Approximately 3% had attended ten or more programs, including 2% of the respondents and 4% of the nonrespondents (Table II).

Overall, comparisons of respondents and nonrespondents using the information available on both groups revealed few differences.

TABLE I. Respondent Status and State of Residence

State	Nonrespondents	Respondents	Total
New York	237 (81%)	246 (80%)	481 (80%)
Pennsylvania	51 (17%)	58 (19%)	109 (18%)
Ohio	5 (2%)	4 (1%)	9 (2%)
Total	293 (100%)	308 (100%)	601 (100%)

RESULTS

Responses from 257 evaluable surveys are included in the following discussion. As noted previously, a number of respondents (51) indicated that the survey was not relevant to their current medical practice; therefore, this group is not included.

A breakdown by medical specialty indicated that the greatest number of respondents were surgeons (22%), followed by internists (17%), family practitioners (16%), oncologists (13%), urologists (10%), gynecologists (7%), and pediatricians (6%).

Approximately half of the respondents maintain private office practices (48%), while 20% participate in group practices. Twenty-three percent are full-time hospital staff members, and another 4% are on staff at hospitals on a part-time basis. Other types of practices mentioned include clinics (1%), nursing homes (2%), and emergency rooms (<1%).

The number of years that each physician had been practicing medicine was estimated from the year that he or she graduated from medical school, and ranged from two to 54 years, with a median of approximately 24 years. The median age of the respondents was estimated to be approximately 50 years.

The number of adult cancer patients seen by the physicians during the past year ranged from zero to more than 1,000, with a median of 25. The number of cancer patients referred elsewhere for treatment ranged from zero to 400; however, the median was less than five. Fifty-two physicians indicated that they are actively participating in cancer clinical trials.

The majority (78%) of these physicians thought that it is possible to measure quality of life, while 20% did not, and another 2% were uncertain. When asked if they thought that quality of life can influence length of survival, 87% responded yes, 8% said no, and another 4% were undecided. An overwhelming majority of the physicians (98%) believed that quality of life alternatives should be discussed with their patients when making treatment decisions, and 89% said that they would use a good measure of quality of life if one were devised.

One hundred fifty-five respondents (60%) indicated that they had heard of one or more of the 16 standard patient evaluation instruments listed in the questionnaire; and 107 (42%) had used at least one. The General Health Questionnaire[11] was the instrument most familiar (65%) to these 155 physicians, followed by the Karnofsky Patient Performance Rating,[12] the Beck Depression Questionnaire,[13] the Katz Activities of Daily Living Index,[14] and Spitzer's Quality of Life Index,[9] with respective percentages of 47%, 24%, 13%, and 13% (Table III).

The patient evaluation instruments most frequently cited as having been used corresponded almost exactly with the physicians' familiarity with them. Of the 107 physicians indicating that they used one or more of the instruments, the General Health Questionnaire

TABLE II. Responder Status and Number of CME Conferences Attended

No. of Conferences	Nonrespondents	Respondents	Total
1	176 (60%)	196 (64%)	372 (62%)
2–5	93 (32%)	94 (30%)	187 (31%)
6–9	11 (4%)	11 (4%)	22 (4%)
>9	13 (4%)	7 (2%)	20 (3%)
Total	293 (100%)	308 (100%)	601 (100%)

TABLE III. Familiarity With and Use of Patient Evaluation Instruments

Questionnaire	Heard About N (% of 155)	Have Used N (% of 107)
General Health Questionnaire	101 (65)	59 (55)
Karnofsky Patient Performance Rating	73 (47)	54 (50)
Beck Depression Questionnaire	38 (24)	17 (16)
Katz Activities of Daily Living	20 (13)	6 (6)
Spitzer's Quality of Life Index	20 (13)	6 (6)
Functional Living Index—Cancer	17 (11)	8 (7)

was mentioned most frequently (55%), followed by the Karnofsky Patient Performance Rating (50%) and the Beck Depression Questionnaire (16%). In addition, 7% of these respondents indicated that they had used the Functional Living Index—Cancer, and 6% had used the Katz Activities of Daily Living Index.

Respondents involved with treating cancer patients were somewhat more aware of the patient evaluation instruments available to them. As shown in Table IV, a greater percentage of physicians who had treated 100 or more cancer patients the previous year had heard about (84%) and used (66%) one of the instruments listed in the questionnaire, than any other group. The percentage of physicians who were aware of and had used one of the questionnaires increased as the number of cancer patients they saw increased. In addition to this, the oncologists indicated a greater awareness of (94%) and use of (85%) the instruments than did physicians with other specialties (Table V). Only 64% of the internists, 62% of the family practitioners, and 54% of the surgeons were familiar with any of the instruments, and only 48%, 50%, and 32%, respectively had used one. Of the 33 oncologists who responded to the survey, 31 had heard about and 28 had used the Karnofsky scale, while 14 were aware of the General Health Questionnaire and six had used it.

Respondents who had been practicing medicine only a short time appeared to be somewhat more aware of the standard patient evaluation instruments available to them than their older counterparts (Table VI). Sixty percent of the 77 physicians who graduated in the 1970s and 67% of the 18 graduating within the last five years were familiar with one of the instruments, while 40% and 50%, respectively, had used one.

Although a number of the physicians expressed familarity with the above-mentioned patient evaluation instruments, few indicated that they used them on a routine basis. Only 94 respondents (37%) stated that they currently have a method of assessing their patients' quality of life, and only six of these included a standard test. The Karnofsky Patient Performance Rating was used by five physicians and the General Health Questionnaire by one other. The most popular method mentioned by these physicians was discussions with patients (74%), followed by discussions with patients and family members (33%). Approximately 9% of the physicians stated that they rely on their clinical assessment, and another 5% rely on information provided by other health care personnel, such as visiting nurses.

TABLE IV. Familiarity With and Use of Patient Evaluation Questionnaires According to the Number of Cancer Patients Seen Yearly

No. of Patients	Heard About N (%) Yes	Heard About N (%) No	Have Used N (%) Yes	Have Used N (%) No	Total
0	6 (32%)	13 (68%)	5 (26%)	14 (74%)	19
1-9	17 (42%)	24 (59%)	11 (27%)	30 (73%)	41
10-19	19 (51%)	18 (49%)	10 (27%)	27 (73%)	37
20-39	27 (60%)	18 (40%)	15 (33%)	30 (67%)	45
40-100	37 (65%)	20 (35%)	28 (49%)	29 (51%)	57
>100	42 (84%)	8 (16%)	33 (66%)	17 (34%)	50
Total	148	101	102	147	249

TABLE V. Familiarity With and Use of Patient Evaluation Questionnaires According to Medical Specialty

Specialty	Heard About N (%) Yes	Heard About N (%) No	Have Used N (%) Yes	Have Used N (%) No	Total
Oncology	31 (94)	2 (6)	28 (85)	5 (15)	33
Internal medicine	28 (64)	16 (36)	21 (48)	23 (52)	44
Family practice	25 (62)	15 (38)	20 (50)	20 (50)	40
Urology	16 (61)	10 (39)	9 (35)	17 (65)	26
Other	30 (60)	20 (40)	19 (38)	31 (62)	50
Surgery	31 (54)	26 (46)	18 (32)	39 (68)	57
Obstetrics/ gynecology	8 (44)	10 (56)	4 (22)	14 (78)	18
Pediatrics	4 (29)	10 (71)	4 (29)	10 (71)	14
Total responses*	173	109	123	159	282
Total responders	155	102	107	150	257

* The total number of responses is greater than the number of responders because some physicians indicated more than one specialty.

Sixty percent of the physicians who made additional comments about assessing cancer patients' quality of life expressed a positive opinion about the subject. Forty-four stated that quality of life should be measured, and a number welcomed the development of a standardized method of doing so. Twelve physicians requested further information on the subject.

Twenty-one physicians thought that quality of life is a very subjective phenomenon, which is difficult to measure quantitatively. Although they too were concerned about their patients' quality of life, they recommended an individualized, personal approach for its assessment.

DISCUSSION

The results of this survey suggest a need for a well-developed and publicized instrument for measuring the quality of life of patients with cancer. A large majority (78%) of physicians surveyed felt that it is possible to measure quality of life, and an even greater majority (89%) stated they would consider using a quality of life measurement instrument. However, only a minority (37%) indicated that they currently have a specific method of assessing their cancer patients' quality of life.

It is apparent that the measurement instruments that have been developed thus far have not been adopted on a large scale by the medical community. Only 60% of the physicians surveyed had even heard of, and only 42% had used, at least one of the 16 standard patient evaluation instruments that were listed in the questionnaire. The underutilization of such instruments may be due in part to a

TABLE VI. Familiarity With Patient Evaluation Questionnaires According to Year Graduated from Medical School

Years	Heard About N (%) Yes	Heard About N (%) No	Have Used N (%) Yes	Have Used N (%) No	Total
1933-1939	4 (40)	6 (60)	3 (30)	7 (70)	10
1940-1949	21 (54)	18 (46)	14 (36)	25 (64)	39
1950-1959	25 (61)	16 (39)	16 (39)	25 (61)	41
1960-1969	41 (62)	25 (38)	29 (44)	37 (56)	66
1970-1979	46 (60)	31 (40)	31 (40)	46 (60)	77
1980-1985	12 (67)	6 (33)	9 (50)	9 (50)	18
Total	149	102	102	149	251

lack of awareness of the existence of formal methods of evaluating quality of life. Thus, future quality of life research, if it is to be fruitful, must be directed toward developing new and improved measurement instruments, while keeping physicians informed about advances being made in this important area. The existence of new instruments must be publicized, and their value demonstrated, if they are ever going to be adopted by physicians on a large scale. Additional attention must be focused on determining the best method for disseminating information to the medical community and persuading physicians to incorporate new ideas into their medical practices. New research in the area of medical technology transfer needs to be initiated to serve this need.

A scientifically sound quality of life measurement instrument designed for persons with cancer would provide physicians with another means of opening up communication with their patients. It would give patients a means of conveying information to physicians which could be pertinent to decisions regarding their treatment. Information obtained by such a method would assist physicians with treatment decisions, while helping patients feel more involved in this decision-making process. On a larger scale, if it were to be used routinely in clinical trials, a quality of life measurement instrument would provide additional information on the effectiveness of new treatment protocols.

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The usefulness of echocardiography in a long term health care facility

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ABSTRACT. Two-dimensional and M-mode echocardiography were used to evaluate a broad spectrum of signs and symptoms in 1,401 patients in a long term health care facility. Five percent of these often bed-bound or wheelchair-bound patients with multisystemic disease were found to have unsuspected disorders for which a change in medical management was indicated. In another 5%, the echocardiographic findings did not mandate a change in therapy during the study period, but currently they might suggest the need for treatment of diastolic left ventricular dysfunction. We conclude that this noninvasive, painless test is an important tool in the evaluation of chronically ill patients with a variety of complaints or findings referable to the cardiovascular system.

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Echocardiography has proved its utility in the diagnosis and management of a variety of cardiac diseases including pericardial effusion, valvular pathology, chamber dilation, and cardiomyopathy. This noninvasive technique has been shown to be an effective tool in confirming suspected diagnoses and, not infrequently, in detecting unexpected disorders in patients referred for cardiac evaluation.[1] We examined the usefulness of M-mode and two-dimensional echocardiography in the management of patients in a long term health care facility, where many patients are chronically bed-bound or wheelchair-bound, and where invasive cardiologic testing is not available and often contraindicated or impractical.

METHODS

In this retrospective study we reviewed the charts and echocardiograms of 1,401 patients hospitalized between 1977 and 1983 at Goldwater Memorial Hospital, a 912-bed long term health care facility in New York City. During that period, 3,257 pa-

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tients were admitted to the hospital. Thus, 40% of the patients had echocardiograms. Of these, 1,324 (94%) had echocardiographic studies that were technically adequate for interpretation. Seven hundred and two male patients and 622 female patients aged 9 to 101 years (mean, 68 years) had been hospitalized on the rehabilitation, medical, and skilled nursing services, for a period of from seven days to 40 years (mean, 482 days) at the time of study. Of these, 435 patients had M-mode studies alone (before 1980), and the remaining 889 had combined two-dimensional and M-mode studies.

Referrals to the echocardiography laboratory were initiated by attending physicians (cardiologists or internists) based on one or more clinical indications arising from the medical history, physical examination, or laboratory tests (Table I).

The earlier M-mode studies were performed with Ekoline 20A (Smith, Kline, and French, Sunnyvale, Calif) equipment using standard M-mode techniques.[2] Most studies were done using the parasternal long-axis approach; occasionally, the subxiphoid approach was employed. The combined two-dimensional and M-mode studies were obtained in the standard parasternal long-axis, parasternal short-axis, and apical four chamber views, using ATL 300 (Advanced Technology Laboratories, Inc, Bellevue, Wash) equipment.

RESULTS

Of the 1,324 interpretable echocardiographic studies, 382 (29%) were considered to be within normal limits, while 942 (71%) revealed one or more (total 2,059) echocardiographic abnormalities (Table II). These included 732 instances of chamber dilation, 513 instances of wall motion abnormality, 399 instances of increased wall thickness, 216 instances of valvular pathology, 42 instances of pericardial abnormality, and 157 other findings, including mitral anular calcification, aortic root aneurysm, intracardiac clot, and atrial septal defect.

In a total of 839 studies (63%), the echocardiographic findings confirmed the clinical impression of the referring physician or provided an explanation for a clinical finding (Table III). Of the remaining 485 studies in which the echocardiographic findings were not suggested by (or in some cases, even consistent with) the clinical indication for examination, 67 (5%) revealed a new diagnosis that resulted in a significant change in therapeutic approach (Tables IV, V). These cases included 23 patients with pericardial effusion, 14 with hypertrophic cardiomyopathy, ten with valvular vegetations, eight with mitral stenosis, five with left atrial thrombus, three with left ventricular thrombus, two with prosthetic valve dysfunction, one with constrictive pericarditis, and one with left ventricular pseudoaneurysm.

In addition, 66 (5%) patients who were referred for echocardiography with the clinical impression of congestive heart failure were found to have left ventricular hypertrophy with normal wall motion

TABLE I. Clinical Indications for Echocardiographic Studies

Patient history and/or complaints (N = 639)	
Previous heart disease or cardiac surgery	131
Chest pain	126
Palpitations	103
Stroke	98
Fever	72
Shortness of breath	54
Peripheral edema	19
Peripheral emboli	11
Cardiotoxic drugs	4
Abnormal physical findings (N = 624)	
Murmur	209
Arrhythmias	191
Signs of congestive heart failure	183
Abnormal sound	25
Pericardial rub	16
Abnormal laboratory findings (N = 354)	
Chest film	201
EKG	116
Previous echo	37

TABLE II. Abnormal Echocardiographic Findings

Chamber dilation (N = 732)	
Left ventricle	286
Left atrium	327
Right ventricle	86
Right atrium	33
Wall motion abnormalities (N = 513)	
Left ventricle	
localized	322
diffuse	160
Right ventricle	31
Wall hypertrophy (N = 399)	
Left ventricle	
symmetric	334
asymmetric (including obstructive cardiomyopathy)	20
Right ventricle	45
Valvular abnormalities (N = 216)	
Mitral valve	83
prolapse	44
stenosis	22
prosthetic	9
vegetation	7
flail	2
Aortic valve	126
thickening (no stenosis)	52
insufficiency suggested by fine flutter of anterior mitral leaflet)	32
stenosis	31
prosthetic	8
vegetation	3
Tricuspid valve	5
vegetation	3
stenosis	1
prosthetic	1
Pulmonic valve	1
stenosis	1
Pericardial abnormalities (N = 42)	
Effusion	39
Thickening	3
Miscellaneous (N = 157)	
Mitral anular calcification	107
Aortic root aneurysm	24
Intracardiac clot	18
Atrial septal defect	6
Tetralogy of Fallot	1
Ebstein's anomaly	1
Total number of abnormal findings	2,059

TABLE III. Anticipated Versus Unexpected Echocardiographic Findings

	No. patients	Percent of Total
Echocardiographic findings consistent with clinical indications	839	63%
Echocardiographic findings do not explain clinical findings		
No change in therapeutic approach	423	32%
Change in therapeutic approach	62	5%

TABLE IV. Echocardiographic Findings Requiring Change in Therapeutic Approach

	No. of Patients	Percent of Total
Hypertrophic cardiomyopathy	13	9.8
Pericardial effusion	23	17.3
Valvular vegetation	10	7.6
Mitral stenosis	9	6.8
Left atrial clot	5	3.8
Left ventricular clot	3	2.3
Prosthetic valve dysfunction	2	1.5
Constrictive pericarditis	1	0.8
Left ventricular pseudoaneurysm	1	0.8
Potential change in therapy (see text)		
Left ventricular hypertrophy	66	49.6
Total	133	100

and no valvular disease. While at the time of the study (1977–1983) this did not mandate a change in therapy for most patients, current concepts regarding the role of diastolic function in the pathogenesis of congestive heart failure might now favor the use of beta blockers or calcium channel antagonists to improve the compliance characteristics of hypertrophied ventricles.

DISCUSSION

Several authors have demonstrated the usefulness of echocardiography in the evaluation of known or suspected heart disease in both cardiovascular referral centers and in the general hospital setting.[3,4] In a retrospective study of M-mode echocardiography in a large teaching hospital, Goldman et al[5] found that the referring physicians felt the tests provided "crucial" information in 12% of the patients studied, while the independent board-certified cardiologist reviewers felt that the information was needed for a change in appropriate management in 8% of cases. The value of this harmless, painless procedure in assessing geriatric patients has also been described.[6] We examined the usefulness of this technique in a long term health care facility to which patients were transferred from acute care hospitals for management of chronic disabilities. Many of these patients had multisystemic disease requiring therapeutic interventions which may have been influenced by underlying cardiac disease.

TABLE V. Change in Therapy Suggested by Echocardiography as a Percentage of Initial Clinical Diagnosis

	Number of Patients	Change in Therapy as Suggested by Echo	Percent
Cardiomegaly	201	18	9.0
Stroke	98	6	6.0
Peripheral edema	19	3	16.0
Fever	72	10	14.0
Previous heart disease or cardiac surgery	131	9	7.0
Peripheral embolization	11	3	27.0
Chest pain	126	4	3.0
Abnormal ECG	116	3	2.5
Congestive heart failure and/or shortness of breath (including patient with left ventricular hypertrophy—see text)	237	77	32.0
Total		133	

We have shown that echocardiography is a useful tool for the diagnosis of structural and functional cardiac disorders suggested by a broad spectrum of signs and symptoms in patients who are frequently bed- or wheelchair-bound. These results are comparable to those obtained by Goldman et al[5] in their 1983 retrospective study of patients hospitalized in an acute care teaching hospital. While some investigators have concluded that M-mode echocardiography alone is rarely useful for the detection of clinically unsuspected disease,[7] we found that echocardiography (including two-dimensional imaging) may reveal "occult" disorders in a substantial number of such patients—more than one third in this series.

These patients included those with congestive symptomatology who were receiving digitalis preparations and were shown to have hypertrophic cardiomyopathies (some with outflow tract obstruction). We also identified patients in whom the finding of intracardiac clot mandated anticoagulation therapy. Findings that not infrequently mandated surgical intervention included large pericardial effusions (with and without evidence of cardiac compression), mitral stenosis, prosthetic valve dysfunction, and valvular vegetations in patients with endocarditis.

Although only 5% of the patients required immediate therapeutic manipulations based on the echocardiographic diagnosis, we suspect that the identification of disorders in others may have a direct bearing on future management decisions in patients with chronic disabilities who are often afflicted with multisystemic disease. This would seem particularly beneficial in institutions in which radionuclide scanning, cardiac catheterization, and invasive hemodynamic monitoring are not available, or in which chronically ill patients are not considered appropriate candidates for such testing. In addition to the 5% of patients whose echocardiograms mandated a change in therapy during the study period, we identified an additional 5% of patients with congestive heart failure whose echocardiograms demonstrated left ventricular hypertrophy and normal wall motion. This group constitutes a patient population which, given current concepts of diastolic function, might benefit from therapy with beta blockers and calcium channel blockers, rather than treatment with digitalis and diuretics. We conclude that echocardiography is a practical technique in the identification of cardiac abnormalities that are frequently present in patients hospitalized in long term health care facilities.

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The anencephalic fetus and newborn as organ donors*

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Anencephaly is a condition of arrested development of the brain; the cerebral hemispheres, and sometimes the entire brain including brain stem, are absent. Anencephaly is a relatively common disorder occurring approximately once in every 1,000 to 2,000 births.[1] An additional comparable number die in utero since the condition is usually lethal before birth or in the first few hours or days after birth. There may be other associated malformations.[2] Very rarely, a patient may survive weeks or months.

Anencephaly is easily detected by sonography, which is a procedure performed in most pregnant women between eight and 16 weeks of pregnancy for the purpose of establishing gestational age. If the condition is detected, the parents are usually offered the option of abortion.

New technology and advances in microsurgical techniques have made the transplantation of small organs into newborns and infants technically feasible. The need for small organs is considerable. In the United States, approximately 300 to 500 children die annually of end stage renal disease, about 400 to 800 children succumb to liver failure, and 400 to 800 die at birth or shortly thereafter of certain forms of congenital heart disease.[3] Today, childhood leukemias and other tumors can be treated with bone marrow transplantation. Children with enzyme and metabolic deficiencies such as insulin dependent diabetes mellitus might be treated with pancreatic transplants.[4-5] Immunodeficiency diseases in infancy and childhood can be treated with fetal lymphocyte or fetal thymus transplants.[6]

Fetal cells and fetal grafts survive much better and longer than organs transplanted from newborn or infant donors.[7-8] Fetal grafts are even successful across different animal species, ie, heterografts.[9-10] Fetal cells can be obtained, without harming the mother, by an invasive transuterine procedure of chorionic villous sampling by suctioning through the cervical os.[11] The villous tissue can be typed and matched to a potential recipient anywhere in the country. Thus, a pregnant woman with an anencephalic fetus could be flown to the location of the matched recipient, there to permit either fetal tissue to be extracted for transplantation, or abortion and organ donation, or delivery of the anencephalic baby which can then serve as an organ donor.[11]

THE CONTROVERSY OF RECENT CASES

A recent case received wide publicity in the lay press.[12-14] On October 12, 1987, a woman from Orillia, Ontario, Canada, gave birth to an anencephalic daughter. The mother knew in advance of her baby's condition but decided to carry the child to term specifically hoping to use her organs to save the life or lives of one or more other children. After birth, the anencephalic infant breathed on her own for about 14 hours, when she was placed on a respirator and moved to University Hospital in London, Ontario. Approximately 30 hours later she was declared brain dead by three physicians not associated with the transplant team who determined, after three separate ten-minute apnea tests, that she had no spontaneous respiration. After each apnea test, she was returned to the respirator to maintain organ perfusion and viability. The infant girl was flown to Loma Linda University Medical Center in California where another team of physicians verified her to be brain dead. At Loma Linda, a woman whose unborn child was determined to have hypoplastic left-heart syndrome underwent a cesarean section. Within hours, the baby with the hypoplastic heart received the heart of the anencephalic baby.

Michael Harrison, co-director of the fetal treatment program at the University of California at San Francisco, said that vital organs taken from anencephalic infants who have died naturally are "just not usable."[13] Harrison is a strong proponent of using anencephalic infants as organ donors and has proposed the establishment of a third definition of death called "brain absent." This would apply exclusively to anencephalics since the classic definitions of death—irreversible cessation of circulatory and respiratory functions and/or irreversible cessation of all functions of the entire brain including the brain stem—do not apply to liveborn anencephalics.[13] Harrison said that the Canadian doctors had performed "an elaborate little shell game" to get around the ethical stumbling block of the issue of brain death.[12]

Another ethical concern expressed in response to the above-cited use of the heart from an anencephalic child

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for transplantation is the possibility of abuse. There is the fear that it might create a "slippery slope" leading to harvesting of organs from severely handicapped newborns and from patients in comas or in chronic vegetative states who are not dead by any definition of death. Loma Linda's pediatric cardiothoracic surgeon-in-chief, Leonard Bailey, defended his use of the anencephalic Canadian girl as a heart transplant donor, saying these babies are nonperson humans and each one could salvage four or five other infants. Bailey claims that many parents of anencephalic babies are desirous of having their babies' organs donated to save the lives of other babies.[13]

LeRoy B. Walters, director of the Center for Bioethics at Georgetown University's Institute of Ethics, said

> if there is going to be any deviation in the policy involving anencephalic newborns...it should not be undertaken simply on the basis of private initiative. Rather, there should be some kind of public study process—presumably with the aid of a government-appointed commission. That should lead to publicly discussed guidelines for these cases and some kind of publicly announced policy for these cases.[13]

Less than a month after the Canadian anencephalic heart donor case was widely publicized, another case was reported in the lay press[15] in which the parents of an anencephalic fetus in Arcadia, California, allowed (or persuaded) physicians at Loma Linda Medical Center to maintain their baby on life support systems so that its organs could be donated to save the lives of other potentially salvageable infants.

LEGISLATIVE PROPOSALS

In 1986, San Francisco's State Senator Milton Marks introduced a bill in the California legislature that would have amended the Uniform Determination of Death Act (UDDA) to include a provision saying that an individual born with the condition known as anencephaly is dead.[16] Ethicist Alexander M. Capron, one of the framers of the UDDA, testified against Marks' proposed bill, saying:

> The proposal to add anencephalics to the category of dead persons would be a radical change, both in the social/medical understanding of what it means to be dead, and in the social practices surrounding death. Anencephalic infants may be dying patients, but they are still alive and breathing. Calling them "dead" wouldn't make them stop breathing...[and] we don't usually bury breathing people.[17]

Capron's position was supported by the United States Department of Health and Human Services.[18] In a letter to Bill Leonard, a Republican assemblyman for San Bernardino County, Dorcas Hardy, then assistant secretary for Human Development Services, wrote, "Because state statutes refer to total brain death, and anencephalic children are not totally brain dead, the use of such children as organ donors would be an abrogation of the uniform brain death code." The department would "not encourage" legislation that would equate anencephaly with total brain death, "as it conceivably could lead to the further expansion of the definition to include other types of neural tube defects, e.g., spina bifida," Hardy wrote.[18]

Capron also testified that such a practice would set

> a very bad precedent to start off with the most vulnerable patients if society wants to adopt a policy of sacrificing living patients for their organs.
>
> Unconsenting, incompetent patients who have never had a chance to express their views about whether, if near death but not yet dead, they would want their bodies cut up for purposes of organ donation, are the least suitable source.[18]

As a result of the medical-ethical-legal controversy surrounding his proposal, Marks withdrew his bill and cosponsored a modified bill with Senators Nielson, Rosenthal, and Watson that would establish a statewide bioethics task force to recommend a policy for dealing with infants with life threatening conditions, including the "feasibility and necessity" of infant organ transplants, the donation of organs from infants born with anencephaly, and any "necessary changes" in the UDDA.[18]

In New Jersey on October 27, 1986, Assemblyman Walter Kern, Jr, introduced a proposal permitting organ transplants from anencephalic infants provided the parents submit a written request for such organ donation "regardless of whether the infant has sustained an irreversible cessation of circulatory and respiratory functions or an irreversible cessation of all functions of the brain stem."[19]

In Great Britain, Magdi Yacoub, a noted British heart surgeon, was threatened with a lawsuit by a pro-life group which charged him with "treating vulnerable human beings as things" following his transplantation of a heart obtained from an anencephalic infant. The stir that followed the announcement of the threatened legal action prompted the British health department "to ask the Royal Medical Colleges to study the policies governing fetal and infant organ donation, to see if additional regulations were needed."[18]

MEDICAL LITERATURE REVIEW

The use of an anencephalic newborn as a possible cardiac transplant donor was first considered by Kantrowitz and his colleagues in June 1966.[20] These investigators transferred such an infant to their medical center and, when spontaneous and reflex motor activity ceased, mechanically ventilated the infant to sustain myocardial contractions. The anencephalic infant's heart ceased beating, attempts to perfuse and resuscitate the heart were unsuccessful, and the operation was abandoned.

In December 1967, Kantrowitz and his group transferred to their medical center another anencephalic infant.[20] The infant needed endotracheal intubation with assisted ventilation to sustain its myocardial contractions. The next day the infant's cardiac rhythm became irregular and both the anencephalic donor and the recipient infant, an infant with severe Ebstein's malformation, were brought to the operating room where external cooling in ice water was begun in both infants. One hour later, spontaneous cardiac activity in the donor infant ceased, his body temperature was 27°C, and a technically successful cardiac transplant was performed. The recipient baby died six and a half hours postoperatively of severe metabolic and respiratory acidosis. Kantrowitz's report[20] does not discuss the ethics of using anencephalics as organ donors but asserts that "anencephalic newborns were a reasonable choice as donors for babies."

The use of anencephalic infants as kidney donors was first reported in 1963 by Goodwin et al,[21] who learned from their obstetrical colleagues of

> a mother near term with an anencephalic monster waiting to be delivered. The plan was to induce labor and deliver the monster, allow it to die and then remove both of its kidneys together with attached segments of aorta and vena cava. They would then be transplanted into the newborn child who was dying with advanced hydronephrosis. . . Contrary to expectations, the anencephalic monster was stillborn because of congenital absence of the trachea. Thus, immediately the time element entered into this situation and may well have influenced the unsuccessful outcome.

The authors provide no ethical discussion but conclude that if a donor is a neonate, expected to die, provisions should be made for perfusion of the aorta and vena cava with a pump oxygenator so that the blood supply to the kidneys may be maintained, and that refrigeration should be employed.

In 1969, Martin et al[22] described "homotransplantation of both kidneys from an anencephalic monster to a 17 pound boy with Eagle-Barrett syndrome."[22] The anencephalic fetus exhibited no spontaneous respiratory activity at the time of birth and tracheotomy was immediately performed. Mechanical respiratory assistance was initiated and maintained in transit to the Children's Hospital where the transplantation was to be done. It was continued in the operating room until the kidneys, aorta, and vena cava of the donor had been isolated. Respiratory assistance was then discontinued. Five minutes after death, both kidneys were removed from the donor and transplanted into the right lower quadrant of the recipient.

The authors note that although not representing as abundant a source of organs for transplantation as cadavers or related volunteers, anencephalic infants are of significance, particularly in cases in which recipients are in an early age group or in need of transplantable vital organs. Associated additional congenital anomalies are rare, continue these authors, suggesting "the value of the anencephalic monster as a suitable donor for multiple organs. Since the condition is known to be fatal at birth, one would anticipate a minimum of legal, moral, or ethical obstacles to procurement of donor material."[22]

In 1970, LaPlante et al[23] described 17 kidney transplants performed in children during the previous eight years.[23] Two of the donors were "anencephalic cadavers." Also in 1970, Fine et al[24] described 23 children who received 24 renal homotransplants from both live-related donors and cadaver donors. One of their donors was a two-day-old anencephalic infant. The authors stated that "the ethical, legal and technical aspects of obtaining cadaver kidneys have been previously described."

In 1971, cadaver kidneys obtained from anencephalic infants dying shortly after birth were transplanted by King et al[25] into two children with renal failure. In the first case, permission to remove the anencephalic infant's kidneys was obtained immediately after death, which occurred one and a half hours after delivery. The kidneys, ureters, bladder, viscera, and great vessels were removed en bloc. In the second case, another anencephalic baby was detected in utero and arrangements were made for a possible kidney transplant. The baby was born precipitously and lived for only a few minutes. Permission was obtained immediately after death to excise the newborn's kidneys, which were removed en bloc. The time from the infant's death to the reestablishment of circulation through the grafted kidneys was two and a half hours.

These authors conclude that it is appropriate to utilize the kidneys from anencephalic babies for renal transplantation when the following criteria are fulfilled: the baby must be born alive; permission must be obtained from the infant's parents (after the birth) to remove the infant's kidneys after the moment of natural death; the ultra-experimental nature of the transplant must be fully explained to the potential recipient and the recipient's family and free and unanimous consent must be forthcoming; the potential recipient must have no close relative willing or able to donate a kidney or must be a poor risk for transplantation because of bladder anomaly, cachexia, or some relative contraindication to transplantation, making utilization of a healthy living donor kidney seem unwarranted because of the decreased likelihood of success.[25] This is the first albeit limited discussion of some of the ethical issues involved in using anencephalic donors for kidney transplantation.

In 1973, Lawson et al[26] transplanted a kidney from an anencephalic fetus of 37 weeks' gestation into an eight-day-old female infant with infantile polycystic kidney disease. Several days before referral of the recipient, a physician from another hospital had notified the transplant team that a cesarean section was pending in a young woman of 37 weeks' gestation who was carrying an anencephalic fetus. This woman desired to donate the kidneys of the fetus for transplantation and her physician planned to do the cesarean section at the transplant team's convenience. The anencephalic fetus was delivered by cesarean section and was kept in a sterile field while the nephrectomies were performed. No attempt was made to resuscitate the fetus during the nephrectomies. The authors do not state whether the anencephalic fetus was dead at the time of the nephrectomies nor do they raise any ethical issues involved in using anencephalics as organ donors.

In 1975, Salvatierra and Belzer[27] reviewed 350 consecutive cadaver kidney transplants. The youngest patient was an 18-month-old boy who received a one-day-old kidney from "an anencephalic monster." In 224 consecutive kidney transplantations at one hospital in West Germany, three patients aged 44, 37, and 13 years received double renal tranplants from pediatric cadaver donors aged one day, 23 months, and 19 months, respectively.[28] The one-day-old donor was an anencephalic newborn infant from a distant transplant center. The status of the donor at the time of the nephrectomies is not cited in the report.

In 1978, Kwun et al[29] transplanted ice-preserved kidneys from an anencephalic newborn infant within six hours of the time they were harvested. The status of the donor at the time of the nephrectomies is again not stated. Also in 1978, Iitaka et al[30] reported that of 14 anencephalic potential donors, the kidneys of six were transplanted to recipients ranging in age from four to 19 years. The other eight anencephalic infants were not suitable as donors because of rapid deterioration of vital signs, blood group incompatibility, and/or inability to perfuse the kidney. In each case, after parental permission was obtained, the anencephalic infant was transferred to the newborn intensive care unit. Most of the infants needed assisted ventilation and sodium bicarbonate to correct metabolic acidosis. To maintain blood pressure, plasma expanders and/or pressor agents were used. In addition, corticosteroids were given. After the infant was pronounced dead, the kidneys were harvested. The harvesting was done within 24 hours in all cases but one, in which the interval was 80 hours.

In 1979, Martin et al[31] reviewed 96 renal transplants in 77 pediatric patients. Kidneys from anencephalic newborns were utilized in six instances, with three being successful. These six cases are probably identical to those reported a year earlier from the same institution.[30] These authors state that "anencephalic newborn infants have been found to be a satisfactory source of cadaver donor kidneys."

In 1981, Kinnaert et al[32] described the case of a 23-year-old man who received both kidneys of a two-day-old anencephalic newborn whose kidneys were offered by the central dispatching office of Eurotransplant in Leiden, The Netherlands. In Leiden, the kidneys were removed en bloc together with the aorta and vena cava, flushed, and stored in cold Collins' solution. Transplantation was carried out in Brussels, Belgium, the following day. The status of the anencephalic donor at the time of bilateral nephrectomy was not stated. These authors also reviewed 14 transplantations of kidneys from anencephalic donors reported in the literature, seven of which were successful. The authors concluded that "the possibility of using anencephalics as a source of organs should be further explored" because of the shortage of cadaver kidneys.

In 1981, Moel and Butt[33] reported the use of four anencephalic newborn infants as kidney donors and noted that "the use of anencephalic donors as sources of kidneys for transplantation has been described" in the medical literature.

In 1984, Kinnaert et al[34] reviewed the nine transplantations performed through Eurotransplant with kidneys from anencephalic donors and cited 14 more cases reported in the literature. These authors supported their earlier contention[32] that anencephalic newborns might constitute a source of kidney donors for both children and adult recipients because of the shortage of cadaver donors.

With the exception of a brief statement by the Cincinnati transplant group[22,30] and a list of criteria cited by the Northwestern transplant group,[25] there is no discussion at all in any of the above reports about the ethical and moral issues involved in using anencephalic infants as organ donors. In 1987, however, Holzgreve et al,[35,36] in describing three cases in East Germany in which anencephalics were used as donors, discuss a variety of ethical problems that they considered before attempting to deliver anencephalic infants as organ donors for either children or adults. In the first case,

> an anencephalic fetus was identified by a practitioner at 36 weeks of gestation. After detailed discussion with the parents, the anencephalic child was delivered two weeks later, intubated immediately after birth, and given ventilatory support. The systolic blood pressure was kept at 80 mm Hg until the nephrectomy was performed about 45 minutes after delivery. The two relatively large kidneys were taken out en bloc...
>
> The second case was a twin pregnancy in which one healthy fetus and one anencephalic fetus were identified on ultrasound screening at 16 weeks of gestation. The parents decided against selected abortion of the affected twin and instead preferred to continue the pregnancy and allow the anencephalic twin to serve as an organ donor. After cesarean section at 36 weeks of pregnancy and delivery of a healthy infant, the anencephalic twin was intubated and given ventilatory support, and the systolic blood pressure was kept at 80 mm Hg. The nephrectomy was performed about 60 minutes after delivery. The kidneys were taken out en bloc...[35]

The third case of Holzgreve et al[36] is mentioned in a letter to the editor but no details are given. The authors concluded that because rapid deterioration of vital signs makes most anencephalic infants unsuitable as donors after death, "there are unfortunately no alternatives at present to intubating, immediately after birth, the anence-

TABLE I. Reported Cases of Anencephalic Newborns Used as Kidney Donors

Reference No.	Authors	Year	No. of Cases	Institution
21	Goodwin et al	1963	1 (case #6)	University of California, Los Angeles
22	Martin et al	1969	1	University of Cincinnati
23	LaPlante et al	1970	2 (cases #1 and 11)	University of California, Los Angeles
24	Fine et al	1970	1 (case #20a)	University of California, Los Angeles
25	King et al	1971	2	Northwestern University, Chicago
26	Lawson et al	1973	1	University of Oregon, Portland
27	Salvatierra and Belzer	1975	1	University of California, San Francisco
28	Dreikorn et al	1977	1 (case #1)	University of Heidelberg, West Germany
29	Kwun et al	1978	1	SUNY Health Science Center at Brooklyn, NY
30	Iitaka et al	1978	6 } same	University of Cincinnati
31	Martin et al	1979	6 } cases	University of Cincinnati
32	Kinnaert et al	1981	1	University of Brussels, Belgium, and the University of Leiden, The Netherlands
33	Moel and Butt	1981	4	SUNY Health Science Center at Brooklyn, NY
34	Kinnaert et al	1984	9 (includes reports in ref nos. 28 and 32)	University of Brussels, Belgium, and the Eurotransplant Foundation
35	Holzgreve et al	1987	2	University of Munster, East Germany
36	Holzgreve and Beller	1987	1	University of Munster, East Germany

phalic infant whose parents request kidney donation, until the kidneys have been removed."

In summary, the medical literature records at least 32 cases in which anencephalic newborns were used as kidney donors. Other reports of kidney transplantation in childhood[37–39] where cadaver organs were used do not specify the source of the donors. Some may have been anencephalics. Twenty of the 32 known cases were reported from six transplant centers in the United States, nine cases were transplanted through the Eurotransplant network, and three cases occurred in East Germany. Except for the latter three cases reported in 1987, none of the earlier investigators addressed in any detail the important ethical issues involved in the use of anencephalic infants and fetuses as organ donors.

LEGAL AND ETHICAL ISSUES

At the 1987 annual meeting of the American Academy of Neurology held in New York City, Ronald Cranford, chairman of the Academy's Committee on Ethics and Humanities, offered numerous arguments both in favor of and against the use of anencephalic newborns as organ donors.[40] Supporting such use are the observations that anencephalics are permanently unconscious patients, that the diagnosis can be made with 100% certainty (unlike chronic vegetative states), and that the condition is easily recognizable by parents and others. Furthermore, these patients are truly dying (unlike those in chronic vegetative states) since the overwhelming majority do not survive more than a few days. Cranford noted that some good may come to the patient's family from a tragic situation, and that certain good will result for the recipient if the transplant is successful. The health care team will also benefit from knowing that the recipient will benefit, and the family of the anencephalic may have less guilt. Finally, Cranford pointed out the social good of providing organs in view of the better techniques for transplanting organs in the very young.[40]

On the other hand, Cranford argued that public debate on the use of anencephalics as organ donors has not yet occurred, and that the removal of organs from such newborns is the proximate cause of death and therefore legally constitutes homicide and ethically constitutes euthanasia. He also suggested that we let the public know that we are contemplating using organs from anencephalics even at the risk of lawsuits. He stated further that such organ use would constitute a radical departure from the whole-brain definition of death. He expressed concern that the public's confidence in medicine may be undermined since society is not yet ready for organ transplants from anencephalic newborns. The issue of anencephalics as organ donors may soon be overshadowed by the issue of the use of fetuses and/or fetal tissue for organ donation, raising the problem of the preconscious fetus and abortion. The pool of anencephalic donors may decrease dramatically as pregnant women who learn by ultrasound or alpha feto-protein screening that their unborn fetus is anencephalic elect to terminate the pregnancy.[40]

Fletcher, Robertson, and Harrison[41] point out that under existing law in the United States, organs cannot be retrieved from anencephalics until brain stem function ceases, but there is no legal or ethical barrier to retrieval upon total brain death. However, the criteria for determination of brain death in children and especially young infants are extremely difficult to apply,[42] all the more so in an anencephalic newborn who has no brain. In the Federal Republic of Germany, the courts accept the concept that the anencephalic fetus has never been alive despite the presence of a heartbeat[35] and allow termination of pregnancy involving an anencephalic fetus at any time during gestation. This concept had been proposed in Germany in 1980 by Beller and Quakernack,[43] whose article is entitled, "Termination of pregnancy in the second and third trimester for eugenic indications."

O'Rourke[44] strongly criticizes the German thesis that "anencephalic infants may be considered brain-dead" or that "the anencephalic fetus, because of the absence of brain development, has never been alive" and insists that no organs should be removed until death has been certified on the basis of clinical signs. The German investigators responded by saying that

> Because rapid deterioration of vital signs makes most anencephalic infants unsuitable as donors after death . . . we believe that there are unfortunately no alternatives at present to intubating, immediately after birth, the anencephalic infant whose parents request kidney donation, until the kidneys have been removed.[36]

These authors argue for a "morally acceptable exception" to the total brain death definition for anencephalics.

In the United States, Chervenak et al[45] propose that third trimester termination of pregnancy is morally justifiable for anencephalics because two conditions are fulfilled: first, the fetus is afflicted with a condition that is either incompatible with postnatal survival for more than a few weeks or characterized by the total or virtual absence of cognitive function; and second, highly reliable diagnostic procedures are available for determining prenatally that the fetus fulfills either of the two parts of the first condition. These authors examined fetal disorders other than anencephaly and concluded that they do not clearly fulfill the two conditions. This article generated considerable correspondence, including a brief letter to the editor which said:

> The special article by Chervenak et al. was entitled "When Is Termination of Pregnancy during the Third Trimester Morally Justifiable?" The answer to that question should have constituted the shortest article ever printed by the *Journal*: "Never."[46]

The suggestion has been made[41] that the law be changed to permit organ retrieval from anencephalics by legislating a third definition of death: brain absence. Supporting this proposal is the fact that brain absence can be clearly defined and limited only to anencephalics, "so that individuals with less severe anomalies or injuries cannot be classed with anencephalic babies as exceptions for brain death guidelines."[47] On the other hand, babies born with hypoplastic lungs or hypoplastic hearts are also doomed to die without lung or heart transplants, but nobody has proposed lung absence or heart absence to constitute a new definition of death. Yet there is growing support for the idea that an anencephalic fetus be considered equivalent to brain dead for legal purposes to allow the family "to salvage from their tragedy the consolation that their loss can provide life to another child."[47] Amending the law to define anencephalics as dead would be a major change in legal policy and would generate enormous controversy.

Another approach is to consider an anencephalic to be a nonperson. Since anencephalic infants have no potential for cerebral development necessary for self-awareness, they are not "persons."[48] But how do we define personhood? We do not consider the absence of a face or eyes or limbs to negate a person's humanhood. Why should a hypoplastic or absent brain be viewed differently from hypoplastic hearts, lungs, livers, or kidneys? If brain transplants become feasible, would that restore the personhood of an anencephalic? We do not remove organs from people who are "nearly dead" whether they were hit by a truck, are dying of a terminal irreversible illness, or were born with a serious physical defect incompatible with life. We do not define personhood by a person's ability to speak or understand or respond cognitively to stimuli. If that were the case, permanently comatose patients or demented or psychotic or severely retarded patients would all be considered nonpersons. Yet no one has yet suggested that we "kill" all these types of patients to use their organs for transplantation.

Capron[17] persuasively argues that proposals to use organs from anencephalic infants to meet the growing need for transplantable organs are well-meaning but misguided. It would be unwise to amend the Uniform Determination of Death Act to classify anencephalics as "dead." They are in the same situation as other patients such as the permanently comatose. Likewise, continues Capron, amending the Anatomical Gift Act to permit organs to be removed from anencephalics would be unjust and would set a bad precedent. Rather, concludes Capron,

> Medical ingenuity should be directed toward finding ways to care for dying anencephalic [and other] babies so that when they become brain-dead, they can be organ donors [with their parents' permission]. Medicine should not embark on a course of sacrificing living but incompetent patients for the admitted social good of transplanting organs.[17]

Obviously aggressive therapy for an anencephalic newborn is also inappropriate. Dialysis is withheld from an anencephalic infant girl precisely because she is so handicapped that she cannot experience any benefit from the treatment.[49] Similarly, principles of justice require that comparisons of neonates for treatment or nontreatment should be done in terms of disease categories. All infants having an effectively treatable birth defect should be treated. If the condition cannot be effectively treated, then aggressive treatment should be withheld from all infants having the condition.[50]

Annas[51] points out that we have become more sophisticated in using anencephalic newborns as organ donors in the past two decades. We must be sensitive to the major ethical, medical, and legal issues.

> An ethically acceptable way may be found to maintain these infants for organ donation just as we now often maintain adults prior to pronouncing death. Changes in the law are neither necessary nor desirable. Whether anencephalic infants can be used as organ donors will depend not on the law, however, but on our ability to deal with them as members of the human race, and to safeguard them from harm as we try to turn their plight (and their bodies) to the benefit of others.[51]

CONCLUSION

Careful discussion is needed to see whether socially, medically, ethically, and legally acceptable ways can be developed for the retrieval and preservation of organs from brain dead anencephalics. Management of pregnancies involving anencephalic donors and the management of the anencephalic after delivery also needs to be carefully studied. If, for example, medical maintenance is determined to cause harm or suffering in anencephalic infants, their use as organ donors might be unacceptable. On the other hand, in the absence of such harm or suffering, if viable anencephalic organs can be retrieved when total brain death occurs, their use may be ethically and legally acceptable.

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MATERNAL IMPRESSIONS OR ACCIDENTAL COINCIDENCES—WHICH?

There are certain malformations of the child in utero that often make delivery difficult. Often embryotomy is necessary. This is usually the case in the treatment of difficult labor with an anencephalic foetus which is classed as being the commonest monstrosity. But common only when compared with double monsters, hydrocephalic foetuses, etc., and we may doubt the rarity of an hydrocephalus as compared with an anencephalus.

Clinical observations seem to bear out the theory that if a pregnant woman receive a mental impression of sufficient intensity that the mental impression received may be the means of arresting the development or causing the deformity of the foetus. All practitioners know or have read of cases in which infants have been born where the markings or deformities, etc., have corresponded in character to certain sights which have been said to have made a strong and lasting impression on the mind of the mother during the earlier period of uterogestation.

It is obvious that strong emotions have and will continue to most powerfully affect the functions of most of the important organs in the human body. Such emotions may derange or stimulate certain organs which, however, are connected with the brain by nerves. To the contrary, however, it must be admitted that the nervous system, and even the blood supply, of the mother and the foetus are distinctly separate. Yet, the pages of testimony, if true, which have been recorded would seem to justify the assertion that maternal emotions of sufficient intensity may be the means of arresting or rendering abnormal the development of the foetus, and if this is true, then the power and range of the human mind is exemplified . . . .

LESTER W. BELLOWS, MD
(*NY State J Med* 1912; 12:733-737)

REVIEW ARTICLE

Advances in the biology and carcinogenesis of basal cell carcinoma

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Basal cell carcinoma (BCC) of the skin is the most prevalent human cancer. Attributed largely to the cumulative effects of long term exposure to ultraviolet light, and predominantly affecting Caucasians, approximately 400,000 new cases are reported annually.[1] Although unprotected sun exposure is believed to be the major etiologic factor in skin cancer development, continued reduction in the stratospheric ozone filter may play a contributory role within the next several decades.[2] Despite the incidence and prevalence of BCCs, investigators have only recently begun to define better their biologic and carcinogenic characteristics. Importantly, exploring these areas can enhance our understanding and management of cancers in general.

This paper describes recent advances in the understanding of the biology and carcinogenesis of basal cell carcinoma. Particular attention is given to the origin and ultrastructural and growth characteristics of these widespread tumors. The possible roles of the enzyme collagenase and the basement membrane glycoprotein fibronectin in local BCC invasivity are also discussed. In addition, a number of less well recognized etiologies of BCC are reviewed. Finally, the complex nature of the interrelationships between tumor and stroma and tumor and host immunity, and their possible effects on local tumor destructiveness and metastatic potential, are explored.

BIOLOGIC CHARACTERISTICS OF BASAL CELL CANCERS

Histologically, BCCs are composed of masses of uniform cells that resemble epidermal basal cells. Usually undifferentiated neoplasms, some BCCs may exhibit differentiation toward appendageal structures, such as hair, sebaceous, apocrine, or eccrine glands.[3] Peripheral palisading of cells and retraction of the tumor mass from the surrounding stroma are typical features. Retraction of the stroma from the tumor mass, a helpful light microscopic diagnostic finding, is generally believed to result from the shrinkage of stromal mucin by the fixation process, but may also be a manifestation of regressive and degenerative changes within the tumor mass itself.[4] A mild stromal inflammatory reaction is another commonly associated histologic finding; ulcerated lesions, however, usually exhibit a pronounced inflammatory cell reaction.

BCC cells differ from normal epidermal basal cells by having a larger nuclear to cytoplasmic ratio; by lacking intercellular bridges under light microscopy (although they are present under electron microscopy); and by demonstrating, ultrastructurally, basement membrane thickening and discontinuities, loss of anchoring filaments and fibrils, and a significant decrease in the area of complete hemidesmosomes.[5,6]

Until recently, studies of basal cell carcinoma have been hindered by the lack of suitable tissue culture or animal models. Nude (athymic) mice, lacking a functional thymus-dependent immune system, have been shown to be capable of accepting transplants of human skin and many types of tumors while preserving their biologic and biochemical characteristics. Previous attempts, however, to transplant BCCs into these mice resulted in too low a yield of transplant acceptance to be of practical experimental value.[7-9] Transplanted cells of BCC survived in only five of 25 cases, and in only two of the five cases were the histologic characteristics of the resultant tumors similar to those of BCCs within the original human host.[10] Such low yields in these mice were believed to be related to the presence of residual immune surveillance mechanisms consisting of both nonthymus-dependent natural-killer-cell cytoxicity and humoral antibody response.[11]

In 1985, Grimwood et al[12] reported the successful transplantation of BCCs into nude mice, using methods found successful for increasing the transplant yield for other poorly growing tumors in these animals. Achieving immunosuppression through splenectomy and the concomitant administration of antilymphocyte serum, a 68% yield was achieved by these investigators. It was further demonstrated that the transplanted BCCs retained the histologic qualities and basement membrane immunopathology of the original tumor. In addition, the transplanted BCCs retained the same basement membrane characteristics of the original tumor with respect to the presence of a variety of noncollagenous and collagenous basement membrane glycoproteins.[13-16] These included the bullous pemphigoid antigen, laminin, fibronectin, and collagen types IV and V.

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BCCs are typically slow growing tumors, often taking months to years to reach significant proportions. Studying tumor cell proliferation by means of in vivo injection of ^{3}H-thymidine in patients with BCCs, Weinstein and Frost[17] calculated the BCC tumor cell cycle to be 217 hours, or a tumor doubling time of only a few weeks. Such a relatively rapid growth rate is at odds with the observed, characteristically slow BCC doubling time of months to years. More recently, however, Terz and coworkers,[18] using in vivo BCC labelling techniques, demonstrated that between 5% and 40% of tumor cells were arrested for variable time periods in the G_2 or G_0 phases of the cell cycle. They contended that such a large "resting" compartment could account for the observed slow growth rates of these tumors. Heenan and Galand[19] later reported a tumoral prolongation of the S (DNA synthesis) phase of the cell cycle, the effect of which could also contribute to the observed slow growth rates of BCCs.

Finally, supporting the notion that a BCC typically grows slowly because a large percentage of its cells proliferate either slowly or not at all, Grimwood et al,[20] using ^{3}H-thymidine in the nude mouse model, found that labelled material was primarily taken up by those cells located at the periphery of a BCC nodule, rather than by those located more centrally. These growth characteristics may also account for BCC's general insensitivity to cell-cycle-oriented therapies and for the previous lack of success in propagating BCCs in tissue culture.

A complex and important relationship has long been known to exist between BCC cells and their surrounding stroma. Arranged in parallel bundles around the tumor mass, the surrounding stroma proliferates along with the tumor.[21] In fact, attempts to autotransplant BCCs have demonstrated that such transplants survive only when they include the surrounding connective tissue stroma.[22] This apparently inextricable interaction of tumor and stroma may explain why the existence of purely intraepidermal BCCs (a situation in which contact between tumor and stroma is precluded) has not as yet been confirmed.[23]

Collagenase, an enzyme found within the dermis, preferentially degrades collagen types I and III, the major collagen components of the normal dermis; it degrades type II collagen poorly and spares type IV (basement membrane zone collagen) and type V altogether. Collagenase is also believed to play an essential role in the pathogenesis of BCC invasivity.[24] Much evidence exists demonstrating collagenase-induced collagenolytic activity within the stroma surrounding BCCs.[25-27] Until recently, however, it was unclear whether tumor or stroma was the source of the collagenase. More recently, using in vitro tissue culture techniques with nodular BCCs, Hernandez et al[28] demonstrated the presence of a factor(s) in their culture supernatants, which was found to stimulate fibroblastic collagenase synthesis. They postulated the mediator to be an aberrant interleukin-1-like cytokine. At least in part, it appears that, in this manner, nodular BCC tumor cells are capable of modulating their own dermal invasion through the production of these mediators.

Fibronectin is an epidermal basement membrane glycoprotein known to play a role in normal cellular attachment to the basal lamina, connective tissue, and surrounding cellular surfaces. It has also been shown to be important for epithelial migration during wound healing.[29] In addition, fibronectin may play an important role in modulating the relationship between BCCs and their stroma. Grimwood et al[30] demonstrated that BCCs grown in the nude mouse model produced and deposited fibronectin within the pericellular matrix. As a result, these investigators speculated that fibronectin may play an important role in facilitating BCC migration and invasivity.

CARCINOGENESIS

BCC is believed to originate from a disordered primary epithelial germ cell located either in the interfollicular epidermis or the outer root sheath of the pilosebaceous apparatus.[31,32] Chronic ultraviolet light exposure accounts for the vast majority of cases of BCC observed. The overwhelming predominance of BCCs on sun-exposed areas of the face, "V" of the chest, neck, and the dorsa of the hands, particularly in fair-skinned individuals, underscores the etiologic importance of cumulative ultraviolet light damage in the causation of these tumors.[33]

Nevertheless, the occasional appearance of BCCs on non-sun-exposed areas, such as the buttocks and genitalia, indicates that other factors may also play independent, additive, or synergistic etiologic roles in the development of BCCs. Clearly, in the autosomal dominant disorder basal cell nevus syndrome, and in the autosomal recessive condition xeroderma pigmentosum, genetic factors are of obvious etiologic significance.[34,35] Ionizing radiation has also been long recognized as another possible cause of BCC development.[36,37] Moreover, chronic systemic exposure to arsenic, while more commonly associated with the development of squamous cell skin cancers, has also been associated with the subsequent development of BCCs.[38]

Finally, numerous reports have suggested diverse forms of antecedent trauma as possible etiologic factors in the development of BCCs. Traumatic wounds reported to be associated with later BCC development include thermal burns, chronic leg ulcers, tattoos, colostomies, and varicella and vaccination scars.[39-44] One possible explanation for the relationship between BCCs and prior trauma suggests that trauma-induced connective tissue scarring may play an initiating role in the neoplastic transformation within the overlying epidermis.[45]

To understand the clinical outcome of BCC in any patient, both the intrinsic properties of the tumor and host factors must be considered. While possessing the potential for considerable destructiveness, BCCs are ordinarily only locally invasive. They rarely metastasize. To date, only 175 cases of BCC metastasis have been documented in the literature.[46] Those tumors that do metastasize are typically located on the head or neck, are massive and ulcerating, and involve deep local invasiveness. In addition, they tend to be recurrent tumors which have been present for at least several years. In general, visceral metastases may occur both lymphatogenously and hematogenously, and the overall prognosis is poor. Interestingly, Dzubow[47] recently reported a case of metastatic BCC in which the primary lesion had been noted for only six months and was nonulcerated.

Lack of tumor autonomy is one possible explanation for the extremely low metastatic potential of BCC. In support of this notion, BCC cells cultured in vitro converted to normal keratinizing epithelium when separated from their surrounding stroma.[48] Moreover, BCCs devoid of stroma fail to grow when transplanted into the anterior chamber of the rabbit's eye, a site which has been shown to support the growth of other heterologous tumors.[49] Blewitt[50] has suggested that metastases are more likely to occur when local destruction has progressed to the point of tumorous invasion of larger caliber blood vessels capable of accommodating both tumor cells and stroma.

Host immune factors are also believed to play a role in local tumor invasiveness or propensity for metastasis. The high prevalence of skin cancer, including BCC, in immunosuppressed renal transplant recipients lends support to this notion.[51] The recent report of the development of metastatic basal cell carcinoma in a patient with acquired immunodeficiency syndrome-related complex provides additional evidence for this contention.[52] Small, well-localized tumors were associated in one study with elevated T cell levels and moderate to heavy lymphocytic infiltration, compatible with delayed hypersensitivity responses.[53] In contrast, large, more deeply invasive tumors were found to be accompanied by low T cell levels and absent lymphocytic infiltration, suggesting poor delayed-type responses. In another study, numerous plasma cells capable of secreting all immunoglobulin classes were observed within the surrounding stromal inflammatory cell infiltrates.[54]

One study in humans suggests an immunologic explanation for the preponderant development of BCCs on ultraviolet-damaged skin.[55] In that study, decreased local cell-mediated immunity was observed in chronically sun-damaged skin as evidenced by both decreased reactivity to the potent sensitizing agent dinitrochlorobenzene, and to intradermally injected antigens. This contrasted sharply with the demonstration of normal cell-mediated immunity to the same antigens in normal skin that had not been exposed to ultraviolet light. More recently, in laboratory animals, Kripke[56] demonstrated both sun-induced cutaneous malignant transformation and specific immune system suppression, permitting tumor growth.

The precise role of immunity in the pathogenesis of BCC remains to be elucidated by further research. However, based on available data, it is tempting to speculate that in man chronic ultraviolet light exposure (especially in genetically predisposed individuals) may not only be responsible for initiating cutaneous carcinomatous transformation within the epidermis, but may also facilitate tumor development by inhibiting the skin's immune "surveillance" system.

In this paper, we have explored recent advances in the understanding of the biology and carcinogenesis of basal cell skin cancers, the most common malignant neoplasm affecting humans. Much has already been learned about this tumor, particularly in the last decade. However, much additional BCC research is needed to further explore such areas as the possible effects, if any, of such soluble factors as lysosomal enzymes, tumor angiogenesis factor, and other as yet unidentified tumor growth promoters. In addition, more research is needed in the area of tumor immunobiology to further define the pathogenetic relationship between neoplasm and host. The benefits of such research will no doubt continue to shape current understanding of carcinogenesis in general. Hopefully, this will result in the development of more effective and specific measures for preventing, detecting, and treating cancers in the future.

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ETIOLOGY OF CANCER

Until the middle of the nineteenth century there prevailed the opinion that cancer arises in the lymph or other fluids of the body. With the appearance in 1858 of Virchow's epoch-making publication, *Die Cellularpathologie*, in which the author presented the maxim, *Omnis cellula e cellula*, the modern conception of the mechanism of tumor formation was established. With few exceptions, the cells of the adult animal or plant organism possess a very limited capacity for proliferation and are endowed instead with differentiated and highly specialized functions. A tumor cell, on the other hand, is a cell which has lost its specialization and has regained the power for proliferation which normal body cells possess only during the fetal life of the organism. This characteristic of a tumor cell is true for both benign and malignant tumors. The cells of malignant tumors, the cancer cells obtain at the same time the power of destruction of the normal tissues which lay in the path of their development and proliferation.

Virchow, who established the conception of the cellular derivation of cancer, maintained that every cancer cell, be it carcinoma or sarcoma, develops from connective tissue cells and that the latter are transformed into carcinoma cells by the aid of a metaplastic "*irritation*." Remak and Hiss, who established the fact that each cell in an adult organism must be derived from one of the three embryonal germ layers, held that a carcinoma cell must be derived from an epithelial cell. Thiersch, through his extensive histological studies of epitheliomata, demonstrated conclusively that a carcinoma cell is derived from an epithelial cell. He defined the cellular theory of Virchow more strictly by the principle, *Omnis cellula e cellula ejusdem generis*. Further investigation of Thiersch, Waldeyer and their followers, studies which continued for years, established irrevocably the fact that cancer in any organ develops from the parenchymatous cells of this organ. Secondary, metastatic tumors, wherever formed, are derived from the cancer cells of the primary tumor and consequently in the ultimate analysis are derived from the parenchymatous cells of the organ in which the primary tumor took its origin. In some instances, like a metastatic carcinoma of the rectum, in the brain or a metastatic carcinoma of the breast, in a bone, the derivation is quite evident. In other cases, the metastatic tumor may differ morphologically to a certain extent from the primary tumor (metastases of a scirrhous carcinoma of the breast obtain the form of a solid carcinoma), but the derivation can always be easily traced. . . .

ISAAC LEVIN, MD

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SPECIAL ARTICLE

A view of life and death from a choir loft

Pascal James Imperato, MD

The choir loft is still there, set against three towering stained glass windows. And so is the organ, whose pipes continue to send their melodies out into the high-vaulted Romanesque church where I sang as a choir boy for several years. It is an enormous church of yellow brick and red roof tiles whose interior is covered with black, red, and cream-colored marble. My schoolmates and I sang in that choir loft at weddings and funerals and also served as altar boys at baptisms. We saw grief and bereavement, the joyous responses to a baby's smile, and the happy faces of hundreds of brides and grooms as they recessed down the aisle. We were intimate participants in the rituals of passage even though we were strangers to those whose passage it was. We witnessed the ritual beginning and end of life and the nuptials in between.

Not a baby was born in the parish that we didn't know about and no death escaped our notice. We were invited to every wedding, not by the bride and groom nor by their families, but by necessity. As we sang we looked and observed and gained a unique perspective of life that few our age had. This view at the time seemed irrelevant to what really mattered for us—baseball, comic books, and a future free of fractions and long division. The value of what we absorbed through repetitive exposure wouldn't be known to us for years to come.

This view from the choir loft was enhanced by our experiences in the world in which we lived. There death wasn't sequestered in hospitals and funeral parlors but was an integral and accepted part of life at home. Multigenerational families lived side by side for decades and this gave children a frequent experience of death linked to bereavement. For the grandmother or great-uncle who died was not an unfamiliar, remote figure off in some nursing home, but the gentle, white-haired lady who tended the rose bushes in the spring and kindly old man who walked the dog soon after dawn. Their passing created a social loss even for us children and the sorrow we experienced prepared us for the future in ways we couldn't foresee.

Most medical students today learn about death and dying in formal courses devoted to the subject. The physical, emotional, and social dimensions of dying are analyzed and probed in lectures and seminars and learned in the abstract. In one course with which I'm familiar, students are asked to fill out their own death certificates. This is an uncomfortable experience for many of them because it challenges that blissful sense of immortality with which youth is endowed. Yet it provides them with a degree of control over their own fate, a say in how they are to die and when. Most live into their late 80s and die abruptly or after a very brief illness of a few days, having lived an otherwise healthy life. Is this wishful thinking or a reflection of their actual perceptions of life and death? I suspect it is a combination of both. I also suspect that my fellow choir boys and I would have told a different story on such certificates. Like most young Americans today, medical students have little direct personal contact with death and dying, unless they have lost a loved one. Their zest for life, their dreams for the future, and the absence of limits to their horizons push thoughts of death aside. It is an event for other times and places and for other people. Yet they must eventually meet death and dying when they become clinical clerks in hospitals. They bring to the encounter little or no experience with either. Their first view of dying is often cluttered with tubes and respirators, monitors and watchful eyes, mustered to forestall dying by barely sustaining life. Beyond the wall of life supports is a human being who once walked in the sun and felt the falling rain, someone who laughed and cried, someone with memories, family, and friends. The encounter is only with death and dying and not with the person to whom they are occurring. Death and dying are seen in a technological context that supports life but which doesn't enhance humanity.

When I grew up in the 1940s in a semirural community in southern Queens, New York, known as Ozone Park, death and dying were events of the home and hearth and not of the hospital. The visual metaphors of death were integral symbols in the life of our neighborhood. Drawn window shades and a bouquet of white flowers tied with purple ribbon attached to the door frame not only announced that someone had died, but also that they were being waked at home. Men tipped their hats to the house as they walked by and we children observed silence within an invisible perimeter around the house. Children couldn't be shielded from death because it was close and intimate. It was down the street and around the corner, often arbitrary and unexpected, affecting not only an individual family but the whole community. It was not just old people who died, but children as well. I vividly remember

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when a five-year-old girl died six doors down the block from us. She was pretty and fair haired, but "her blood turned to water," as my mother explained. My sisters and I watched in silence as her white casket was carried out of the house to the waiting hearse.

Our next door neighbors, the Brennans, had a great-grandmother living with them. Maureen Brennan, whom we called "Buttercup," regularly took me to visit her in an upstairs bedroom. I remember her as a soft-spoken, gray-haired old lady lying in bed who always had a kind word for us and a smile even though she was dying. I didn't fully understand what dying meant then and thought of it as leaving for a trip to a far-off place. When she died, it perplexed me that they carried her out of the house in a brown wooden casket, for I had expected to see her come out with a suitcase in hand. I ran alongside the slow moving hearse for a few blocks shouting goodbye, and was disappointed that she didn't look out and smile back.

My comprehension of the meaning of death evolved as I grew older. However, it was singing in the boys' choir at Nativity Church that truly opened broad vistas not only on death but also on other concerns of adult life. This choir was something of a chaotic institution until 1948 when Mother Julia Rollo arrived. She was one of three sisters of the Pallottine order whom the pastor brought into the parish to do social work. Monsignor Leopold A. Arcese, the pastor, whose formidable sounding name matched his parishioners' perceptions of him, essentially had Mother Julia do triple duty. She not only supervised the parish's social work, but also played the organ and directed the boys' choir. The previous director of the choir was a priest whose temperament was ill matched to controlling bands of high spirited boys who usually turned the choir loft into a scene reminiscent of life aboard a Viking *knarr*.

It was clear from our first encounter with Mother Julia that a new day had dawned in the choir loft. She was candid, outgoing, and experienced, and knew all about throwing snowballs at the nearest chandelier and punching a fellow chorister in a vital area just as he hit a high octave during a solo. Whereas previous directors had opted to fill the loft with 30 or more boys, Mother Julia swept most of them out, retaining only a dozen who could and who wanted to sing. For Easter and Christmas, when the loft had to be filled with an army of boys in black cassocks and white surplices, she conscripted 20 more a month before, drilled them in Latin and how to carry a tune, and then let them go, much to her relief and theirs. She had all of our telephone numbers, visited our homes at regular intervals, and phoned each Saturday morning to tell our parents the schedule for the weekend. And what schedules they were! Often there was a funeral and two weddings on Saturday morning and as many as three weddings on some Saturday and Sunday afternoons.

At the time that Mother Julia arrived a newly ordained priest was assigned to the parish who was placed in charge of the altar boys. Father Dominick Adessa, like Mother Julia, represented a wave of change in the management of the altar boys. He built up morale and chose dependability over numbers. In scheduling the weekly assignments which were posted each week in the front vestibule, he had to be careful not to deplete the choir since some of us were also altar boys. Thus my fellow choir boys and I usually functioned as altar boys at services where no singing took place.

Funeral services in those years were grim affairs. As the funeral cortege arrived the bell in the church tower would slowly toll, which during weekdays was a signal for us to bolt from our arithmetic lesson and position ourselves in the choir loft. The processionals and recessionals were dirges which brought even the bravest of mourners to tears, and all of the vestments worn by those officiating were black. The most somber hymn was the Dies Irae—that day of wrath, that dreadful day. As family and friends processed behind the coffin into the church, Mother Julia played a dirge chosen from one of several printed in the black-bordered requiem music book. We observed who among the mourners cried and who didn't and from this deduced, perhaps wrongly, who really loved the deceased and who didn't. We also noted the order in which people processed, next of kin coming first, then other relatives, then friends. A man or a woman's social connections walked beneath our gaze and from them we came to a number of conclusions. Even the kind of coffin used—oak, walnut, metal, or cloth covered—was a social message reflecting the values, attitudes, and financial means of the survivors. It wasn't so much these conclusions that really mattered, but the fact that we had come to them. We had observed, thought, and reflected, unknowingly using a ritual for the dead to obtain insights into the broader canvas of life.

Mother Julia took her cues from the altar through a rear view mirror on the organ which one of us occasionally turned when she wasn't looking. This caused a momentary flurry of exasperation on her part and a lapse in the order of the service. However, she always took it in good humor, as she did our giggling while singing. She dealt with our giggling by drowning it out with a loud rush of sound from the organ.

Our first contact with a wedding was usually when we asked the groom and best man for a tip as they waited in the front vestibule. Most obliged with a dollar or two which we took over to Mike's gasoline station. There Mike opened the cash register and handed out the coins to the four or six outstretched hands. As much as we disliked arithmetic in school, we all did simple division on the way over to Mike's and had a pretty good idea of our just share by the time we arrived. When a small amount was left over he acted as the arbiter and gave it to the youngest choir boy. On occasion grooms and best men refused to give us anything, claiming that they had already paid the church for the services, or else expressing doubt that we were legitimate. This put us in a foul mood and our conspired retaliation took the form of singing off key during the high octaves of the Ave Maria. Mother Julia would drown us out with a jolting blast from the organ which turned every head in the church at the peak of the wedding service. But we were pleased that we had had our revenge.

Dress and comportment were the social signals of guests who arrived for a wedding. A man who displayed effortless comfort in a tuxedo said as much about himself as one who struggled against a starched collar and stiff new shoes. How a woman carried her handbag, walked down the aisle, and acted in the pew said more than the expensive new dress she wore. Usually the bride's side of the aisle held many more guests than the groom's. We reasoned, probably wrongly, that because the bride's family paid for the wedding reception (the groom only paid for the flowers and the limousines), they invited more people. When my own three sisters walked down the same aisle years later I learned that brides at that time invited large numbers of their friends and acquaintances only to the church and that many of them came, in part out of curiosity to see the bride's gown and those of the attendants. I should have surmised this earlier on because my sisters constantly interrogated me about weddings but never asked about funerals and baptisms. The bride's gown was especially important to them, as were her veil and bouquet. We choir boys certainly came to judgments not only about the gowns but also about the people wearing them. However, my sisters were only interested in the gowns.

One cold Sunday afternoon in February, Joseph Hassan and I were the only two choir boys who showed up for a wedding. The groom looked angry and nervous so we decided to ask the best man for a tip. He didn't look much happier than the groom and refused, replying that the singing came with the "fee" the groom had paid the church for the service. We tried to reason with him, saying that it was customary to tip the choir boys even though the church had been given a "donation"—we knew the correct noun. He became quite agitated at this and threatened to punch us if we didn't leave them alone. It perplexed us that they were so unhappy on what should have been a joyful occasion.

Mother Julia only had to take one look at us to know that something unusual had happened. She decided to speak with them herself. With the two of us following behind, she briskly walked down the side aisle toward the vestibule where they were waiting. She was a small, frail woman, but with her black habit billowed by the quickness of her step, she looked twice her size. The two men stared in disbelief as she headed straight for them. Mother Julia couldn't have spoken a half minute before the groom gave her a dollar. With the dollar in hand she told him it was insufficient. The best man then pulled a five dollar bill from his wallet which made six, more than we had ever received before. "That will do," she said. She then asked the groom why he was getting married in February, telling him it would have been better had he waited until June. He said that his future wife didn't want to wait, that they were sort of in a hurry. "I see," she replied understandingly, obviously aware of something that wouldn't occur to me until years later.

Neither Mother Julia nor Father Adessa liked the idea of our asking for tips. However, they tolerated it as a small price to pay for our dependability. They rewarded us themselves with chocolates at Easter, hard candies at Christmas, and outings in the spring and fall, but must have wisely surmised that an additional enticement was necessary to get boys to give up their Saturday and Sunday afternoons.

By the time I was in the eighth grade I had shared final rites with widows and widowers, brothers and sisters, parents and orphans, and had also been there for the man who had died alone. I had looked beyond the tears and sobbing, wondering and worrying about the fate of both the mourners and the deceased. Although I didn't carry the burden of bereavement I saw its effects on those who did. This knowledge wasn't devoid of feeling because it evoked sympathy for the mourners and fears for myself and my family. I reluctantly came to the conclusion that in the far beyond this scene might enter my life in a more intimate way. I found it difficult to reconcile this reality with my boyhood hopes and expectations and my view of life as almost eternal. Yet I now knew that death was there, lurking as an inextricable part of life that couldn't be wished away or negotiated with regard to time and place.

My voice began to change in the eighth grade and my parents were convinced that my singing career was over. One of our neighbors, Henry Gruber, who was a cantor in the local temple, had over the years passed judgment on my voice and my mother asked him to give an opinion about my future as a singer. Sitting at our kitchen table he listened as I sang a few lines of the Kyrie Eleison, and nodded approvingly. Then turning to my mother he said, "A tenor he won't be but a bass he will." And he was right.

Mother Julia's parting gift to me was a book, *Sixty Saints for Boys*. Inside of it I found a note that began with, "For the faithful but mischievous choir boy. . ." With that one brief phrase she revealed her own special view of humanity which emphasized virtue and minimized fault. She continued playing the organ for another 17 years and peacefully died on a spring day in 1967 at the age of 71 in the convent where she had lived for so many years.

When I left the choir loft for the last time I carried away more than just memorized lyrics and the ability to read a musical score. I had shared in some of life's most poignant moments which had taken me to the other side of my boyhood horizons. It was both these moments and the insights they brought forth that would outlive the memorized lines of Latin. In future years they would help me to see that the person attached to the respirator had family and friends and once saw a summer's breeze dance across the grass.

On the history of schizophrenia

Evidence of its existence before 1800

NIGEL M. BARK, MBBCHIR

Schizophrenia was named by Eugen Bleuler[1] in 1911 in the introduction to his book, *Dementia Praecox or the Group of Schizophrenias*, but, as he states, "the whole idea of Dementia Praecox originates with Kraepelin" who "established . . . this disease group." Bleuler does trace the origins of the concept back through Morel's "demence precoce" to Esquirol early in the 19th century. He does not indicate whether he believes it was a new disease or merely recognized as distinct from other psychoses and then separated by definition.

However, many people now believe that schizophrenia was very rare or did not exist before the 19th century. This is because the concept was new and because there was a great increase in the number of mentally ill persons—probably with schizophrenia—during the 19th century. They also point to the absence of good descriptions of schizophrenia before the 19th century. Cooper and Sartorius[2] ask, "Why have these ideas [current concepts of schizophrenia] been so late in developing?" They suggest that it was because the disease was either new or at least had become chronic with industrialization.

Several authors conclude from the apparent absence of descriptions of schizophrenia before the last century that schizophrenia did not exist then. Hare[3] states, " . . . descriptions of a disorder corresponding to Kraepelin's schizophrenia are almost unknown in the literature before 1812," and " . . . one may search in vain for any such description before the nineteenth century."[4] Cooper and Sartorius[2] ask, "Why are good descriptions of what can now be recognized as chronic schizophrenia so scarce in European Medieval and earlier literature?" Torrey[5] provides a more detailed discussion of the subject. He states that although descriptions of madness, including delusions and hallucinations, date to ancient times, none of them describe schizophrenia as we know it, with onset in early adulthood, progressive deterioration, the constellation of symptoms, and a chronic course. He says the first actual description of what we now call schizophrenia may have been made in 1656, with the first clinical descriptions being made by Pinel in 1806 and by Haslam in 1809.[5]

The enormous increase in the number of the mentally ill in the last century is thought by some to be further evidence that schizophrenia was a new disease. Rosen[6] has collected the contemporary comments of 19th century writers who believed that the incidence of serious mental illness had increased greatly. Hare[4] gives reasons for believing that the increase was due to schizophrenia and he provides detailed evidence for the increase in the numbers of the hospitalized mentally ill in England and Wales in the 19th century.

These views are at variance with the traditional view of psychiatrists as enunciated in, for example, the *Comprehensive Textbook of Psychiatry.*[7] This view holds that people with schizophrenia always existed but were hidden away or "cloaked in the shadows of superstition and religious zeal."[7(p1105)] Before the last century such people were in "jails and poorhouses and the like or wandering abroad,"[7(p60)] the apparent increase in numbers being due to an increase in hospitalizations only. A recent paper by Jeste et al[8] discusses why early references to possible schizophrenia might be rare even if the condition was not. The present paper aims to show that descriptions of schizophrenia were not so rare.

Does it matter whether schizophrenia existed before the last century? I think it does, because, for example, Torrey[5(pp181,183)] and, to a lesser extent, Hare,[9] use the earlier absence of evidence of schizophrenia as support for their theories of a viral cause of schizophrenia. Likewise, Murphy,[10] in a brief review, while concluding that the relationship between civilization and schizophrenia remains doubtful, suggests that it is the complexity of civilization that may be harmful. Cooper and Sartorius[2] suggest that industrialization leads to chronicity. Evidence of schizophrenia in earlier, "simpler" ages may possibly render such arguments void. The incidence of illnesses that have one major cause tends to vary from time to time and place to place. Illnesses that have multiple causes or that can be viewed as multiple diseases (like cancer and mental retardation) tend to be more stable in number in place and over time. Thus the history of schizophrenia may tell us something about its nature.

Clearly there are problems in looking for past evidence of any condition: there may be insufficient evidence to permit accurate diagnosis, translation from one language

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to another may result in inaccuracies, and words in one language may mean different things at different times (or even at the same time, as exemplified by *mania*, *melancholia*, and the term *schizophrenia* itself).[11] However, where detailed descriptions of people and behavior and illness are given, less depends on the meaning of individual words. Various authors such as Murphy[12] or Westermeyer and Sines[13] have demonstrated that neither language nor culture is in itself a barrier to diagnosis.

Schizophrenia can now be reliably diagnosed according to several different definitions.[14] Questions arise as to validity: which is the "real" or "best" definition, and whether such a current definition could be used to diagnose schizophrenia in earlier ages. There is considerable overlap among definitions[14] and there is good evidence for validity by prognosis[15] and by genetics.[16-18] Furthermore, it is generally agreed that despite changes over time in the nature and frequency of some symptoms like catatonia, the current concept and most frequently used definitions (RDC, DSM-III, PSE derivations, ICD-9) of schizophrenia are closer to Kraepelin's original concept of dementia precox than some of the concepts used in the intervening years.[19,20] This concept of schizophrenia, and its definitions, have been shown to be useful and valid across cultures—both in mental health facilities[21] and in general populations—in a variety of underdeveloped countries.[12,13,22] Therefore, it is reasonable to assume that where there is sufficient information it can be applied in different ages. This concept of schizophrenia is of a chronic condition with characteristic thought disorder, delusions and hallucinations, with inappropriate affect, psychomotor restlessness or catatonia, bizarre behavior, deterioration in ability and self-care, and an absence of gross brain ("organic") pathology.

Although Cooper and Sartorius[2] state that current concepts of schizophrenia did not develop until the late 19th century, there are, as Torrey[4(p27)] acknowledges, earlier good descriptions of schizophrenia from the beginning of that century: by Pinel,[23(pp160-164)] calling it Dementia, by Haslam,[24,25(pp64-67)] by Burrows in 1828,[26] and by Griesinger in 1845.[26,27] This paper examines the evidence before the 19th century. It complements rather than duplicates the paper by Jeste et al.[8]

EARLIEST DESCRIPTIONS

Paranoid delusions and auditory hallucinations are described in the ancient Mesopotamian Old Babylonian period (the first half of the second millenium BC).[28] For example, a treatment manual prescribes a certain ritual to be performed "if a man has 'mischief makers' who persecute him with tongues, spread rumors, tell tales about him or slander him, if . . . evil machinations of men are turned upon him." There are descriptions of "witches" that cause irritability and "spoken and overheard phrases of ominous significance to the hearer."[28] However, the full syndrome of schizophrenia is not described.

The ancient Indian system of medicine, Ayurveda, provides the first systematized descriptions of psychopathology. Dube[29] identifies seven Ayurvedic diagnostic categories as being comparable with the schizophrenia described in *International Classification of Diseases*. The compendium of Caraka (or Charak) who is supposed to have lived around 600 BC[29] talks of endogeneous insanity (as opposed to exogenous, caused by possession) and lists as premonitory symptoms: "feeling of voidness in the head, noises in the ears, anorexia, misplaced mental absorption, anxiety, intoxicated condition of the mind, frequently dreaming of roving, moving . . . "[29] The general symptoms are "confusion of intellect, extreme fickleness of mind, agitation of the eyes, incoherence of speech, mental vacuity, lack of mental ease."[29] Other symptoms suggest that organic conditions are included, but the symptoms of different types of endogenous insanity are interesting:

> a) Vata variety—constant wandering, jerking of eyelids, brows, lips, and other parts of the body, incoherent talk, laughing, dancing, singing, loudly imitating the sounds of the lute, flute, conch; adorning with queer and unornamental objects; hankering after unobtainable viands, emaciation, swelling; redness of the eyes.
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> b) Pitta variety—irritability, anger, excitement in the wrong place, striking oneself or others with weapons, sticks or fists; nudity, craving, prolonged attacks of anguish, coppery green, yellow and furious look of the eyes. [Dube[29] includes here unfounded and bizarre delusions of suspicion, intolerance, visual hallucinations.]
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> c) Kapha variety—rooted to one spot, silence, no inclination for movement, dribbling of saliva, lack of desire for food; love of solitude, constant somnolence; whiteness and fixity of eyes.[30]

It is not stretching credibility to think that hebephrenic, paranoid, and catatonic schizophrenia, respectively, are included in these three descriptions, although Dube[29] identified Vata as mania (a modern diagnostic problem). Among the other Ayurvedic diagnoses encompassed in schizophrenia he includes "Pishachagraheet," which has symptoms that include "keeping limbs in bizarre position; incoherent and irrelevant talks on unrelated topics, thought blocks, ambivalent activities, mind wandering, preference for lonely places, collecting and decorating with rubbish, dirty, smelly, degraded and dilapidated, laughing, singing, dancing, cries."[29] This could certainly be a description of someone with schizophrenia. Haldipur[31] discusses the concept of insanity in Charaka Samhita and states that a strong case can be made that schizophrenia existed in India and was described in that book. I agree, but as Dube[29] cautions, details of history and course of illness are not given in the Ayurveda so diagnosis cannot be certain.

In the Bible there are few descriptions of conditions that could be schizophrenia.[32(pp311-315)] The seven years in which Nebuchadnezzer "ate grass like oxen" and neglected his self-care, with no antecedents and complete recovery, was more like a prolonged depression than schizophrenia.[33] David's feigned madness to escape from Achish, the King of Gath, of whom he was "sore afraid," is worth noting: "He changed his behavior before them . . . he scrabbled on the doors of the gate and let his spittle fall down upon his beard." Achish's reponse was, "Ye see the man is mad . . . have I need of mad men that you brought this fellow to play the mad man in my presence."[34] This shows that despite the paucity of descriptions of "madmen" in the Bible, "madness" could be used as a disguise, and was not so rare or unusual that it drew attention. Smith[35] states that Ezekiel is the finest example of a case of schizophrenia in biblical times, as he experienced visions and received messages from God, adopted taboo rituals, and exhibited bizarre behavior. The visions or elaborate visual hallucinations included "four living creatures" each having "four faces and . . . four wings" and "the face of a man . . . or a lion . . . an ox and . . . an eagle . . ." These were followed by auditory command hallucinations: " . . . I do send thee unto them and thou shalt say unto them 'thus saith the Lord God . . . ' " and gustatory hallucinations: "Then did I eat [the roll of a book] and it was in my mouth as honey for sweetness."[36] However, it is not clear that Ezekiel actually adopted the taboo rituals and bizarre behavior that he records God demanding of him. Smith[35] includes a pertinent discussion of religious conversion and schizophrenia, demonstrating the difficulties of using such descriptions as evidence for schizophrenia. He states that the differences between the prophet and the schizophrenic depend on outcome and overall function and that Ezekiel was not schizophrenic because he was a successful and inspiring public figure. William James[37] makes a related point (which is also relevant to some of the other examples in this paper) in the first chapter of his *Varieties of Religious Experience*, that the spiritual significance of writings or visions or behavior is based on religious or moral values and not on the origin in psychopathology or nervous instability. Conversely, the religious importance or significance of, for example, visions or writings, does not by itself affect consideration of the psychopathology or diagnosis of the visionary or author.

It is not difficult to consider the "possessed" of the New Testament as having had schizophrenia, though it is difficult to prove it. The possessed are exemplified by the story of the Gadarene swine in which there was a

> certain man who had devils a long time, and wore no clothes, neither abode in any house, but in the tombs . . . and always, night and day, he was in the mountains and in the tombs crying, and cutting himself with stones . . . [He was] exceedingly fierce so that no man might pass that way . . . often times [the unclean spirit] had caught him; and he was kept bound with chains and in fetters and he broke the bonds and was driven of the devil into the wilderness. He said to Jesus: "I beseech thee torment me not," "My name is Legion for we are many" . . . and all the devils besought him saying "send us into the swine . . ."

The people were afraid later when they saw him behaving normally: "him that was possessed with the devil and had the legion, sitting clothed and in his right mind."[32(p314),38]

MANIA AND MELANCHOLIA

Classical and medieval medical literature can be considered together since the former was the basis and source for so much of the latter. A superficial view of this literature is deceptive as regards descriptions of schizophrenia because of the names used. *Mania* and *melancholia* are the most common, and since they have very specific meanings today, we tend to dismiss them from consideration in the history of schizophrenia. If one considers how the meaning and use of the word schizophrenia have varied and changed in different countries and in different parts of countries (for example, in the United States and Britain and within the United States[11]) it is easy to see how *mania* and *melancholia* could be used so differently over a period of 2,000 years and in many different countries and languages. Some terms appear to have had fairly consistent meanings and it is important to recognize the implications: *delirium* and *phrenitis* were always recognized as separate from *mania* and *melancholia*, as being accompanied by fever and as being "organic." By implication mania and melancholia were not considered "organic." The English word "madness" used in medical and philosophical writing before the last century sometimes included and sometimes excluded mania and melancholia.

Mania and melancholia were terms introduced in classical Greece, but they were causing problems by the time the Romans adopted them. Cicero (106–43 BC), not a physician but an astute observer, philosopher, and politician, made some chauvinistic comments and complaints about this nomenclature[39] which have a counterpart today in the distinctions between diagnoses of depression and mania in Britain and the United States.[11] By Cicero's time the terms furor, frenzy, and mania were used to mean the same thing, and Cicero, confessing that he could not readily give the origin of the Greek term mania, said: "Romans discriminate better between insania and furor (frenzy) than do the Greeks." He then complains that "what we call frenzy they call melancholia," and he cannot agree that the mind is influenced by black bile.[39] These terms, *mania* and *melancholia*, have continued to cause problems for the 2,000 years since Cicero wrote.

MELANCHOLIA BEFORE 1400

Symptoms of melancholia included delusions and hallucinations unrelated to depression. Galen (129–200 AD) included "fixed delusions, for example, thinking himself made of glass, believing himself to be without a head, believing he had been poisoned, thinking that by enchantment his enemies had caused demons to chase him."[40] Alexander of Tralles' (525–605 AD) description was like Galen's, with additional delusions such as "believing that one was a cock or a nightingale and trying to imitate its cry or song, believing that one was Atlas supporting the heavens and fearing that the burden would fall crushing oneself and the whole world," and "believing one had been beheaded for having been a tyrant" or "having animal delusions, or cheerful delusions, laughing without reason," "having sleepless anxiety, or being irritable, agitated and given to savage or furious attacks."[40] It was emphasized by Soranus (98–138 AD) that melancholia was a chronic disease, but sometimes with intervals of rationality and sometimes with continual madness according to Alexander. In the tenth century Avicenna (980–1037 AD) described nine types of melancholia. One was characterized by sadness leading to malicious behavior and agitation, by visual hallucinations of fire or the color red, auditory hallucinations of noises and trumpets, and disorder of thinking and judgment, with fear, violent anger, and fearful delusions of robbers, demons, and wolves.[40] Clearly melancholia was not just depression—it included a chronic illness with bizarre grandiose and persecutory delusions, visual and auditory hallucinations, thought disorder, and variable and inappropriate affect (laughing without reason), which could very well be schizophrenia.

Mania Before 1400

About mania, Soranus in the second century AD said it is

> a state of mental derangement with severe excitation and often wild behavior . . . a chronic disorder without fever, contrasted with phrenitis in these respects . . . it manifests itself now in anger now in merriment, now in sadness or futility and now . . . in an overwhelming fear of things which are quite harmless . . . it is sometimes continuous and at other times relieved by intervals of remission . . . Mental aberration is recognized by unusually quiet behavior or loud laughter, singing or sadness, muttering rage or fear. Hallucinations are stressed . . . talking to the dead, movement of the hands before the eyes . . . In the course of mania fever, which is not related to the cause of the illness may occur but in delirium fever comes first, in mania loss of reason is the first sign . . .[40]

The patients may express delusions of "being a sparrow, a cock, an earthen vessel, a god, an orator, an actor, a baby, or occupying the center of the universe."[41] Caelius Aurelianus, who translated Soranus into Latin, includes the above quotations as well as:

> Mania is an impairment of reason . . . The Greeks call it mania because it produces great mental anguish . . . ; or because there is excessive "relaxing" or "looseness" of the soul or mind . . . ; or because the disease defiles the patient . . . or because it makes the patient desirous of being alone and in solitude . . . or because the disease . . . is persistent; or because it makes the patient hard and enduring . . .[42]

These quotations show that while the classical concept of mania included the current concept of mania, it probably also included schizophrenia as evidenced by hallucinations, bizarre delusions, variable and probably inappropriate affect, thought disorder, social isolation, and chronicity. The exclusion of organic causes (especially with fever) is emphasized.

A 14th century description of mania and melancholia (a chapter in *Lilium Medicine* by Bernard de Gordon, quoted by Clarke[43]) presents the symptoms progressively as "occulta" (hidden or warning signs), "manifesta" (which refers mostly to speech indicators), and "completa," or overt derangement. Occult signs included imagining or thinking wrong thoughts, a lowered moral sense in discriminating about goodness and proper conduct, trying to grasp the impossible and irrational while showing poor judgment, alternating between laughter and tears, having disturbed sleep with terrible apparitions, and dreaming of black monks, hanged men, and the dead. "Manifest" or speech signs included talking to oneself, childish silliness, making remarks without head or tail, failure to finish sentences, and inability to explain them. "Complete" signs were erratic behavior and odd movements of limbs, tongue, and eyes, and wandering here and there like a cutubut or "water goat" (this is a surface insect—a pond skater—which dashes in one direction, stops before it arrives, and goes in another direction).[43] Here is evidence of formal thought disorder, visual and probably auditory hallucinations, labile and inappropriate affect, delusions, bizarre behavior, stereotypic movements or mannerisms, and deterioration in judgment, supporting the contention that schizophrenia is included in descriptions of mania and melancholia.

Mania and Melancholia After 1400

In the 16th century Paracelsus (1493–1541) spoke of mania as a condition with intermittent attacks and recovery of reason.[44(pp50–51)] "Melancholici," however, he described as one of the four kinds of insane people who "by their nature lose their reason and turn insane." The insane are permanently so: "without reason, till they die." The other kinds of insane people according to Paracelsus were lunatici (related to the moon), insani (from birth), and vesani (poisoned).[44] This demonstrates that for Paracelsus melancholia was very different in its course and outcome from mania and much more like schizophrenia in this respect than either mania or depression.

Medical writings of the 17th and 18th century record descriptions of both mania and melancholia with symptoms of schizophrenia. For example, Gluck in 1685[41(p61)] described excitement in which patients are noisy, aggressive, and

> do not take care of themselves, eat their feces, and rip their clothes. They are awake at night and in a continual turmoil of hallucinations (phantasms) and delusions, disturbed by ghosts and demons. The manics have this in common with the melancholics: wakefulness and a continual jumble of things in the imagination so that the delusions of mania are more external and of melancholia, internal. They exercise great strength and convince themselves they are driven by ghosts and that their insanity is caused by demons.[41]

Nicolaus[41] in 1692 wrote on mania:

> . . . There are persistent periods of wakefulness, terrible dreams, headaches, imbalanced laughter, irrational anger, continual worry, brightness and shiftiness of the eyes, frequent nocturnal pollutions, ringing in the ears, a quiet and pensive air as if in a state of stupor. There is a tendency to look down on the ground and there is fear and dread. The victims of this foretell the future. Under the force of the disease, they fall into various kinds of delirium, some laugh, some are angry, some are sad, some frightened by trivial things. All of them are rash, inclined to hurt their friends or themselves for they will injure or sometimes even kill their friends. They jump out of windows, tear their clothes, spit out their food, act ferociously and scowl.

Here again are the paranoid and bizarre delusions, hallucinations, psychomotor excitement, inappropriate affect, withdrawal and stupor, and grossly disorganized behavior of schizophrenia.

Another case recorded by Diethelm[41] was reported in 1741 by Muller, who described in detail a young man whose illness started with depression, went on to silence, then to catatonia for many months:

> his arm, statue like, kept for a long time the curvature into which someone had moved it. As he improved, his expression was no longer sad, but foolish or fretful with interruptions of smiles . . . Then three months smiling or drawing up his cheeks like a monkey and continuously drooling. A month later, there was insatiable hunger; he ate a bunch of fresh flowers and tallow candles when he had the chance. After another month he was in the same bad mood, stupid, dullness, drooling, but he would direct a fatuous smile or stiff or yokel's expression towards those who spoke with him. He then vomited, offered some obscure statement with a faltering tongue and a laugh like Heraclitus but blandly and then he suddenly became normal. He became fat, physically healthy and strong as a bull. His will, however, was

> wandering, inconstant and sick now, so that you would have called it a case of that mental lapse or wandering melancholy.[41(pp77-80)]

We might call it residual schizophrenia, after an illness with catatonic and hebephrenic features.

Several authors equate mania and melancholia with madness and give descriptions which again fit schizophrenia rather than affective disorder, and they attempt to exclude organic causes. For example, Barrough, in *A Methode of Phisicke*, published in 1583,[45(p27)] states:

> Of Madness: Mania in Grecke is a disease which the Latines do call Insania and furor. That is madness and furiousness. They that have this disease be wood and unruly like wild beasts. It differeth from the frenesie because in that there is a fever. But mania cometh without a fever . . . There goeth before madness debility of the head, tinckling of the ears and shinings come before the eyes, great watchings, thoughts and strange things approach his mind and heaviness with trembling of the head. If time proceeds, there is raised in them a ravenous appetite, and a readiness to bodily lust, the eyes waxe hollow and he do neither wincke nor becken. But madness caused of blood only, there followeth continual laughing, there cometh before the sight (as the sicke thinketh) things to laugh at. But when Choler is mixed with bloud, then the pricking and fervent moving in the braine maketh them ireful, moving angry and bold. But if the choler do waxe grosse and doth pricke and pull the brain and his other members, it make them wood wild and furious and therefore they are the worst to cure.[45]

This could be the mania of affective disorder, but the auditory and visual hallucinations and inappropriate laughter suggest schizophrenia.

If it is accepted that schizophrenia is included in descriptions of melancholia, one might conclude that the absence of schizophrenia[5] in Robert Burton's *Anatomy of Melancholia*[46] means that schizophrenia was absent or rare in 1620. However, Burton's melancholia according to his own definition is nearer modern depression, and he specifically excludes "madness" and so schizophrenia from his definition. (However, in his narrative he often uses his source's definitions, so his examples include some very bizarre and psychotic symptoms.) He lists and discusses "madness, phrenzy and melancholy" which were, he says:

> confounded by Celsus and many writers; others leave out phrenzy and made madness and melancholy but one disease—but most of our neotericks do handle them apart, whom I will follow in this treatise. Madness is therefore defined to be a vehement form of dotage, or raving without a fever, far more violent than melancholy, full of anger and clamor, horrible looks, actions, gestures, troubling the patients with far greater vehemency, both of body and mind, without all fear and sorrow, with such impetuous force and boldness that sometimes three or four men cannot hold them.[46]

Schizophrenia would surely be included in this description. Burton notes in the definition of melancholy that the humor was "depraved, to distinguish it from folly and madness in which those functions are not depraved, but rather abolished."[46] Although Burton chose not to write of "madness" in his treatise, the playwrights of that age wrote of it extensively (see below).

Richard Napier was a contemporary of Burton who did not use the term "mania," and whose concept of "melancholy," like Burton's, included delusions and hallucinations but excluded "madness" and "lunacy."[47(p150)] This is perhaps the distinction that DSM III[48] emphasizes between Major Depression with Psychosis and other Chronic Psychoses. Napier was a clergyman, physician, and astrologer whose patients' notes as described and interpreted by MacDonald[47] provide a wonderful insight into early 17th century views of and attitudes towards the mentally ill. About 2,000 of his 60,000 consultations over 40 years were with mentally disturbed patients.[47] Detailed descriptions of individual patients or their thought disorders or courses of illness are not included but the terms "light headed," "idle talk," and "distracted" probably described formal thought disorder.[47] "Mad" and "lunatic" are terms Napier and the relatives of his patients used for people described as follows: "Raving, seemingly incomprehensible speech,"[47(p124)] "frantic, talked too much, laughed oddly,"[47(p125)] "frantic energy, fits of wildly inappropriate laughter or rage . . . restless wandering or aimless running," "strange laughing and can give no reason," and "laugh and clap hands . . . will be sullen and mopish," "by fits will laugh . . . will cry out that she is damned," "sometimes laughing, sighing, then sad, fearful, rash, doting, crying out, threatening, skipping, leaping then serious, etc."[47(p139)] The impaired understanding of thought disorder, inappropriate affect, psychomotor restlessness,

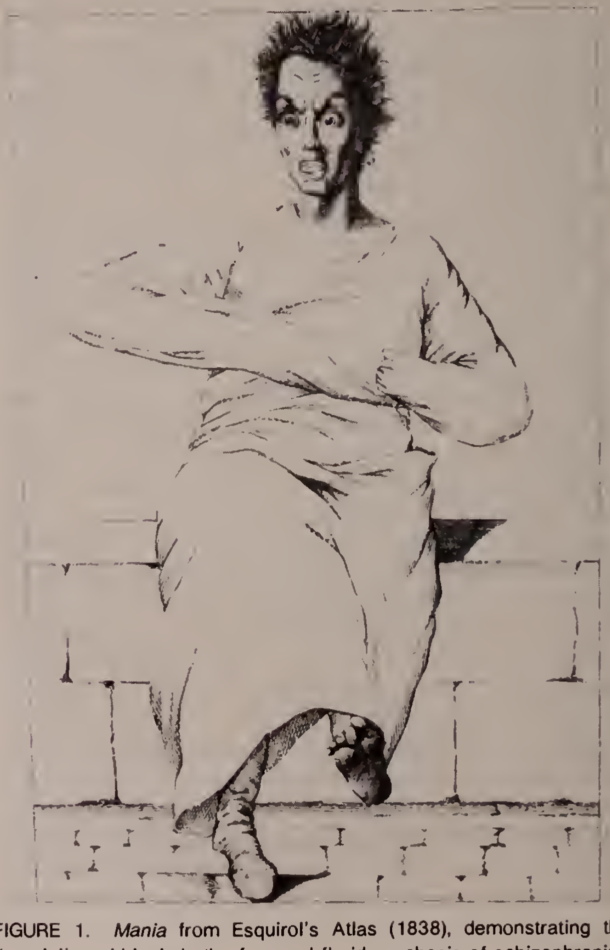

FIGURE 1. *Mania* from Esquirol's Atlas (1838), demonstrating that "mania" could include the fear and florid psychosis of schizophrenia.

and bizarre behavior suggest schizophrenia. About 270 of Napier's patients were described as "mad," "lunatic," or "distracted."[47]

Despite the narrower use of the term "melancholy" by Burton and Napier, this and "mania" have continued to be used by many authors to include conditions that could be schizophrenia. Gluck in 1685 and Muller in 1745 were two examples from Diethelm[41] quoted above. Burrows in 1828[27] talked of "demency" alternating with or following mania or melancholia. "Demency" is the "dementia" of Pinel[23(pp160-164)] and dementia precox (which is schizophrenia). Griesinger in 1845[26] divided thought disorder into two types: chronic mania with persisting hallucinations and delusions, and dementia without them (an early precursor of Crow I and Crow II schizophrenia).[49] This was 42 years after a very clear description by Beddoe in 1803[44(pp579-581)] of mania and melancholia as the two aspects of what is now called bipolar disorder. Beddoe castigated those who tried to separate mania and melancholia, supporting the view that they are not opposite diseases. Rather, the most furious manics may suddenly sink into profound melancholy, and patients whose lives are divided between furious and melancholic paroxysms under both states may retain the same set of ideas. Kraepelin restricted the term melancholia to depression in older people, and the term was resurrected in DSM-III[48] for a particular type of depression. It is only since Kraepelin's time that mania has had its current specific meaning, and that the use of the term melancholia has been restricted to depressive illness.

Possession

There were of course many conditions described by other names such as lycanthropy and erotomania. Some of these may have been schizophrenia. *Schizophrenia 1677*[50] is a fascinating account of a man with a severe psychotic illness who feels persecuted by the devil. He has persecutory delusions and auditory and visual hallucinations, bizarre behavior, and trances over the course of a year, and, for the next 20 years until he dies, has a slight relapse whenever he drinks alcohol. In the absence of descriptions of thought disorder and social or economic deterioration it is difficult to prove the diagnosis of schizophrenia. Furthermore, the authors of this book disparage diagnoses and do not explain or justify their title. Such "good prognosis schizophrenics" and patients who relapse on alcohol are not rare and their diagnosis is today sometimes hotly debated. "Possession" states in general today are often even more difficult to diagnose. The term describes the content of, and reaction to, the delusion whose cause may be schizophrenia or depression or hysteria.

Other Descriptions of Schizophrenia

MacDonald[47(p113)] states that since antiquity insanity has been defined by experts but discovered by laymen. So far we have quoted mainly the "experts," physicians and philosophers or theologians with a strong interest in mental disorder. Let us now see what laymen have described in the past.

Henry VI of England could perhaps have had schizophrenia. Much of the relevant data are described in detail by Clarke.[43(pp176-202)] Henry VI entered his teenage years healthy, precocious, and ambitious.[51(p12)] In his 20s he began to display excessive grandiosity in, for example, tearing down seven years of building at Eton College to ensure that his church there would be larger than any other; his other plans for the foundation of Kings College, Cambridge, were also too grandiose to complete—but the magnificent chapel was completed.[51(pp141-142)] He also was perhaps paranoid as evidenced by his vicious and excessive treatment of Eleanor and her husband, his own uncle and nearest relative, the loyal though sometimes critical "Good" Duke Humphrey of Gloucester, against whom he "fortified himself with armed guards," believing Humphrey wanted to kill him and seize the throne.[51(p129)] At this time people were being tried for calling him a "lunatic," "not steadfast of wit," a "fool," etc.[52(pp34-35)] At the age of 31, "he fell . . . into such a weak state of health that for a whole year and a half he had neither natural sense nor reason capable of carrying on the government; and neither physician nor medicine could cure that infirmity."[53(p31)] He did not speak despite all kinds of entreaties: . . . "the King gave no manner of answer . . . or countenance, saving only that once he looked on the Prince and cast down his eyes again."[53(p33)] Three months later a deputation of lords and bishops received absolutely no response in words or movements to repeated attempts to have him appoint a new chancellor—yet they had watched him dine.[51(p272)] He recovered, relapsed again into this state, recovered again, and was apparently physically fit. His body, when examined in 1912, was that of a fairly strong, middle-aged man,[43] but, according to contemporary descriptions, he had remained "inane,"[51(p332)] "a shadow on the wall, submissive and mute, like a crowned calf," a "mild-spoken, pious king but half-witted in affairs of state."[51(pp19,334)] Others in effect ruled until 1461 when he was 40 and Edward IV usurped the throne. From 1465 until 1471, Henry was kept in the Tower of London—well fed and well clothed but doing nothing—emerging in 1471 for a few months as king, "not so cleanly kept as befitted such a prince."[51(p338)] At the second battle of St Albans (1460) he was "placed under a tree a mile away where he laughed and sang."[43(p200)] He apparently "often saw the Lord Jesus presenting Himself in human form in the sacrament of the altar in the hands of the priest,"[54(p36)] and he was heard to tell of a "repeated revelation . . . three years running at that feast of St Edward . . . of the glory of the Lord appearing in human form . . . and of a vision of the assumption of the Blessed Mary both corporal and spiritual."[54(p42)] When he was a fugitive in 1464, "an audible voice sounded in his ears for some seventeen days before he was taken . . . " foretelling what would happen to him. "But when he told this to some of his people [his chaplains] they were incredulous and believed it not, but thought all to be vain wanderings until the event assured them of the truth.[54(p43)] These quotations, translated by James, are from Blacman who wrote to demonstrate the holiness of Henry at a time when visions were not considered a sign of mental illness.[55] (Joan of Arc, whose life was guided by "voices," had been found guilty by a French ecclesiastical court in Henry VI's reign, when he was ten.) However, surely the following description of preoccupation with hallucinatory voices would not have been made up to prove his piety:

> . . . many who yet survive and were once of his household say that he was wont almost at every moment to raise his eyes heavenward like a denizen of heaven or one rapt, being for the time not conscious of himself or of those about him, as if he were a man in a trance or on the verge of heaven, having his conversation in heaven, according to that word of the apostle, "our conversation is in heaven.[54(p38)]

The suggestions of psychopathology before his severe illness, the lack of physical symptoms or sequelae, the long duration of the catatonia, and his living 15 more years, weigh against its being an organic psychosis.[56] The lack of any mention of depression or mania, the apparently normal appetite, and, after 1455, a tendency to sleep too much[52] make an affective disorder less likely. Although prolonged catatonia can occur in affective disorders,[56,57] Henry VI's change of personality at around the age of 20 before the onset of catatonia, his auditory hallucinations, delusions, and inappropriate or flat affect (at St Albans and when catatonic), and the deterioration in his work and social relations suggest schizophrenia. One weakness of the case for schizophrenia is the source of information about the hallucinations—Blacman (about whose value as evidence Clarke[43(p195)] and Wolffe[51(p7)] disagree), because the purpose of his writing was to demonstrate Henry's piety.

There was a family history according to Clarke.[43(pp203-206)] Henry VI's great grandmother, Jeanne de Bourban, "lost her good sense and memory" for a few months at the age of 35. Her son, Henry VI's maternal grandfather Charles VI of France, appears to have had heatstroke during an episode of overactivity and wild behavior in 1392, and went on to become paranoid and have relapses of psychotic symptoms for the next 30 years, with listlessness and carelessness between relapses.[43(p203)] In 1393,

> he was saying that he had no wife and no children, that he was not king but a man called George, and he defaced the royal arms on vessels . . . He also asserted that he was made of glass . . . poor attention was noted in the short remission of 1397. Later he became quite indifferent to death among family and friends and fell into a listless state. By 1405 he was verminous as well as infected having dug a piece of iron into himself; he had to be forcibly washed and retrained to look after himself.[43(p205)]

However, although he did not appear to recover completely between episodes his condition was cyclical—with 44 episodes in 30 years according to Heim.[58(p120)] As Clarke [43(p205)] notes, "the case brings out the difficulty of understanding now, a multiply determined and extended disorder of the fourteenth century, but the veil is not quite opaque and it is possible to feel that Charles' condition is not of a different order from those of modern times." Schizo-affective disorder appears to be an appropriate modern diagnosis.

SHAKESPEARE

If kings had schizophrenia one would have expected Shakespeare (1564–1616) to have described it. Torrey[5(p25)] states that there are no descriptions of schizophrenia in Shakespeare's writings and uses that as evidence for the absence of the condition at the time. However, the theme and content of literature and art are dependent upon the fashions, attitudes, and philosophy of the time and only with care can they be used to draw conclusions about illnesses at the time. There was, for example, a remarkable change in the treatment of madness by playwrights at the end of Shakespeare's life: the Jacobean stage was full of people who could have had schizophrenia. This change in literary description was too rapid to reflect a real change in the prevalence of mad people.

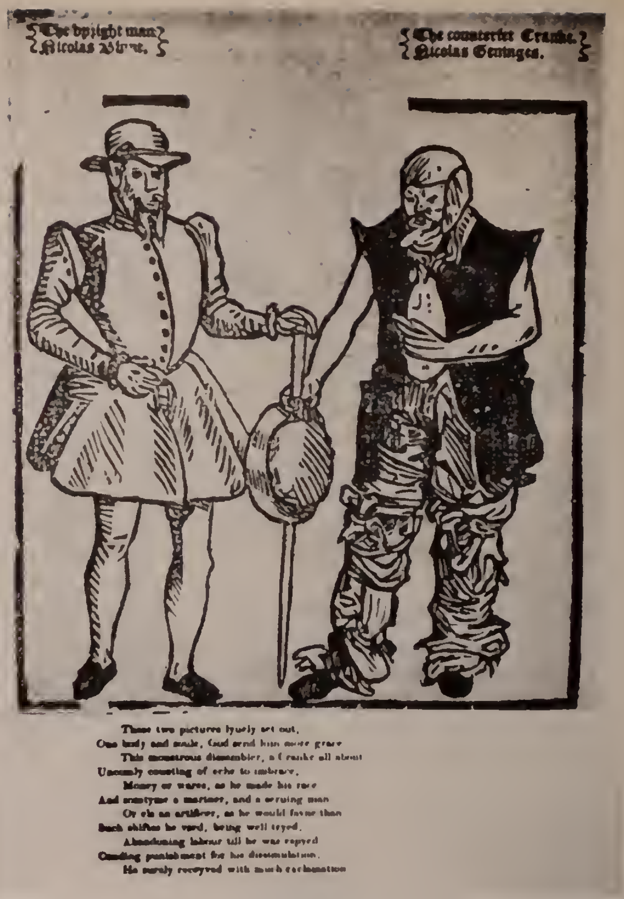
The vpright man
Nicolas Blunt.
The counterfet Cranke.
Nicolas Genings.

FIGURE 2. "Harmon's Rogue—Nicholas Blunt alias Nicholas Gennings as Upright Man and Counterfeit Cranke," from *The Groundwork of Conny-Catching* (anonymous), 1592.

But Shakespeare did know madness and describes chronic schizophrenia, as I have shown,[59] in the guise of Poor Mad Tom whom Edgar becomes in *King Lear*. He simulated a condition that came on in early adulthood, caused a lowering of socioeconomic status and deterioration in self-care, and was accompanied by delusions, hallucinations, and formal thought disorder. Surely this condition was schizophrenia, and it must have been not only familiar but common enough for an audience to accept that someone could conceal himself by feigning it. Such "bedlamites" and charlatans pretending to be mad beggars were numerous in the 16th and 17th centuries.[43(pp237-240)]

OTHER LITERATURE

That the depiction of madness in literature has more to do with "fashion" than with the prevalence of mental illness is amply illustrated by the contrast mentioned earlier between Elizabethan dramatists and the Jacobeans as described by Reed.[60] Elizabethan mad folk were, he says, capable of imagining superhuman achievements and in this sense were consistent with the Elizabethan superconfidence in the high destiny of man.[60] Formal thought disorder and inappropriate affect were not consistent with this image. After 1600 this confidence was lost and the madman on the stage expressed the frustration of mankind; he testified to the folly and impotence of human effort; he rarely recovered and nearly always exhibited "melancholy or choler."[60(p5)] It did not matter that his reason or emotion had broken down, for his purpose on the Jacobean stage was pure show: to provide a cheap laugh or diversion and to be the instrument or butt of satire. Jacobean plays by Dekker, Webster, Fletcher, Jonson, and others are full of such madmen. They incidentally show that authors and audiences were well acquainted with the Bethlem Hospital and its patients and the abuses and scandals going on there.[60] It was at that time one of the popular sights in London. The "madmen" in these plays have delusions of jealousy, of grandeur, and of persecution amd they have visual hallucinations, loose associations, or drivelling speech. For example, in *The Witch of Edmonton* by Dekker a poor girl is "tuned out of her wits" and blabs idiotic snatches, "See see see! The man in the moon has built a new windmill . . . Prither let me scratch thy face." After being touched by the incarnation of the devil in a dog, she becomes maniacal, runs off stage, and dashes out her brains.[60(p55)] There is much exaggeration for theatrical effect but there is also abundant evidence of the playwrights' familiarity with chronic psychotic illness. However, though many of these characters could have had schizophrenia the descriptions are too brief and incomplete to make the diagnosis certain.

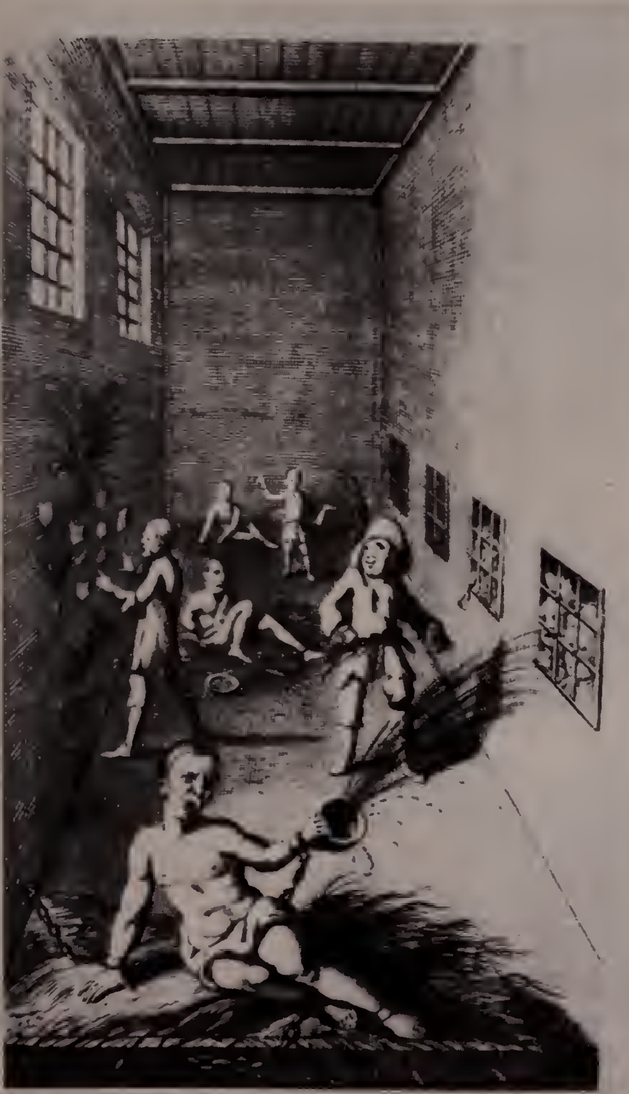
FIGURE 3. Bedlam, in Jonathan Swift's *Tale of a Tub*, 1710.

It was noted earlier that Burton excluded madness, and so schizophrenia, from *Anatomy of Melancholia*.[46] I speculate that Burton's reasons for excluding "madness" from his book are somewhat similar to the reasons (mentioned above) for the differences between Shakespearean and Jacobean treatment of madness. Unlike depression, madness and schizophrenia are not part of the normal experiences of all human beings; they are not the result of nor are they related to the great and universal emotions and conflicts of mankind—love and hate, jealousy and revenge, ambition and humiliation, separation and union, which are the essence of Shakespeare's plays. Such concepts and persistent emotions are hardly possible in someone with schizophrenia. Whereas the melancholic can add to the understanding of human nature and in particular the expression of grief, madness and schizophrenia are outside the normal understanding of human nature. They are expressions of the failure of human nature—by definition the breakdown of reason and emotion. For this reason people with these conditions cannot easily play the dramatic roles required in literature or theater—embodiments of emotion or conflict. The theatrical or real embodiments of evil (like Hitler) may be "mad" in another popular sense, in that their acts are terrible or hard to understand—but such people are not totally disorganized in thought or emotion. If they were their literary role would have no meaning, and they would not have been able to achieve what they did (in reality or in literature). Such people are also the mainstay of classical Greek literature, as studied, for example, by Simon.[61] Study of such literary characters sheds no light on the history of schizophrenia. However, the truly mad who are depicted on the stage, as in Jacobean times, not for their literary role but for diversion or often cruel amusement, or occasionally pathos, can sometimes tell us what their contemporaries knew of madness and schizophrenia.

THE QUESTION OF ORGANICITY

I have mentioned many descriptions of illnesses that could be schizophrenia. Torrey,[5(p21)] to support his thesis, suggests that those who were psychotic in earlier ages may have had "organic" conditions and not schizophrenia. "Organic" is used in the traditional sense of indicating gross brain or metabolic pathology. As some of the examples have shown, many earlier writers were concerned with this subject. The Ayurveda[29] described three types of "organic" psychoses distinct from the seven psychoses that could be schizophrenia. Many of the examples quoted so far show that mania, melancholia, and some forms of madness were specifically defined as not being accompanied by fever, with the implication, and sometimes the definitive statement, of a lack of "organic" cause. Hartley[44(p381)] states it more clearly: "The causes of madness are of two kinds, bodily and mental. That which arises from bodily causes is nearly related to drunkenness, and to the deliriums attending distemper. That from mental causes is the same kind with temporary alienation of the mind during violent passions . . . " In fact it had been spelled out 1700 years earlier by Aretaeus the Cappadocian.[39(p719)] "Insanity . . . is a chronic derangement of the mind without fever and it is thus distinct from the deliriums of drunkenness and from mental disturbance caused by certain edibles such as mandragora and hyoscyamus." Further, "the insanity of which we are speaking has no resemblance to the dotage which is the calamity of old age; this never remits and it accompanies the patient to the grave whereas insanity intermits and with care ceases altogether."[39] Such concern with organic causes weighs against all psychoses before 1800 being "organic." One could argure, rather, that some chronic psychoses ascribed to "organic" causes in the past were not so in fact for the poisons, fruits, and edibles thought to be causative rarely cause chronic conditions.

Hare, in his article on the origin and spread of dementia paralytica,[62] casts an interesting light on the theory that earlier mental psychosis may have been mainly "organic." He is discussing the fact that the association between insanity, paralysis, and the postmortem brain change of dementia paralytica had not been noticed before 1822 as evidence that dementia paralytica had not existed before then. He indicates that postmortem examinations of the mentally ill were frequent at the end of the 18th century and early 19th century. He states that it had long been known that paralysis was the result of brain change "as Willis's cases sufficiently illustrate." There were minor changes in the vessels and membranes and increased fluid was found by Chiariugi and Greding in the brains of some insane patients but not of others. The general opinion was that these changes were not due to the mental disorder but were secondary. Pinel "seemed to give up hope of elucidating the pathology of mental derangement by necroscopical research."[62] Hare quotes Esquirol in 1814: "the opening of the body teaches us nothing with regard to [the seat of dementia] and all the organic alteration of the brain belongs less to insanity than to its complications . . . " Hare says, "such a vein is understandable if organic causes of mental disorder were rare or associated only with simple dementia . . . "[62] We may conclude that psychosis was not usually caused by "organic" brain conditions at that time.

CHRONICITY

Somewhat related to this argument that earlier psychoses were due to organic conditions is the question of chronicity. "Organic" conditions in general have a much shorter life expectancy and so, to some extent, long lasting illness argues against organicity. Torrey[5(p23)] argues that what is lacking in earlier ages are descriptions with the initial breakdown in early adulthood, the constellation of symptoms, and the *chronic* course of schizophrenia. However, in addition to many of the examples already given there is ample evidence of numbers of chronically mentally ill people in all ages. Laws relating to mental illness provide some such evidence, for if the conditions were not long lasting special laws would not be required. In the Mishnah of the Talmud,[32(pp315-316)] there were a large number of laws concerning "imbeciles" which from the descriptions clearly include the mentally ill. In classical Greek times Plato (427–348 BC) demonstrated in his laws the chronicity of mental derangement: "If the father is then found to be insane he shall be treated as a minor for the rest of his life."[39] Evidence of chronic mental illness is found in two Irish law tracts not later than the eighth century, covering long term care, maintenance, compensation, and divorce.[43(pp31-34)]

In England the "Laws of Henry I" covered responsibility for the care of the insane.[43(p60)] Allderidge[63] (1979) demonstrates that the "mad" did require long term care and support, with common law in the 13th century stipulating that the parish was to provide for this or to pay for it. This case law was put into written form in the Poor Law of 1601, under which those "disabled in wit or member" could be kept in their own home or taken to the prison or boarded out by court order or at parish expense. The care of the insane was clearly both costly and lengthy from early times, and it was not hospital based.[63] In addition to those kept at home many were helped by the church, as demonstrated by the effects of the suppression of the monasteries in the reign of Henry VIII, which reduced the "sick, lame and impotent poor . . . to great distress" according to a petition by the City of London to regain control of Bethlem Hospital in 1538.[60(p16)] The Bethlem Hospital had been founded in 1247, was a hospital from 1329, and by 1403 housed six deranged men.[60(p15)] In the mid-16th century it was a place where people came to be "restoryde into hyr witte and helth a-gayne," but "sum ben abyding there yn for evyr . . . uncurerabylle into man."[63] In 1598 there were 21 insane persons in residence who had been there for from one month to 25 years. By 1674, patients were being discharged back to their own parishes if they had not been cured after 12 months. Guys Hospital was founded in 1728 with 20 beds for the incurably insane, and a few years later, in 1733, Bethlem set aside 110 beds for incurables.[63] Thus laws and services as well as literature show that chronic mental illness was not rare before 1800.

CONCLUSION

I have demonstrated that schizophrenia was described for 2000 or more years before the 19th century and was common enough to be described not only by physicians but by philosophers, clergy, authors, and playwrights, and to be recognized by their readers and audiences. I have shown that the descriptions were often included under the terms *mania* and *melancholia*. Many of the descriptions are admittedly incomplete, but in view of the evidence here it is incumbent on those who deny the earlier existence of schizophrenia to demonstrate that these incomplete descriptions are not of schizophrenia. Chronic mental illness was common enough for there to be specific laws and services covering it, and it was not usually caused by gross brain pathology or metabolic or toxic conditions. There is thus no reason not to extrapolate back from today's estimates of schizophrenia as comprising 20–50% of the chronically mentally ill.[64] The continuing debate about the rise in number of the hospitalized mentally ill in the last century[4,5(pp31-44)] should not obscure this conclusion that chronic mental illness and probably schizophrenia were not rare before 1800. Nor should the difficulties of assessing numbers and rates in earlier times[47(p148),5(pp61-64)] inhibit attempts to look more closely at earlier times and places, using original sources such as those used by MacDonald[47] and Neugebauer,[65,66] to try to identify the rates of specific disorders.

The conclusion that schizophrenia was not rare before 1800 does not affect, for example, the viral theory of schizophrenia.[5] The historical evidence is largely irrelevant to the theory (rather, it has been suggested that the chronic infectious diseases are historically unrelated to "civilization" or population density).[67] It may affect some of the earlier theories relating schizophrenia to industrialization or other social changes of the past 200 years. It does emphasize that we must first be certain of the facts that our theories explain. That schizophrenia has always existed may support the view of schizophrenia as a syndrome with many causes, of which the historical variation of any one may be masked by the constancy of others. Perhaps in the future, with new laboratory and clinical methods, the syndrome of schizophrenia will be subdivided into circumscribed conditions with known causes and then we can go back over the historical data to look for variations over time of these specific conditions.

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Benign epithelial inclusion cyst in an axillary lymph node

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Benign heterotopic epithelial inclusions in axillary lymph nodes are rare phenomena. Only a few reports have appeared in the English language literature. Most of the cases have occurred in patients with benign breast diseases. This report describes a benign epithelial inclusion cyst with concurrent carcinoma of the breast.

Case Report

A 51-year-old woman had been followed at the breast clinic since a papillary adenoma of the right nipple had been diagnosed a year previously. A routine mammogram showed an area of microcalcification in the right breast. After radiologic localization, an excision biopsy was performed which revealed intraductal carcinoma with small foci of infiltration and extensive papillomatosis. A right modified radical mastectomy was performed 36 days later. On dissection, one of the axillary lymph nodes appeared to be involved by metastasis.

Examination of the mastectomy specimen showed extensive traumatic fat necrosis and foreign body reaction to suture material. Remnants of intraductal papillomatosis were also present. There was no residual ductal carcinoma. Twelve lymph nodes were found in the axillary content which ranged from 0.5 to 3 cm in size. The largest node in the upper level measured 3 × 2 × 1 cm. On sectioning, it was grayish white and appeared to be necrotic. Microscopic examination of this node disclosed a cyst lined with stratified squamous epithelium (Fig 1). The epithelium was focally flattened, and the lumen contained eosinophilic amorphous material. The epithelium with granular cells indicated epidermal differentiation (Fig 2). No adnexal elements such as sweat and apocrine derivatives were present. The cyst was completely surrounded by lymphoid tissue. The rest of the node showed features of sinus histiocytosis.

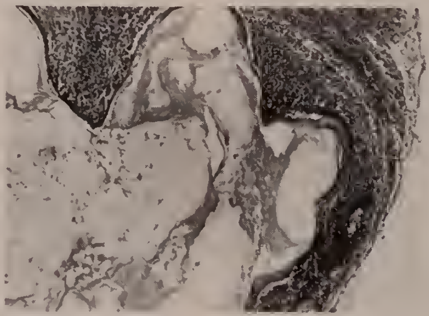

FIGURE 1. Axillary lymph node showing epithelial inclusion cyst (hematoxylin-eosin stain; original magnification × 40).

Discussion

Benign epithelial inclusions in axillary lymph nodes were first reported in the English language literature by Garret and Ada[1] in 1957. The authors described cysts lined with stratified squamous epithelium and numerous duct-like structures in an axillary lymph node. The ipsilateral breast showed sclerosing adenosis and intraductal hyperplasia. McDivitt et al[2] described two instances of breast duct-like structures associated with epidermidization and cyst formation. Haagensen[3] mentioned apocrine cysts and keratin-filled cysts in axillary lymph nodes removed because they presented as a palpable firm mass. Osmond and Kalinovsky[4] reported a case of axillary malignant tumor associated with benign inclusion cysts in lymph nodes. However, histologic features of the inclusions were not stated in detail. Edlow and Carter[5] reported a variety of epithelial structures in several axillary lymph nodes removed incidentally as a part of bilateral total subcutaneous mastectomies for chronic cystic disease.

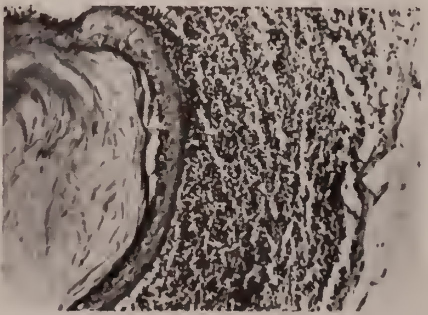

FIGURE 2. Cyst wall with squamous lining. Granular layer and lamellated keratin are present (hematoxylin-eosin stain; original magnification × 100).

The case reported here deals with the occurrence of a benign cyst lined by stratified squamous epithelium in an axillary lymph node removed for infiltrating ductal carcinoma of the ipsilateral breast. Unlike previous reports, this case did not demonstrate other structures or different types of epithelium indicating the origin of the cyst. The absence of adnexal elements suggests that this is not a true dermoid cyst. The histogenesis of such benign inclusion cysts is uncertain. Most authors have suggested that they are of mammary origin.[1,2,5] They have found structures resembling breast ducts or apocrine-type epithelium and squamous lining which were regarded as metaplasia.[2,3,5] Also, there have been several reports of benign breast tissue being found in axillary lymph nodes.[2,6,7]

The mechanism of existence of benign heterotopic tissue in lymph nodes is controversial. The explanation appears to be different depending on the types of epithelial structures included in lymph nodes.

Embryologic displacement theory has been proposed for the salivary tissue in periparotid lymph nodes[8] and nevus cell aggregates in lymph nodes at various locations.[9] The idea of benign metastasis has been suggested for the presence of normal thyroid follicles in the cervical lymph nodes[10] and endometrial inclusions in lymph nodes around the female genitals.[11] However,

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Farhi and Silverberg[12] postulated that the glandular inclusions in pelvic and abdominal lymph nodes are derived from metaplasia of coelomic epithelium trapped in lymph nodes.

In the case described here, we cannot be certain of the histogenesis of the lesion. The epithelial structures may have been transported to the axillary lymph node during the previous biopsy, or they could have been trapped in the node during embryonic development. The significance of benign epithelial inclusions in axillary lymph nodes is that they may simulate malignant metastasis clinically or on frozen section, especially when they are accompanied by benign proliferative lesions of the breast.

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Acute rheumatic fever in New York City

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During the past 30 years, the incidence of acute rheumatic fever has fallen dramatically in the United States.[1-4] In addition, the disease seems to have changed its clinical profile and appears to manifest a less florid course. Recently, however, there have been reports of outbreaks of acute rheumatic fever in the intermountain area centered in Salt Lake City, Utah,[5] Pittsburgh, Pennsylvania,[6] Columbus, Ohio,[7] and Northeast Ohio.[8] These outbreaks suggest that a resurgence of acute rheumatic fever may be occurring throughout the United States. At Beth Israel Medical Center, a 916-bed university-affiliated teaching hospital which serves the population of the lower east side of Manhattan in New York City, a similar increase in the incidence of acute rheumatic fever was noted during the time period January through June 1985. A diagnosis of rheumatic fever was made in six patients during this six-month period. In the ten-year period from 1975 to 1985, approximately one case of rheumatic fever per year was seen.[9] Although this sample is smaller than those reported in other series, it represents a real increase in incidence of acute rheumatic fever in the population in question. Each of the six patients with the diagnosis of acute rheumatic fever had a complete history and physical examination. Laboratory tests done on each patient included a throat culture, complete blood count, erythrocyte sedimentation rate (ESR), electrocardiogram, chest film, streptozyme titer, urinalysis, and M-mode and two-dimensional echocardiogram. Doppler echocardiography was also done on each patient.

CASE REPORTS

Case 1. A seven-year-old girl was admitted to the hospital because of pain and swelling in both knees. She had been well until ten days prior to admission, when pharyngitis developed, and she was treated for five days with oral penicillin. Auscultation of the heart revealed a grade 3/6 blowing, systolic ejection murmur at the apex. The ESR was 130 mm/h. The creatine phosphate (CrP) was positive. The streptozyme titer was 1:800. The electrocardiogram, chest film, and M-mode and two-dimensional echocardiogram were within normal limits. Doppler echocardiography showed mitral regurgitation. The patient was treated with aspirin and benzathine penicillin.

Case 2. A ten-year-old girl was admitted to the hospital with a chief complaint of right wrist pain and pain in both hips of three days duration. There was no history of recent upper respiratory infection. Auscultation of the heart revealed a grade 2/6 holosystolic murmur at the apex. Physical examination of the extremities revealed marked limitation of range of motion of both hips. The ESR was 130 mm/h, an antistreptolysin 0 titer was 1,250 Todd units, and the streptozyme titer was 1:600. An electrocardiogram, chest film, and M-mode and two-dimensional echocardiogram were normal. Doppler echocardiography showed mitral regurgitation. The patient was treated with aspirin and benzathine penicillin.

Case 3. A 12-year-old boy was admitted to the hospital because of swelling of the left knee and left ankle. He had had a first attack of acute rheumatic fever with carditis four years prior to admission. Two weeks prior to admission he had had pharyngitis but received no treatment for this. Cardiac auscultation revealed a grade 2/6 systolic ejection murmur at the lower left sternal border and a grade 1/6 basal diastolic murmur. The ESR was 127 mm/h, and the streptozyme titer was 1:600. The electrocardiogram and chest film were normal. An echocardiogram showed dilation of the left atrium and left ventricle, and Doppler echocardiography showed mitral regurgitation and aortic regurgitation.

Case 4. A 9½-year-old boy was admitted to the hospital because of pain and swelling of the left ankle. He had been well until two weeks prior to admission, when he complained of a sore throat. A throat culture was reported as negative, and he received no treatment. The patient's sister had had acute rheumatic fever five years earlier. On

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TABLE I. Summary of Clinical and Laboratory Findings in Six Patients with Acute Rheumatic Fever, January–June, 1985*

Case No.	Age (yr)	Throat Culture	History of URI/P	Polyarthritis	Carditis	Chorea	Subcutaneous Nodules	Erythema Margination	Streptozyme Titer	ESR (mm/hr)	ECG	Fever	Arthritis
1	7	Negative	Pharyngitis	Yes	MR	No	No	No	Positive	130	Normal	Yes	Yes
2	10	Negative	None	Yes	MR	No	No	No	Positive	130	Normal	Yes	Yes
3	12	Negative	Pharyngitis	Yes	MR, AR	No	No	No	Positive	127	Normal	Yes	Yes
4	9½	Negative	Pharyngitis	Yes	MR, AR	No	No	No	Positive	60	Normal	Yes	Yes
5	8	Negative	None	Yes	MR	No	No	No	Positive	120	Normal	Yes	Yes
6	11	Negative	Pharyngitis	No	MR	Yes	No	No	Negative	Normal	Normal	No	No

* Abbreviations: URI, upper respiratory infection; P, pharyngitis; ESR, erythrocyte sedimentation rate; ECG, electrocardiogram; MR, mitral regurgitation; AR, aortic regurgitation.

physical examination, a grade 2/6 systolic ejection murmur was noted along the left sternal border. The left ankle was swollen and tender. The ESR was 60 mm/h, the CrP was positive, and the streptozyme titer was 1:400. Doppler echocardiography showed mitral regurgitation and aortic regurgitation. Pain and swelling in the right ankle subsequently developed, and the patient was placed on aspirin and benzathine penicillin.

Case 5. An eight-year-old girl was admitted to the hospital with pain in her right hip. A diagnosis of septic hip was initially made, and she was treated with intravenous antibiotics. Subsequently, pain developed in her right knee, and a heart murmur was noted. The streptozyme titer was 1:600. Auscultation of the heart revealed a grade 2/6 blowing, holosystolic murmur at the apex. Doppler echocardiography showed mitral regurgitation. She was treated with aspirin and benzathine penicillin.

Case 6. An 11-year-old girl was seen in the pediatric clinic with purposeless, jerky movements and emotional lability of one week's duration. She had had a sore throat four months before the onset of these choreiform movements. A diagnosis of Sydenham chorea was made. Cardiac auscultation revealed a grade 2/6 systolic ejection murmur at the lower left sternal border. The ESR and the streptozyme titer were within normal limits. An electrocardiogram, chest film, and M-mode and two-dimensional echocardiogram were normal, but Doppler echocardiography showed mitral regurgitation. The choreiform movements slowly subsided over the course of one month.

DISCUSSION

All six patients described above met the revised Jones criteria for the diagnosis of acute rheumatic fever. They were all found to have carditis by auscultation and/or by Doppler ultrasound examination. One patient had Sydenham chorea in combination with carditis, and five patients had migratory polyarthritis. These children ranged in age from seven years to 12 years and were all from lower middle class families with good access to medical care.

What accounts for this increased incidence of acute rheumatic fever across the country? It might be due to an increase in the virulence of the streptococcus over time. It might be due to a change in the prevalence of so-called rheumatogenic types of streptococci in communities. Or, it might reflect a change in the host, with genetic alterations in the human population. Gordis[1] suggested that streptococcal infections may occur in a cyclical pattern of increments and decrements, and that if such a cyclic phenomenon exists, there could conceivably be another rise in streptococcal infections and a concomitant increased incidence of acute rheumatic fever. Socioeconomic factors have been known to contribute to the spread of acute rheumatic fever; the disease has been found to be prevalent in areas with overcrowding, poor housing, and lack of access to good medical care. While all the patients described here were from poor families who received financial assistance for medical care, the majority of patients in other recent outbreaks of acute rheumatic fever[5–8] were from middle class populations with good access to medical care. Therefore, socioeconomic factors do not necessarily provide an explanation for the resurgence. Particularly virulent mucoid strains of Group A streptococci have been obtained from throat cultures in children,[5,7] strains that had not previously been seen with great frequency. However, it cannot be determined if these strains are responsible for the resurgence, since the cultures were not taken from children with acute rheumatic fever.

Three of the patients presented here gave a history of having had sore throats which were never treated. One patient had pharyngitis which was inadequately treated. Two patients did not recall having had a sore throat within the three-month period preceding the onset of acute rheumatic fever. Since four of these patients were not treated or were inadequately treated for sore throat, it appears that increased efforts on the part of physicians aimed at the diagnosis and treatment of pharyngitis might be indicated at this time, as well as heightened awareness by parents of the complications of Group A beta hemolytic streptococci.

Forty years ago, there was a plethora of rheumatic fever control programs designed to educate parents and physicians that rheumatic fever could be prevented by treating Group A streptococcal pharyngitis promptly with penicillin. If this explained the decline in the incidence of acute rheumatic fever, perhaps the time has come again to evaluate the need for the establishment of public health programs designed to prevent this disease.

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See also pp 352–353.

Elevated urinary specific gravity in acute oliguric renal failure due to hetastarch administration

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Specific gravity is the common, rapid, and inexpensive measure of urinary concentrating ability used in clinical medicine. Under usual conditions, the specific gravity of the urine and urinary osmolality have a fairly constant relationship.[1] Urinary concentrating ability is frequently used to assess the differential diagnoses of acute oliguric states.[2,3]

As specific gravity is a comparison of weights, it is related to, but not an exact measurement of, the number of particles in solution. The true measure of concentration is the number of particles in solution, which is measured by the osmolality.

If high molecular weight compounds are present in the urine, the specific gravity can be elevated out of proportion to the actual concentration of the urine. Thus, a discrepancy between specific gravity and urinary osmolality will result. A number of substances, both endogenously produced, such as glucose and protein, and exogenously administered, such as intravenous radiographic contrast material, dextrans, and carbenicillin, may significantly raise the urinary specific gravity.[4,5] The osmolality, on the other hand, is affected to a much lesser degree by the presence of these high density compounds in the urine.

We report here on another substance, hetastarch (hydroxyethyl starch), which can cause a discrepancy between urinary specific gravity and osmolality, and which may then cause confusion in the evaluation of a patient with oliguria.

CASE REPORTS

Case 1. A 54-year-old man suffering from alcoholism was admitted following a grand mal seizure. On physical examination he was obtunded. His temperature was 103°F; blood pressure, 170/90 mm Hg; pulse, 120 beats/min. Examination of the lungs revealed bilateral rales and rhonchi. Pertinent laboratory values included a creatinine level of 0.7 mg/dL and a white blood cell count of 14,200/mm.[3] A chest film showed a right lower lobe infiltrate.

A diagnosis of aspiration penumonia was made, and antibiotic therapy was begun with aqueous penicillin G, 2 million units every four hours, and gentamicin, 100 mg every eight hours. Subsequent blood and sputum cultures grew *Klebsiella pneumoniae*, and cefamandole was substituted for penicillin. The patient's clinical course was complicated by progressive respiratory failure, and he needed mechanical ventilation. Upper gastrointestinal bleeding with hypotension ensued, and the patient was given 1 liter of hetastarch for volume expansion. Oliguria and progressive azotemia developed. Specific gravity of the urine obtained approximately four hours after hetastarch administration was 1.035. A diagnosis of prerenal azotemia due to volume loss was made. The patient continued to bleed and was given packed cells, saline, and intermittent infusions of hetastarch (total 2,500 mL) during the next 24 hours. Three samples of urine obtained from the patient during this time showed specific gravity values of between 1.030 and 1.035. Essentially identical readings were obtained by urinometer and refractometer. Despite blood and volume expansion, the patient's condition continued to deteriorate, and he died the following day. Determinations of urinary osmolality, by freezing point depression method, done on the same samples revealed values of 313–350 mosm/kg H_2O (Table I).

The values of urinary osmolality are more consistent with a diagnosis of acute tubular necrosis (ATN) than prerenal azotemia.

Case 2. A 78-year-old man was admitted to the emergency room suffering from hypotension and an obtunded affect. On physical examination, absent bowel sounds and a rigid abdomen were found. The patient was started on gentamicin and cefoxitin, and an exploratory laparotomy was performed, which revealed a perforated colon secondary to a colonic neoplasm. Hetastarch and intravenous fluids (Ringer's lactate and saline) were administered before and during surgery. A total of 2 liters of hetastarch was given during the first 24 hours of admission. Oliguria and progressive azotemia ensued, and the patient died after 36 hours in the hospital. Urinary specific gravities on three samples of urine obtained after surgery were 1.015, 1.018, and 1.020. Concomitant measures of urinary osmolality on the same specimens were 261, 262, and 289 mosm/L, respectively. Urinary protein by dipstick was 2+. The urinary osmolality values are more consistent with a diagnosis of ATN secondary to hypotension rather than prerenal azotemia.

In Vitro Study. Serial additions of a 6% hetastarch solution in 0.9% NaCl (calculated osmolality approximately 310)[6] were added to distilled water to create solutions of varying specific gravity. Osmolality was determined on the same samples by the freezing point depression method. The results are shown in Table II.

One of the authors (LH) was given 300 cc

TABLE I. Specific Gravity and Urinary Osmolality Determinations After Hetastarch Administration in Case 1

Time Interval	Specific Gravity*	Urinary Osmolality**	Dipstick Proteinuria
4 hrs after hetastarch	1.035	350	2+
8 hrs	1.035	325	3+
12 hrs	1.030	313	not determined
24 hrs	1.035	320	not determined

* By urinometer and refractometer.
**Advanced Osmometer, Needham, MA.

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TABLE II. In Vitro Study Comparing Specific Gravity and Osmolality

Specific Gravity (Refractometer)	Osmolality (mosm/kg H_2O)
1.005	253
1.011	279
1.015	284
1.020	294
1.025	312
1.028	330

of hetastarch 0.9% saline intravenously. This subject had normal renal function and no proteinuria by dipstick determination. Concomitant determinations of urinary specific gravity and osmolality were made. In this subject with normal renal function and no proteinuria, there was no discrepancy between multiple specific gravity and osmolality determinations made during and up to 24 hours after infusion of a 6% hetastarch solution.

DISCUSSION

Hetastarch or hydroxyethyl starch is an artificial colloid which is administered intravenously for use as a plasma expander. Chemically, it is derived by introducing hydroxyethyl groups on to the glucose units of the starch molecules. This material is then subjected to acid hydrolysis, which results in a product with an average molecular weight of 450,000 daltons (range, 10^4–10^6).[6] How the substance is metabolized is complex and is related to the difference in size of the particles contained in the solution. It is believed that the smaller molecules, those with a molecular weight of less than 50,000 daltons, are excreted unchanged by glomerular filtration. Some larger molecules are rapidly metabolized to smaller molecules, while the largest molecules are distributed to various body tissues and may undergo hydrolysis by the reticuloendothelial system or enzymatic degradation by amylases, followed then by urinary excretion.[6,7]

Smaller molecules of hetastarch demonstrate similar urinary excretion patterns, as do other artificial colloid solutions such as dextran 40 and dextran 70. Molecules of hetastarch, however, tend to be of higher average molecular weight than the dextran solutions available for clinical use. Dextran 70, for instance, has an average molecular weight of approximately 70,000 daltons, while dextran 40 has an average molecular weight of 40,000 daltons, and thus they are generally cleared more rapidly by glomerular filtration.[8]

As urinary specific gravity is a comparison of weights, it is related to, but not an exact reflection of, the urinary osmolality, which depends on the number of particles in solution.[1,4] The specific gravity is affected by a variety of substances, which if they are high density molecules, will elevate the value, while the osmolality will be little, if at all, changed by their presence. This is seen with urinary excretion of radiographic contrast material, for instance, which can elevate the specific gravity to >1.040, which is considered to be above the level of maximal urinary concentrating ability.[1] The same is true for dextran solutions, which are relatively large molecular weight particles eliminated by urinary excretion. High dose carbenicillin therapy has also been reported to cause "hypersthenuria."[5]

Reported here for the first time to our knowledge is the finding that hetastarch can also cause this discrepancy between urinary specific gravity and osmolality. The in vitro experiment demonstrates that the large molecular weight molecules present in the hetastarch solution will cause a disproportionate rise in specific gravity compared to the simultaneously determined osmolality measurement. We believe that this accounts for the discrepant findings seen in these patients between the urinary specific gravity and the measured osmolality. In the normal volunteer, this discrepancy did not occur, possibly indicating that a normal, undamaged kidney represents a selective barrier to all but the smallest of the hetastarch molecules. Both patients in this report probably had ATN secondary to hypotension and demonstrated abnormal glomerular permeability (demonstrated by positive dipstick proteinuria), thus allowing larger molecular weight particles of hetastarch to be filtered and excreted in the urine.

The elevated urinary specific gravity readings that were obtained prior to the urinary osmolality measurements caused a delay in determining the probable cause of oliguria in the reported patients. We think that hetastarch particles caused the elevation in these values. To date there has been no evidence to suggest that hetastarch itself is intrinsically nephrotoxic.[9]

Oliguric patients with prerenal azotemia will have concentrated urine, while those with ATN will usually have urine that is isotonic to plasma. In addition to urinary concentration, other significant indices or differential points between prerenal azotemia and ATN include low urinary sodium, low fractional excretion of sodium and high U/P creatinine ratio in prerenal azotemia, with inverse findings noted in ATN.[3]

Using only the values of urinary specific gravity in the reported patients, a diagnosis of prerenal azotemia was supported, leading to fluid administration for volume expansion. However, the relatively fixed urinary osmolality determinations were consistent with ATN, which should have led to an entirely different fluid management approach. Furthermore, had the other indices as noted above been utilized, the misleading elevation of the specific gravity should have been recognized.

In summary, in the setting of intrinsic renal damage, hetastarch can elevate the urinary specific gravity with little effect on the determined urinary osmolality or true urinary concentration ability. This misleading information in the oliguric patient could lead to inappropriate fluid therapy. We reiterate the fact that urinary osmolality should be the standard determination for urinary concentration rather than specific gravity, especially after the administration of high molecular weight colloid solution.

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Metastatic calcification: An unusual cause of lower intestinal hemorrhage

MATTHEW M. COOPER, MD

Massive lower intestinal bleeding, often seen in the elderly or other compromised patients with limited reserve, constitutes a medical emergency that requires expedient and focused diagnostic and therapeutic intervention. The more common causes of lower intestinal hemorrhage in adult and elderly patients include diverticular disease, vascular ectasias, or arteriovenous malformations.[1,2] The bleeding associated with colonic malignancy is rarely massive, and while inflammatory bowel disease, colonic polyps, and internal hemorrhoids may account for occasional instances of severe bleeding, such occurrences are uncommon.[1,2] Reported here is a case of severe colonic bleeding resulting from metastatic calcification secondary to multiple myeloma. This case illustrates that uncommon causes of such bleeding usually yield to the standard approach and may warrant no extraordinary diagnostic or therapeutic endeavors.

CASE REPORT

A 65-year-old man was admitted to the medical service of the Manhattan Veterans Administration Medical Center with guaiac positive stools and altered mental status. The history was remarkable for alcohol abuse of long duration and cigarette use of one pack per day. The physical examination was notable for lethargy, cachexia, a blood-stained pharynx, spider angiomata, hepatomegaly, ascites, and testicular atrophy.

Pertinent laboratory values on admission included total calcium, 12.7 mg/dL; blood urea nitrogen, 26 mg/dL; creatinine, 1.8 mg/dL; serum phosphate, 4.4 mg/dL; albumin, 2.0 g/dL; alkaline phosphatase, 65 mU/mL; lactate dehydrogenase, 358 mU/mL; serum glutamic-oxaloacetic transaminase, 124 mU/mL; serum glutamic-pyruvic transaminase, 24 IU/L; CO_2 content, 26 mEq/L; prothrombin time, 17.5 sec; platelet count, 50K/mm^3; bleeding time, > 15 min; and hematocrit, 25%. A chest radiograph showed a right paratracheal mass and a lesion overlying a right anterior rib within soft tissue. Nasogastric lavage yielded coffee-ground-like material and some clots. A subsequent upper endoscopic examination revealed gastritis and moderately severe duodenitis without discrete ulcers or varices.

On the day following admission, the total serum calcium level rose to 14.1 mg/dL, and the patient became rousable only to painful stimuli and suffered focal and grand mal seizures. No abnormalities were detected on computed tomography (CT) of the head. The hypercalcemia was sequentially treated with intravenous hydration, furosemide, glucocorticoids, phosphate, calcitonin, and mithramycin, which lowered the calcium level to 8.5 mg/dL. Melena developed at this time, presumed secondary to the presence of gastritis and duodenitis in the face of a coexistent coagulopathy. Results of serum protein electrophoresis and bone marrow biopsy were diagnostic of multiple myeloma of the IgG Kappa type. Reevaluation of the chest film and physical examination performed on admission demonstrated pulmonary and subcutaneous plasmacytomas.

Ten days after admission, hematochezia developed. An initial flexible sigmoidoscopic and colonoscopic examination revealed multiple areas of erythematous mucosa. A repeat colonoscopy performed three days later due to persistent hematochezia associated with hypotension revealed a small, polypoid, actively pumping source of arterial bleeding in the mid-ascending colon. Cauterization of the bleeding point was not attempted. The hematocrit at this time had fallen ten percentage points. Surgical consultation was obtained, and rapid resuscitation with blood products was begun. Systematic administration of vasopressin was also begun while awaiting an angiogram. An emergent superior mesenteric angiogram obtained for the purpose of confirming the bleeding source(s) and selective infusion of vasopressin demonstrated active arterial bleeding in the mid-ascending colon from a branch of the right colic artery (Fig 1). Selective infusion of vasopressin failed to stop the bleeding. An exploratory laparotomy was performed. Operative findings included macronodular cirrhosis, ascites, diffuse colonic mucosal irregularity, and colonic bleeding from the region previously identified; a right colectomy was performed. Subsequently, gram-negative sepsis developed, presumed secondary to a preexisting pneumonia, and the patient died on the 11th postoperative day.

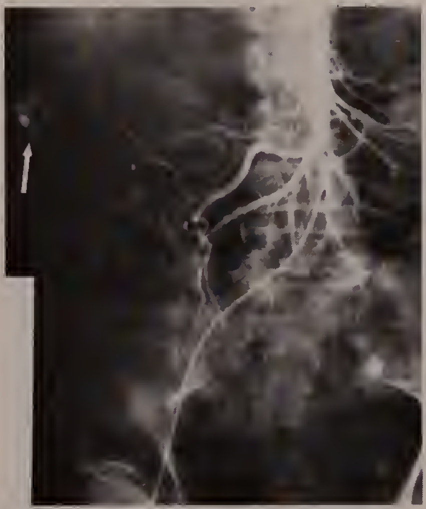

FIGURE 1. Selective superior mesenteric arteriogram. This early film demonstrates extravasation of contrast material from an actively bleeding branch of the right colic artery (left upper corner, arrow).

Pathologic Findings. The resected specimen demonstrated multiple areas of edematous and focally hemorrhagic mucosa with distinct areas of deep ulceration. The ulcers were of similar age and showed granulation and healing. Many necrotic arteries with calcification of the vessel walls were seen within the ulcers (Fig 2). These findings suggested a single causative insult, such as vascular damage resulting from metastatic calcification, occurring one to three weeks prior to operation. A microscopic section taken through an area with adherent blood clot showed a ruptured, medium-sized artery at the surface of an ulcer. Calcification of the vessel wall confirmed the suspicion that the bleeding originated from an artery weakened by deposition of calcium salts. A stain for amyloid was negative.

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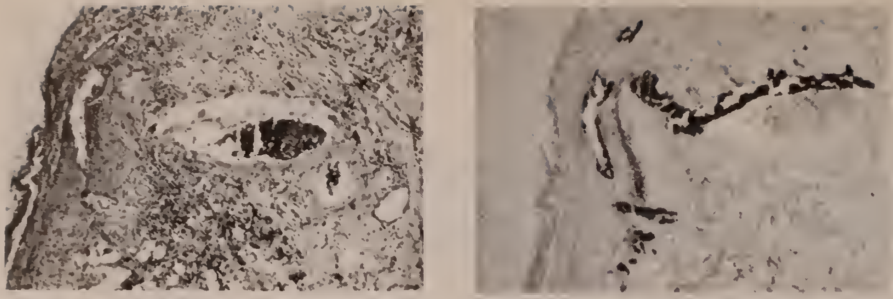

FIGURE 2. **Left,** photomicrograph of necrotic artery within bowel wall (original magnification ×10). **Right,** same artery with Von Kossa stain demonstrating calcific precipitation (original magnification ×10).

DISCUSSION

Early recognition of hypercalcemia of malignancy is critically important if reversal of the renal failure and other associated clinical features is to occur with appropriate therapy.[3,4] The hypercalcemia of cancer is usually steadily progressive and of shorter duration than that of primary hyperparathyroidism.[4,5] As a result, patients may experience symptoms with a relatively low serum calcium concentration. Such features as renal calculi, band keratopathy, hypertension, and pruritus may be absent.[4] The differential diagnosis of hypercalcemia also includes sarcoidosis, vitamin A or D intoxication, the milk-alkali syndrome, thyrotoxicosis, prolonged immobilization, secondary hyperparathyroidism in patients with renal disease, the diuretic phase of acute renal failure, and the use of thiazide diuretics.[3,6-9]

Myeloma is the most common hematologic malignancy associated with hypercalcemia, which occurs in 20-30% of patients with myeloma.[4,5] Osteoclast activating factor, a lymphokine released by normal lymphocytes and produced in increased amounts by malignant cells, is thought to mediate bone destruction and hypercalcemia in multiple myeloma.[4-11]

Deposition of a solid mineral phase of calcium and phosphate in abnormal sites such as the walls of blood vessels, connective tissue, gastric mucosa, cornea, or renal parenchyma (ie, metastatic calcification) occurs in the presence of persistent hypercalcemia, especially when accompanied by normal or elevated levels of serum phosphate.[6-9] Such renal deposition may produce or aggravate kidney damage, with further impairment of calcium excretion.[3] Histologically, metastatic deposits of calcium salts resemble those of dystrophic calcification but are usually more delicate and rarely give rise to large concretions or calculi.[12]

As indicated above, a variety of agents are commonly used to treat hypercalcemia. These include hydration, phosphate supplementation, furosemide, glucocorticoids, calcitonin, mithramycin, and mobilization when possible. Indomethacin has also been used effectively in the treatment of hypercalcemia of malignancy.[5] Noteworthy is the relative effectiveness that has been reported for the combination of glucocorticoids and calcitonin in rapidly decreasing the hypercalcemia of hematologic malignancy, especially in patients with myeloma who have poor renal function.[4,6-9,13] Phosphate supplementation must be used with caution, because at least on theoretical grounds, further metastatic calcification may occur as a consequence of such therapy. In addition, furosemide may exacerbate hypercalcemia in the dehydrated patient.[4]

The case presented here illustrates a rare cause of massive lower intestinal hemorrhage. It is likely that metastatic calcification in this patient occurred in the presence of some degree of preexisting atherosclerotic disease contributing to the total extent of vascular deterioration. Regardless of the etiology of presumed lower intestinal hemorrhage, precise localization of the bleeding source is desirable following confirmation that the bleeding is indeed originating from the large intestine. The possibility of operation underscores the importance of precise localization.

Metastatic calcification is a rare cause of massive lower intestinal bleeding. However, regardless of the cause, the clinician must adhere to the proven tenets of aggressive diagnosis and treatment required in patients with life-threatening lower intestinal hemorrhage.

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Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Nonphysician healers

TO THE EDITOR: You certainly did a service to your readers by publishing the brief case report, "Malpractice by nonphysician healers," which appeared as a letter to the editor in your August 1987 issue.[1]

Why do patients go to nonphysician healers? I thought, when I first went into practice, that it was because of ignorance, but in recent years I have observed intelligent and well-informed patients regularly going to such healers. These healers often offer patients a choice, the right to participate in their own treatment. Because of this, when treatment is unsuccessful, it is claimed that the patients did not participate strongly enough or enthusiastically enough. With a bad result, the patients very often bear both the physical and the psychic scars because of the suggestion that had they participated more strongly or tried harder, they would have gotten better! The transfer of part of the responsibility for therapy to patients—and therefore of the guilt for treatment failures—has shielded these practitioners from malpractice suits. They are able to claim that it is not their negligence but rather a patient's lack of enthusiasm that has caused poor results.

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[1] Nores J-M, Remy J-M, Nenna AD, et al: Malpractice by nonphysician healers. *NY State J Med* 1987; 87:473-474.

Do-not-resuscitate orders

TO THE EDITOR: The recent New York State legislation on do-not-resuscitate (DNR) orders for patients in health care facilities[1] is a big step towards assuring a caring response to people's needs. For 15 years my involvement on committees of the medical staffs of local hospitals has included deliberations of DNR and brain death issues. For years I have listened to patients complain about their feelings of helplessness, and the lack of trust that their wishes would be respected during their final days. The issues of living wills, DNR orders, etc, have been addressed at many levels and yet patients continue to have reasonable concerns for their implementation. I have found that most patients consider it a matter of dignity and a source of peace that they have made these difficult decisions. They fear, however, that, even when a DNR order is written and well documented on the chart, or living wills have been received by the institution in good faith, they could be faced with a cardiopulmonary resuscitation attempt fully implemented. This could occur if they had an arrest in a place where the personnel were not familiar with their personal wishes or the chart was not immediately available.

I would like to make a suggestion, recognizing the appropriate fears and mistrusts expressed by patients, that entails the placement of a discreet identifier on the identification bracelets worn by all patients and residents in health care institutions. The identifier need only be clear enough to be recognized by hospital personnel and discreet enough not to act as a fluorescent message for all to see. This could be accomplished, for example, with a bracelet identical to the usual one but with two colored bands of green a quarter of an inch wide, half an inch apart, discreetly across its width.

This process would help address a variety of issues. It would be a clear identifier of the patient and the orders that are documented on the record. If coordinated identification between nursing homes and acute care facilities were developed within regions, it would permit continuity of information when a patient is transferred from one institution to another. It would be an immediate identifier to a CPR team of the patient's status. But more importantly, it would allow those patients who are alert and competent to know and be able to discuss a decision that is so evidently defined on their bracelets. It would permit the reevaluation at periodic intervals by the patient and the patient's family of the meaningfulness of the status so decided.

Patients who have discussed this with me describe the helplessness that they feel while in institutions; wearing such an identification is, for them, one small piece of control in the awesomeness of their life situations. Of course, some patients may not wish to wear such an identification. In these cases the institution could simply maintain documentation on the chart, as under current prevailing arrangements.

I support the current legislation and find that its elaborate detail protects both the providers of health care and the civil rights of patients, but it does little to address the issues of mistrust and helplessness for those patients who fear that their wishes may not be appropriately carried out. I do not think that legislation is necessary to implement such a simple procedure as I have proposed, but think that each institution can develop a simple mechanism for identification of patients with DNR orders. I further suggest that a uniform fashion of identification such as I have mentioned might be used with sufficient discretion.

I have no objection to the use of the letters "DNR" on the bracelet as a simple identifier. I do object, however, to additional identifier bracelets being added ad nauseum to the patient's extremities. The proliferation of allergy bracelets, high-risk-fall identifiers, and the like is already posing problems. Certainly our technology would permit the implementation of one plastic identifier with sufficient coding to address each of these needs.

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[1] NYS Public Health Law, article 29-B, §§ 2960-2978.

Boxing and the transmission of HIV

TO THE EDITOR: It is important to bring to the attention of the medical profession, the public, and athletes involved in contact sports the issue of possible transmission of the human immunodeficiency virus (HIV) via blood during sports activities.

Several state boxing commissions have recently instituted rules and regulations allowing referees, handlers, and physicians working with boxers to use gloves, thereby diminishing the possibility of HIV transmission via blood from wounds boxers receive during boxing matches.[1] However, to my knowledge, no one has yet raised the issue of the possibility of HIV transmission from one boxer to another during a boxing match. This could occur when a boxer is hit in the head, resulting in a bleeding wound, or when one boxer butts another in the head, causing an open wound and resulting in blood-to-blood contact.[2] This can result in transmission of the HIV virus from a boxer who is infected to one who is not.

This potential risk is of concern in view of the fact that several boxers have publicly admitted that they have had an intravenous (IV) drug abuse problem.[3,4] IV drugs abusers represent the group with the second highest rate of AIDS infection among the more than 55,300 reported cases of AIDS in the United States.[5]

I am not recommending that these known IV drug abusers be prevented from continuing their boxing careers. However, I would suggest that boxers who have a history of IV drug abuse be required to undergo confidential blood testing for HIV status. If found positive they should employ certain precautions such as the wearing of head protection to diminish the chance of transmitting the HIV virus via an open bleeding wound.

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Physician penmanship

TO THE EDITOR: I work at a facility that serves physically disabled people. Our adult clients are regularly referred to physicians for medical examinations and treatment. Physicians are provided with a specific, neatly written or typed referral stating the patient's relevant history, problems, and requested services. The physician sends back to the facility a report describing the findings.

I find it disturbing that the majority of the reports we receive are completely illegible. Not only are the examination summaries and recommendations sloppily handwritten, but the signatures are usually scribbled so that, if it is a hospital clinic, for example, it may be impossible to identify the physician responsible in order to clarify the report.

It is a dangerous and time consuming practice to guess at words in an attempt to decipher diagnoses. It is also a frustrating experience for the client to undergo hours of testing, and to have no results available due to poor professional habits.

When presented with the problem, support personnel give such responses as, "If it's so important, send the patient back in and we'll reexamine him," "Doctors have bad handwriting—that's a fact of life," "We have no time to worry about that," or "The doctor is not in."

Many physicians do not provide typewritten reports. Your organization should encourage physicians to type reports and send them out the following day rather than writing them illegibly in an effort to give patients a report on the spot. If typing is not feasible, I think that clear hand-printing is not too much to ask.

I realize that physicians are busy people (as we all are) and that the issue of penmanship seems like a trivial matter. It is, however, a larger problem of professional attitude and of fostering workable relationships between disciplines. It is also well to remember that a medical record or chart that cannot be read is one that cannot easily be defended later on.

I hope that this letter will at least raise awareness of this problem among the members of the Medical Society of the State of New York.

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Lyme disease

Introduction

Lyme disease is an emerging worldwide public health problem which has disproportionately affected New Yorkers, especially in the southern portion of the state. The first New York State case was recognized in 1977 and by early 1980, anecdotal case reports approached 100. The discovery in 1981 of the spirochete from Long Island ticks provided the agent for diagnostic evaluation. In 1982, with 167 cases serologically confirmed, New York State accounted for 40% of the total cases reported in the United States. Since 1982, approximately 3,000 cases have been documented among New York State residents. Lyme disease was included among the list of mandated reportable diseases in January 1986. Presently, there is a high but stable level of transmission in the endemic areas of eastern Long Island and the lower Hudson Valley. There is, however, increased transmission in areas peripheral to these endemic zones.

Lyme disease is an acute febrile systemic illness which involves the skin, joints, nervous system and heart. Because of its protean manifestations, Lyme disease can be divided into stages. Primary, nondisseminated Lyme disease is restricted to the skin. Secondary and tertiary stages represent the disseminated phases of this disease to other organ systems. It has a tendency to become chronic with relapses and remissions, especially of arthritic symptoms, that can continue for years. Most New York State cases have had a history of erythema chronicum migrans (ECM). In addition, 30–50% have had arthritis, 20% neurologic manifestations and 8% cardiac manifestations. Hospitalization was required in 22% of the New York State cases. In the early years of Lyme disease investigations, most hospitalizations were for differential diagnoses. Presently, most Lyme disease hospitalizations are for treatment. It is now known that Lyme disease can affect most organ systems and can be transmitted via the placenta, leading to fetal death and potentially to congenital malformations. Death has been rare.

The distribution of cases within New York State has been a result of agent, host and environmental factors. *Borrelia burgdorferi*, the agent, is a spirochete which is transmitted to humans from small wild rodents (mice and voles) by the tick *Ixodes dammini*. While the animal and human hosts for the agent are located throughout the state, the *Ixodes dammini* ticks, until recently, had been found only in Long Island and the lower Hudson Valley. The environmental reasons for this limited tick distribution are unclear, but may be artifactual as evidence now suggests a northward expansion of the tick through the Hudson Valley. In 1982, there were no *Ixodes dammini* seen on deer in Dutchess County during the fall hunting season. In 1984, these ticks were the primary ectoparasite in the deer sampled at the same time. By the beginning of 1987, *Ixodes dammini* ticks were also found in Columbia and Washington counties. However, all human and dog cases of Lyme disease north and west of Dutchess and Ulster counties had travel histories to the southern endemic areas of New York or other states. During 1987, however, reports of the first two local cases from Dutchess and Ulster counties were received, providing evidence for a northern spread.

The following is a descriptive profile of 600 patients with serologically confirmed Lyme disease, surveyed between 1982 and 1985.

Age and Sex Distribution

Males outnumbered females in all age groups until the seventh decade of life where the numbers of females equaled the numbers of males, and thereafter with females outnumbering males (Table I). Nearly half of Lyme disease (LD) cases consisted of people under the age of 19 (Table I). Similarly, higher incidence rates of Rocky Mountain spotted fever (RMSF) have been seen in children and young adults than in older age groups.

Geographic Distribution

As of May 1985, native LD occurred only in Suffolk, Westchester and Putnam counties. However, one native case occurred in Rockland in 1985. The New York City cases are not native to any of the five boroughs. (As of July 1987, native cases of LD have also been reported in Ulster, Dutchess and Orange counties.)

Incidence figures by county are misleading. Native LD is virtually nonexistent in Western Suffolk and in urban southern Westchester, which are areas that contain the bulk of the population for each county.

A general pattern of incidence has emerged:

- areas where transmission is intense can have 20 or more cases per 1,000; these areas could be viewed as

TABLE I. Age and Sex Distribution of 600 New York Patients with Lyme Disease, 1983–1985

Age	Male (%)	Female (%)	Total (%) by Age
0–9	90 (65)	48 (35)	138 (23)
10–19	92 (66)	47 (34)	139 (23)
20–29	32 (62)	20 (38)	52 (9)
30–39	41 (63)	24 (37)	65 (11)
40–49	35 (57)	26 (43)	61 (10)
50–59	33 (56)	26 (44)	59 (10)
60–69	29 (50)	29 (50)	58 (10)
70–79	8 (35)	15 (65)	23 (4)
80 +	2 (40)	3 (60)	5 (.8)
Total	362 (59%)	238 (41%)	600 (100%)

epicenters of endemic zones;

- areas where there are two to five cases per 1,000 are the most common and may indicate an endemic situation;
- areas with one case per 1,000 are almost always at the fringes of the above first two areas and may represent developing foci.

Although noncontiguous spread of LD (for example, by migrating birds carrying infected ticks) is possible, the incidence patterns suggest a spread consistent with expansion to areas immediately adjacent to the endemic zones.

In the group of 600 cases, 497 (71%) did not report a travel history for at least one month before onset. Furthermore, 357 (59%) patients acquired LD in the towns of their residence where more than ten cases had occurred. These data strongly suggest that exposure to infected vectors occurs in or near the homes of the patients.

Clinical Manifestations of LD in New York

All of the known manifestations of Lyme disease were represented in this group. Table II presents a breakdown by sex of all the cases according to the number of organ systems involved in the disease at the time of diagnosis.

The most common pattern of LD follows a sequence beginning with skin involvement (primary) to neurological and cardiovascular involvement (secondary) to arthritis (tertiary). This pattern is clinically recognizable. However, many patients experience involvement of only one organ system thereby decreasing clinical recognition. The erythema migrans (skin lesion) of LD can be confused with other dermatoses of summer onset. Furthermore, because antibodies are not often detected during the skin (primary) stage of the disease, the number of patients with EM is underrepresented in this cohort of 600. Erythema migrans (EM) is the single most common manifestation of this disease.

TABLE II. Major Clinical Manifestations of LD in Study Group

	Male (%)	Female (%)	Total
I. Involvement of 1 organ system			
A. Erythema migrans (EM)	68 (61)	43 (39)	111
B. Joints	104 (67)	52 (33)	156
C. Central/Peripheral Nervous (NS)	17 (63)	10 (37)	27
D. Cardiovascular (CV)	2 (66)	1 (34)	3
Subtotal	191	106	297
II. Involvement of 2 organ systems			
A. EM and joints	98 (56)	77 (44)	175
B. EM and NS	30 (56)	23 (44)	53
C. EM and CV	5 (55)	4 (45)	9
Subtotal	133	104	237
III. Involvement of 3 or more organ systems			
A. EM and NS and joints	20 (64)	11 (38)	31
B. EM and CV and joints	5 (45)	6 (55)	11
C. EM and CN and CV	0	5 (100)	5
D. EM and CN and CV and joints	14 (73)	5 (27)	19
Subtotal	39	27	66
Total	362	238	600

Joint Involvement

A total of 156 patients (26% of the 600) had tertiary LD (arthritis) as the only manifestation of this disease. That is, these patients never had or never noticed an EM lesion and did not develop secondary manifestations prior to diagnosis. This group of patients presented some difficulties in that the classical pattern or sequence of LD manifestations was bypassed leaving the clinician or diagnostician with nonspecific joint involvement as the sole clue to management of the illness. Although exposure to infected ticks is primarily a summer occurrence, the initial manifestations of joint involvement are not. The onset of Lyme arthritis is most frequent in the fall and winter months.

Other Manifestations

Patients who experience some form of the sequential stages of LD (Table II) can be diagnosed by the clinical findings and by eliciting a good history. Few diseases can mimic LD in the sequence of manifestations which by themselves can be nonspecific. Neurological manifestations were noted in 135 patients in this study group (23%). The most common neurological sign was Bell's palsy followed by meningitis (requiring spinal taps or hospitalization) and by peripheral neuritis. Headache and fatigue as neurological symptoms were not measured as they appeared to be extremely common complaints among patients of all categories. Papilledema were reported in five patients and there was one case of blindness. These ocular manifestations are included in the NS categories. Cardiovascular manifestations were reported in 8% of the patients (47/600, Table II). These manifestations included the following: tachycardia, atrioventricular blocks, carditis and congestive heart failure.

For more information about Lyme disease or to receive copies of a public information Lyme disease pamphlet, contact Dennis J. White, PHD, Director, Arthropod-borne Disease Services Unit, at (518) 474-6730.

Injuries from falls

Falls are the second leading cause of unintentional injury deaths in New York State. In 1986, there were 920 deaths attributed to falls (*International Classification of Diseases, 9th Revision External Cause Codes 880–888*) for all ages, compared to 2,162 deaths from motor vehicle accidents, the highest category, and 333 from fires and flames, the third highest category.

For the elderly, falls are the leading category of injury-related deaths and hospitalization. Hip fractures are the most common type of injury resulting from falls in people aged 65 and older and constitute the single most common diagnosis among all injuries in all age groups leading to hospital admission. In New York State, there was an average of 2,860 hospital discharges per year for acute care of fractured hip in men ages 65+ for 1980–85 and 11,173 such discharges for women, accounting for 450,598 bed days of acute care per year. Because of the increasing proportion of elderly people in the population, it is especially important to find ways to reduce the incidence and costs of hip fractures in the elderly.

The majority of falls to children under age ten and the elderly occur in or near a residential setting, and for all ages combined, falls are the leading cause of home injury mortality and morbidity (Figure 1). Because of this, falls are a major focus for prevention in the New York State Department of Health's Home Safe Home Program. Further study is needed to define the epidemiology of falls in New York State, but for the United States, some groups at high risk of fatal falls are black males under age 70 and white females over age 70. Urban children living in high-rise buildings have a high rate of falls out of windows.

Factors related to the high rate of falls in the elderly are living alone, mental confusion or dementia, a number of chronic conditions, alcohol use, cardiac arrhythmias, use of tranquilizers and other drugs affecting the central nervous system, disturbances of balance, vision and hearing, reduced muscle mass, osteoporosis, arthritis and neurological diseases.[1,2] In the absence of physical limitations, environmental factors play a relatively larger role.

A home safety checklist has been developed for use by local health professionals, community outreach workers and volunteers to conduct home hazards surveys in high-risk communities, such as low-income neighborhoods and aggregate housing for the elderly, as part of the Health Department's Home Safe Home Program. Health workers also assist residents with correction of hazards in their homes by repairing broken steps, installing railings or bathtub grab-bars and safety gates on stairs.

Home injury prevention demonstration projects for the elderly in Niagara and Westchester counties are being supported by grants from the department's Injury Control Program. There is a need to evaluate the effectiveness of changing the design and materials used in private homes and institutions in order to reduce the impact of falls, and thereby reduce the frequency and severity of the injuries associated with such falls. These same principles have been applied successfully in the design of automobiles and in the use of protective clothing in sports such as football and hockey.

Prevention of osteoporosis on a communitywide basis through increasing exercise levels and calcium intake in all adults might reduce the rate of hip fractures resulting from falls. Reduction in alcohol consumption could also reduce the rate of falls and related injuries, since as many as 70% of fatal falls in adults may be alcohol related.[3] Better studies are needed in this area, since blood alcohol and other drug levels are not routinely measured in patients treated for fall injuries.

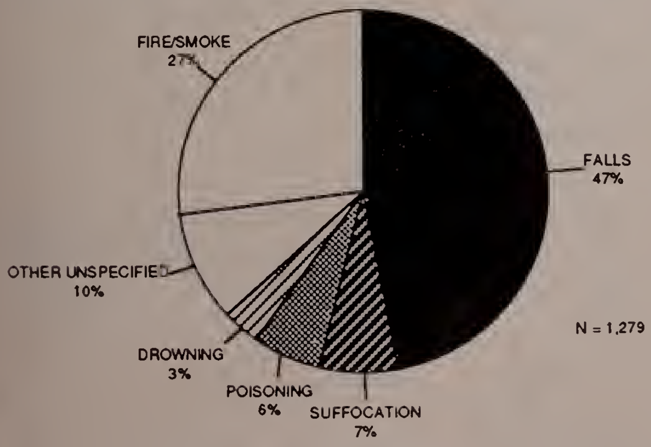

FIGURE 1. Proportion of home injury deaths by category of injury, New York State, 1984.

For more information about injury prevention, contact: Dr Susan Standfast, Director, Injury Control Program, (518) 473-1143.

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Prevalence of HIV infection in blood donors

In future issues, *Epidemiology Notes* will report the results of departmental studies of the prevalence of human

TABLE I. All Blood Donors (for Transfusion), HIV Antibody Seroprevalence Trends, New York State, April 1985–September 1987

Quarter	# Tested	# Positive	% Positive
New York State			
Apr–Jun '85	170,754	133	0.078
Jul–Sep '85	194,609	190	0.098
Oct–Dec '85	182,181	137	0.075
Jan–Mar '86	199,327	105	0.053
Apr–Jun '86	196,232	98	0.050
Jul–Sep '86	191,974	80	0.042
Oct–Dec '86	196,826	85	0.043
Jan–Mar '87	209,071	68	0.033
Apr–Jun '87	201,133	63	0.031
Jul–Sep '87	201,687	63	0.031
Downstate[1]			
Apr–Jun '85	90,328	121	0.134
Jul–Sep '85	94,478	170	0.180
Oct–Dec '85	81,700	121	0.148
Jan–Mar '86	98,355	91	0.093
Apr–Jun '86	92,156	91	0.099
Jul–Sep '86	97,275	73	0.075
Oct–Dec '86	97,921	75	0.077
Jan–Mar '87	105,331	58	0.055
Apr–Jun '87	99,797	58	0.058
Jul–Sep '87	102,992	56	0.054
Upstate[2]			
Apr–Jun '85	80,426	12	0.015
Jul–Sep '85	100,131	20	0.020
Oct–Dec '85	100,481	16	0.016
Jan–Mar '86	100,972	14	0.014
Apr–Jun '86	104,076	7	0.007
Jul–Sep '86	94,699	7	0.007
Oct–Dec '86	98,905	10	0.010
Jan–Mar '87	103,740	10	0.010
Apr–Jun '87	101,336	5	0.005
Jul–Sep '87	98,695	7	0.007

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1. Downstate (New York Blood Services, Long Island and Hudson Valley Blood Services).

2. Upstate (Albany, Syracuse, Rochester and Buffalo American Red Cross Blood Services).

TABLE II. First Time Blood Donors (for Transfusion), HIV Antibody Seroprevalence, New York State, July–September 1987

	Males			Females			Total		
	# Tested	# Pos.	% Pos.	# Tested	# Pos.	% Pos.	# Tested	# Pos.	% Pos.
NY State	11,673	16	0.137	11,378	5	0.044	23,050	21	0.091
Downstate[1]	7,203	15	0.208	6,857	3	0.044	14,059	18	0.128
Upstate[2]	4,470	1	0.022	4,521	2	0.044	8,991	3	0.033

1. Downstate (New York Blood Services, Long Island and Hudson Valley Blood Services).
2. Upstate (Albany, Syracuse, Rochester, and Buffalo American Red Cross Blood Services).

Notes: Tables do not include blood donors who indicate that their blood should not be used for transfusion purposes. Nearly 75% of the New York State blood supply is provided by regular, repeat donors. All donors are tested each time they donate blood. Those who test positive are notified and their blood is removed from the transfusion pool.

Source: New York State Council on Human Blood and Transfusion Services.

immunodeficiency virus (HIV) in New York State. This edition focuses on the results of a nondepartmental testing program—the HIV antibody testing of blood donors.

Table I depicts the HIV antibody test experience in New York State for blood donors from April 1985 through September 1987. The HIV antibody seroprevalence rate in blood donors has decreased gradually since testing of blood supplies began in April 1985. Among the approximately 200,000 blood donors tested each quarter in New York State, the percent positive for HIV antibodies has decreased from a high of 0.098 (one in 1,025) during the third quarter of 1985 to 0.031 (one in 3,200) during the third quarter of 1987.

The fact that donors identified as HIV positive are not eligible to again donate blood is believed largely to account for the steady decline.

The rate of HIV infection among first time blood donors during the third quarter of 1987 (Table II) is comparable to the rates observed during the first two reported quarters in 1985 when from the perspective of HIV antibody testing, virtually all blood donors were first time donors.

Although there has not been systematic follow-up and reporting of the risk factors of those blood donors who test HIV positive, individual reports indicate that the preponderance of HIV positive donors could be categorized within known AIDS risk groups.

Breast cancer screening

Breast cancer is a major cause of illness and death in American women today. About one in ten women in the United States will develop breast cancer during her lifetime. Until the causes of the disease are better understood and preventable, the focus of public health attention will be on secondary prevention through early detection and treatment. If a woman follows a complete breast screening program, breast cancers are more likely to be detected when they are in an early, treatable stage.

Breast cancer is primarily a disease of women and every woman is at risk as she grows older. Because of genetic and lifestyle differences, some women are at greater risk than others. Some of the factors that increase risk include advancing age, a family history of breast cancer, being older than 30 years at the first live birth, never giving birth, eating a diet high in fat and obesity. Breast cancer is not known to be associated with hormone use, nor is it believed to be related to chemical pollution in the environment.

A physician may recommend a mammogram (a low-dose breast X-ray) which can identify cancers too small to be felt. Mammography screening is recommended annually for all women over age 50. For younger women or women in high-risk groups, the physician should discuss with patients the recommended guidelines. Recent improvements in equipment and technique have reduced the amount of radiation necessary for high quality mammograms. A personal physician can discuss the benefits and potential risks of mammography for individual women. All women should be told where they can obtain the best quality mammogram with the lowest X-ray exposure.

A clinical breast exam should complement a mammogram and should be carried out during regular medical checkups. It is recommended that all women over the age of 20 examine their breasts once a month. Breast self-exam is encouraged as the third component of a complete breast cancer screening program because symptoms can develop and be noticed between clinical breast exams or mammography. Breast self-exam includes several important components:

- regularity;
- complete coverage;
- consistent pattern;
- adequate pressure;
- use of finger pads.

For more information about breast cancer screening, contact Lois G. Youngblood, Associate Director, Cancer Surveillance Program, at (518) 474-4871.

DISTURBED BEHAVIOR IN THE ELDERLY

Edited by A. G. Awad, MD, Henry Durost, MD, H. M. Rosemary Meier, MB, and W. O. McCormick, MB. 189 pp. Elmsford, NY, Pergamon Books, Inc, 1987. $40.00 (hardcover)

The title of this book does not do justice to its contents. This is more than a clinical presentation of "disturbed behavior;" it is a consideration of the broad issues of social planning, community organization, and manpower training, all in the service of the needs and care requirements of the growing segment of our population over 65 years of age. This dual approach stems from the fact that the book was developed from two conferences held in Toronto. The first, a clinical meeting, was organized with the Toronto Western Hospital and held at the Queen Street Mental Health Center; the second, a community oriented conference, was sponsored by the Division of Geriatric Psychiatry of the University of Toronto and organized by the Whitby Psychiatric Hospital and Sunnybrook Medical Center. The editors' introduction states that the conferences were a "collaboration between members of staff of provincial government hospitals and teaching general hospital departments of psychiatry" and were "planned for providers of care for the elderly."

With such a broad audience, the resulting papers are unfortunately spotty. Many lack the depth required by the expert in the field, some deal with material of little clinical relevance, some offer new thoughts and understanding. The clinical issues section, for example, is particularly lacking in depth in its chapters on clinical use of psychotropic medication and dementia. Pharmacokinetics of medication is only briefly outlined, research on individual drugs is not presented, and the voluminous research on memory is not considered. On the other hand, the clinical approach to disturbing behavior is practical and multidimensional, although not unlike comprehensive evaluation done in settings other than Toronto.

The issues in social planning and community organizations are not presented to provide definitive solutions but largely to raise questions and to offer directions. Robertson (Division of Geriatric Medicine, University of Saskatchewan) for example, points out that although in Canada 10% of the elderly are in long term care facilities, that figure must be broken down to only 1.4% of 65–74-year-olds in such facilities, contrasted with 36.4% for those aged 85 and over. The demographic shift to the older population must thus be considered in planning. Arie (Head of the Department of Health Care of the Elderly, Nottingham University, England) asks, "are supportive services more effective when they are supporting supporters (ie, patient's family) than when they are supporting the patient who lives alone?" A system-wide view of services for the elderly is stressed.

A provocative chapter on law and mental health care for competent and for incompetent elderly persons is presented by Somerville (Faculties of Law and Medicine, McGill University, Montreal). What are the rights of the elderly to use and refuse mental health care, the duties of community health professions and family to make care available? What are the principles of informed consent in the elderly, the legal limitations, the disclosure of information, the rules which "seek to balance the conflict between a mentally ill patient's right to automony and intervention on the basis of beneficial paternalism?" This chapter is informative and thoughtful.

In all, this book is not meant to be a textbook on geriatric psychiatry or even on health care for the elderly. It is a series of papers, reflecting the diverse experiences of physicians, psychologists, social scientists, and legal experts as they have attempted to solve the problems of the elderly in their care. The edited papers are collected into a small volume, easily skimmed for its occasional gem.

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REALITY ORIENTATION FOR THE ELDERLY

Third Edition. By Sylvester Kohut, Jr, PHD, Jeraldine J. Kohut, RN, Joseph J. Fleishman, PHD. 160 pp. Oradell, NJ, Medical Economics Co, 1987. $18.95 (paperback)

Providing psychosocial support and orientation cues to the demented patient is the general theme of *Reality Orientation for the Elderly* by Sylvester Kohut, Jeraldine Kohut, and Joseph Fleishman. The authors provide the reader with a brief review of psychosocial and medical information on common problems of older adults, especially those persons requiring institutionalization. The second section of the book provides a detailed description of a reality orientation program that can be established in most institutional settings.

All the authors and contributors to the book have had professional experience in hospital and long term care settings. It should be noted that the chapters dealing with medical and therapeutic topics were in part prepared by either a physician or a pharmacologist. As is the case in many situations, these authors are well known in nursing and gerontological circles, but not as well recognized in medical ones. This factor, however, does not in any way detract from the authority or accuracy of the book.

This reviewer thought that the first section, dealing with general gerontological topics, was a good introduction for the beginning student in this area. None of the chapters went into any great depth as to current research or medical information. Some of the demographic data were not as current as they could have been. The chapter on the institutionalized elderly could have been expanded in the area of alternative care settings available to the chronically ill or demented patient. In addition, the section on sexuality was excessively brief and not very informative, even for the beginning student. The second part of this book describes in greater detail a program of reality orientation (RO) for the confused and demented elderly. For anyone who has lived with a demented person or worked in a setting with a large number of mentally impaired individuals, this issue of providing visual, verbal, and environmental orientation is a crucial one. These chapters provide the reader with several types of programs that can be implemented either at home or in health related institutions or nursing facilities. The methods varied from visual cueing to that of group sessions. This section also included evaluation tools that can be utilized by the clinician.

The proposed audience for this book is social workers and nursing home and hospital personnel. Physicians can, however, benefit from reviewing the second section of this book, even though most reality orientation programs are headed by gerontologists. This reviewer feels that it is important for the referring physician to understand programs such as this in order to make appropriate referrals. This book will appeal to physicians who work in long term care settings. This book can be reviewed in a short period of time and then used as a reference for reality orientation programs.

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PSYCHIATRY AND THE MENTAL HEALTH PROFESSIONALS. NEW ROLES FOR CHANGING TIMES

Formulated by the Committee on Governmental Agencies, Group for the Advancement of Psychiatry. 216 pp. New York, Brunner/Mazel Publishers, 1987. $13.95 (paperback)

Since its formation in 1946, the Group for the Advancement of Psychiatry (GAP), made up of several hundred psychiatrists representing a broad spectrum of interests in the field, has formulated committee reports that amount to important policy statements on issues requiring thoughtful guidance. GAP committees freely call on consultants from other disciplines in making their reports. Such was the case in this report, *Psychiatry and the Mental Health Professionals—New Roles for Changing Times*, formulated by the Committee on Governmental Agencies. This book is worth reading even though it describes a generally disturbing situation.

Section I, "The Mental Health Field," describes the development of psychiatry and the mental health movement. Within a few pages the problem begins clearly to emerge. Following World War II there was considerable governmental support for expanding the number of mental health professionals. Psychiatrists numbered only about 7,000 in 1950 but by 1980 their number had quadrupled; psychologists, however, now number about 50,000, or twice as many; social workers now number approximately 87,000; and the number of psychiatric nurses is estimated at 56,000. The problem lies in the fact that all of these mental health professionals function best as members of a team, all making an essential contribution to diagnostic assessment, treatment planning, and intervention. Ideally, and especially in hospitals or clinics, the psychiatrist is the team leader and carries ultimate clinical responsibility for the patient's management. In real life the mental health disciplines are all drawn to solo private practice, with strong rivalry for status, reimbursement, and control. In private practice psychiatrists rarely work on a team. US-educated and trained psychiatrists do not usually work in state, county, or city hospitals, or in community mental health centers. Medical leadership, desirable as it may seem for physicians, is unattractive to many other disciplines who feel they have greater specialized competence in certain areas. In the Appendices a psychologist, a social worker, and a nurse state their strong objections to the way psychiatrists have tried to define the roles for the other mental health disciplines. It is well known that, especially in some of the community mental health centers, all staff roles and functions seem similar. Only psychiatry recently has made a strong move toward coming closer to medicine, and with a growing base of knowledge in the neurosciences and neuropharmacology, psychiatry probably will become increasingly "medicalized." At the same time, more than in any other medical specialty, psychiatry will remain embedded in a biopsychosocial systems model.

Section II, "The Providers," makes the important observation that 60% of the estimated 32,000 sufferers from mental illness receive care only from primary care physicians; only 21% are treated entirely by mental health specialists; and the remaining one-fifth receive no treatment. Ideally there would be a great advantage in a system of care in which primary care physicians screened and triaged patients for the rest of the health care team. Primary care providers would then continue the patient's care after special health or mental health services were provided. The disturbing fact is that primary care physicians in most settings do a poor job of assessing their patients' needs for special emotional care, making skillful referral, or providing maintenance care. Physicians in other branches of medicine can profit from the excellent background information offered in this book concerning the roles of other mental health disciplines. Some patients are appropriately referred to them.

Section III, "Changing Times," does not strike a positive note. Given the fact that approximately 15% of the United State population suffers from clinically significant mental illness, our present system of mental health care is not adequate to meet the need. Certainly our mental health teams, at their present level of organization, cannot handle the task. Over the past quarter of a century a national policy of deinstitutionalization has resulted in a sharp reduction in numbers of patients in state hospitals, without adequate provision of community-based programs of treatment and care. With pressures to reduce health and mental health expenditures, pressures will mount to hire nonmedical mental health professionals at low levels of pay, especially as efforts will be made to provide mental health care as a money-making enterprise. Serious planning will be required to develop a health and mental health system designed to provide manpower, program, and budget to meet our national need.

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DEPRESSION PREVENTION. RESEARCH DIRECTIONS

Edited by Ricardo Munoz, PHD. 301 pp, illustrated. New York, Hemisphere Publishing Corporation, 1987. $49.95 (hardcover)

This book consists of 16 chapters written by researchers and clinicians who attempt to show various preventive strategies for depression. The editor, Ricardo Munoz, PHD, has assembled a group of 27 psychiatrists and psychologists who have all had previous experience in depression research. Dr Paula Clayton, Chair of the Department of Psychiatry at the University of Minnesota, has written extensively in the field of widowhood and depression. Her chapter in this book deals with preventive strategies for bereavement. Hagop S. Akiskal, MD, Director of the Affective Disease Program at the University of Tennessee, and a recipient of a 1981 Clinical Research Award of the American Academy of Clinical Psychiatrists, has written the chapter, "An Overview of Biobehavioral Factors in the Prevention of Mood Disorders." Dr Robert E. Roberts, of the University of Texas Health Science Center at Houston, who has done a great deal of research on depression involving various ethnic groups, wrote the chapter on "Epidemiological Issues in Measuring Preventive Effects."

Various other authors from other centers including the University of Wisconsin Medical School, the VA Medical Center at Palo Alto, California, and the Michigan Department of Mental Health present chapters in areas as diverse as running therapy, postpartum depression, depression in low income populations, and childhood and adolescent depression. All of the chapters cover preventive strategies.

The book is written in a sometimes technical but clear fashion. Several of the chapters lay out research protocols in a step-by-step approach. Many of the tables are well presented and there are good reference listings for many of the chapters.

Depressive illness is a major cause of psychological, medical, psychosocial, and economic distress throughout the world, and certainly not enough emphasis has been placed on dealing with possible preventive strategies for it. This book tries in a sometimes successful way (depending on the particular chapter) to deal with the problem of how to set up research treatment protocols for the prevention of depression.

For those readers who are interested in looking into various research protocols, this book offers some valuable suggestions. But for those who are more fascinated by clinical case discussions, this book is probably unappealing. Overall, I believe that a book of this nature could be an important addition in the area of research involving the prevention of depression.

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MENTAL RETARDATION. NATURE, CAUSE, AND MANAGEMENT

Second Edition. By George S. Baroff, PHD. 541 pp, illustrated. New York, Hemisphere Publishing Corp, 1986. $44.95 (hardcover)

This is the second edition of a book providing both an overview and coverage of the important current aspects of this topic. It is written by a highly respected professional in the field of mental retardation. He has updated the initial edition of 1974 by succinctly including major advances which have occurred since then regarding research findings, program developments, and the rights of the retarded.

The book presents clearly the latest findings as to the nature, types, and etiologies of mental retardation. The individual psychological and familial dimensions of these chronic handicaps are presented in a straightforward yet well-balanced fashion. The developmental approach which the author takes to educational and recreational program needs reflects the latest approaches to this topic across our country. Similarly, the delineation of key vocational and residential treatment-management approaches is clearly organized and technically excellent. The section on managing behavioral problems in the severely retarded tends not to live up to the excellence of the overall book. This chapter unduly focuses on aversive behavior modification approaches, while excluding the wide range of other treatment/management techniques/approaches which are available for these complex retarded citizens.

This book is a fine overview of the modern dimensions of the field of mental retardation. It will meet the information needs of a wide variety of advanced undergraduate and graduate students in the area of clinical psychology, education, medicine (eg, medical students), social work, nursing, and vocational rehabilitation.

The clarity and high professional quality of the author's coverage of this topical area are outstanding.

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THE ROLE OF DRUG TREATMENTS FOR EATING DISORDERS

Edited by Paul E. Garfinkel, MD, and David M. Garner, PHD. 192 pp, illustrated. New York, Brunner/Mazel Publishers, 1987. $20.00 (hardcover)

Over the past 25 years, approaches to the treatment of the classic eating disorders, anorexia nervosa (AN) and bulimia nervosa, reveal a telescoping of the history of psychiatric treatment in general over the past century. An initial enthusiasm for classical, libidinally oriented psychoanalysis has typically given way to a more flexible, ego psychology-oriented psychotherapy. Family and group therapy have been tried and often found helpful, and behavior modification and cognitive-psychoeducational approaches have also gained adherents. But probably the most dramatic development has been the application (often merely empirically) of psychoactive drugs.

It is thus not surprising that the first book specifically devoted to the topic of pharmacotherapy in the eating disorders has now been published. A monograph edited by Drs Paul Garfinkel and David Garner, whose 1982 book, *Anorexia Nervosa: A Multidimensional Perspective* (New York, Brunner/Mazel), remains the standard by which all other such texts must be measured, *The Role of Drug Treatments for Eating Disorders* presents us with a thoughtful, generally scholarly overview of the possible application of medication in the treatment of anorexia nervosa and bulimia nervosa. The authors of all the chapters are obviously expert with the particular class of drugs they review, and all are wise enough to emphasize firmly that medication can only be part of a much broader treatment regimen (and that often it may well be the least important component, and indeed even clearly contraindicated in certain situations).

The introduction by Garfinkel and Garner is an excellent, if brief, overview of the rationale for the current growing interest in the use of medications in treating AN and bulimia, and the ensuing chapters on monoamine oxidase inhibitors (MAOIs), tricyclic antidepressants (TCAs), antianxiety agents, neuroleptics, lithium, anticonvulsants, and serotonin altering drugs serve as a nice review of psychopharmacology in general.

The chapter on MAOIs by Kennedy and Walsh provides an intelligent, succinct summary of the controversy surrounding whether a common etiologic relationship exists between eating disorders and affective disorders. Unfortunately, some redundancy on this issue is found in the Herzog and Brotman chapter on tricyclic antidepressants. Kennedy and Walsh remind us of the paradox that, while antidepressants were first employed in bulimia because of the frequent concurrence of depressive symptoms, the actual presence of depressive symptoms in a given patient does not necessarily predict a positive response to MAOIs (ie, by a reduction in bingeing), and similarly their absence need not predict a negative response. Comparable paradoxes are noted by other chapter authors as well. Anderson relates that anxious feelings are as ubiquitous as are depressed feelings among anorectic and bulimic patients, and thus the judicious use of antianxiety agents should not be overlooked during the current enthusiasm for antidepressants (at least for bulimia). Vandereycken, in his chapter on the use of neuroleptics in anorexia nervosa, reveals that, while many clinicians (including this reviewer) still believe that chlorpromazine can have a beneficial role in the acute, inpatient management of the out-of-control anorectic, there have simply been no controlled studies to confirm or refute this belief. Kaplan notes the truly inconsistent findings that have emerged in attempts to replicate early reports of a positive response to phenytoin in bulimia. Kay and Goldbloom, in their respective chapters on the possible roles of opioids and serotonin in the eating disorders, note the intriguing theoretical speculations that emerge from animal studies, while revealing that clinical studies have been far more ambiguous.

This is a readable, interesting, up-to-date book which certainly should be consulted by all physicians who work with eating-disorder patients. However, one would hope that, a decade from now, considerably more will be known about which drugs genuinely do have a significant role in the treatment of these frequently pernicious disorders and which can be discarded.

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BOOKS RECEIVED

AIDS

AIDS. Ethics and Public Policy. By Christine Pierce and Donald VanDeVeer. 241 pp. Belmont, Calif, Wadsworth Publishing Co, 1988. $13.50 (paperback)

AIDS: Principles, Practices and Politics. Edited by Inge B. Corless, RN, and Mary Pittman-Lindeman, DrPH. 252 pp. New York, Hemisphere Publishing Corp, 1988. $15.95 (hardcover)

HEALTH CARE

Home Health Care. Second Edition. By Allen D. Spiegel, PhD. 442 pp, illustrated. Owings Mills, Md, Rynd Communications, 1987. $49.00 (hardcover)

HISTORY OF MEDICINE

The Age of Miracles: Medicine and Surgery in the Nineteenth Century. By Guy Williams. 234 pp, illustrated. Chicago, Ill, Academy Chicago Publishers, 1987. $16.95 (hardcover)

Resources in Medical History. On Insanity and its Classification. By Vincenzo Chiarugi. 380 pp. Canton, Mass, Watson Publishing International, 1987. $35.00 (clothbound)

An Account of Bellevue Hospital. Edited by Robert J. Carlisle, MD. 381 pp, illustrated. New York, The Society of the Alumni of Bellevue Hospital, 1983. $17.50 (hardcover)

INFECTIOUS DISEASE

Smallpox and Its Eradication. By F. Fenner, MD, D. A. Henderson, MD, I. Arita, MD, Z. Jezek, MD, and I. D. Ladnyi, MD. 1,460 pp, illustrated. Geneva, Switzerland, World Health Organization, 1988. $200.00 (hardcover)

LABORATORY MEDICINE

Clinical Decision Levels for Lab Tests. Second Edition. By Bernard E. Statland, MD, 224 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $21.95 (softcover)

MEDICAL EDUCATION

Medicine. Preserving the Passion. By Phil R. Manning, MD, and Lois DeBakey, PhD. 297 pp, illustrated. New York, Springer-Verlag, 1987. $35.00 (hardcover)

Clinical Teaching for Medical Residents: Roles, Techniques, and Programs. Edited by Janine C. Edwards, PhD, and Robert L. Marier, MD. 270 pp, illustrated. New York, Springer Publishing Co, 1988. $35.95 (hardcover)

MISCELLANEOUS

The Home Medical Handbook. A Patient's Guide to the Most Up-to-Date Treatments, Tests and Technologies in the Field of Home Care Including Safe, Inexpensive Advice on the Prevention, Diagnosis, and Treatment of Common Ailments. Jack I. Stern, MD, and David L. Carroll. 370 pp, illustrated. New York, William Morrow & Co, Inc, 1987. $17.95 (clothcover)

American Voices. Prize-winning Essays on Freedom of Speech, Censorship and Advertising Bans. 440 pp. New York, Philip Morris, 1987. $4.75 (hardcover)

Choices for a Healthy Heart. By Joseph C. Piscatella. 580 pp, illustrated. New York, Workman Publishing Co, Inc, 1987. $14.95 (spiral binding)

Executive Retirement Management. A Manager's Guide to the Planning and Implementation of a Successful Retirement. By Jack J. Leedy, MD, and James Wynbrandt. 260 pp. New York, Facts on File, Inc, 1987. $24.95 (hardcover)

Vietnam: A Casebook. By Jacob D. Lindy, MD. 353 pp, illustrated. New York, Brunner/Mazel Publishers, 1987. $40.00 (hardcover)

When Living Hurts. By Sol Gordon, PhD. 160 pp. New York, Dell Publishing Co, 1988. $3.95 (paperback)

Enhancing Marital Intimacy Through Facilitating Cognitive Self-Disclosure. By Edward M. Waring, MD. 256 pp. New York, Brunner/Mazel Publishers, 1988. $25.00 (hardcover)

Hard Bodies. Express Workout. The Twice-a-Week-Weight-Training Program That Works! By Gladys Portugues and Joyce L. Vedral, PhD. 160 pp, illustrated. New York, Dell Publishing Co, Inc, 1988. $12.95 (paperback)

Social Learning and Systems Approaches to Marriage and the Family. Edited by Ray DeV. Peters, PhD, and Robert J. McMahon, PhD. 340 pp. New York, Brunner/Mazel Publishers, 1988. $37.50 (hardcover)

Payment in Full. A Guide to Successful Bill Collecting. By Leonard Bendell. 174 pp. Gainesville, Fla, Triad Publishing Co, 1987. $24.95 (hardcover)

NUTRITION

Popular Nutritional Practices: Sense and Nonsense. By Jack Z. Yetiv, MD. 416 pp. New York, Dell Publishing Co, Inc, 1986. $4.95 (paperback)

Controlling Cholesterol. By Kenneth H. Cooper, MD. 357 pp, illustrated. New York, Bantam Books, 1988. $18.95 (hardcover)

OBSTETRICS—GYNECOLOGY

Critical Care Obstetrics. Edited by Steven L. Clark, MD, Jeffrey P. Ohelan, MD, and David B. Cotton, MD. 508 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $52.95 (hardcover)

OCCUPATIONAL MEDICINE

Occupational Back Pain—Spine. Volume 2. Edited by Richard A. Deyo, MD. 168 pp, illustrated. Philadelphia, Hanley & Belfus, Inc, 1987. $32.00 (hardcover)

ONCOLOGY

Manual of Clinical Oncology. Fourth Edition. Edited by Charles D. Sherman, MD, Kenneth C. Calman, MD, Sandor Eckhardt, MD, Ismail Elsebai, MD, Dincer Firat, MD, Dieter K. Hossfeld, MD, Jean-Pierre Paunier, MD, and Bruno Salvadori, MD. 372 pp, illustrated. New York, Springer-Verlag, 1988. $28.00 (softcover)

Liver Tumors: Multidisciplinary Management. Edited by W. John B. Hodgson, MD. 323 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1988. $47.50 (hardcover)

OPHTHALMOLOGY

Manual for Eye Examination and Diagnosis. Third Edition. By Mark W. Leitman, MD. 80 pp, illustrated. Oradell, NJ, Medical Economics Books, 1988. $18.95 (paperback)

PEDIATRICS

The State of the World's Children 1988. By United Nations Children's Fund. 86 pp, illustrated. New York, Oxford University Press, 1988. $6.25 (softcover)

Medicaid and Pediatric Primary Care. By Janet D. Perloff, Phillip R. Kletke, and Kathryn M. Neckerman. 190 pp, illustrated. Baltimore, Md, The Johns Hopkins University Press, 1987. $30.00 (hardcover)

Pediatric Trauma Care. Edited by Martin R. Eichelberger, MD, and Geraldine L. Pratsch, RN. 219 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1988. $33.00 (hardcover)

Assuring Quality Out-Patient Care for Children. Guidelines and a Management System. By Charles D. Cook, MD, and Jane Heidt. 204 pp, illustrated. New York, Oxford University Press, Inc, 1988. $35.00 (hardcover)

PSYCHIATRY

Issues in Community Mental Health: Youth. By Eugene Aronwitz and Robert Sussman. 103 pp.

Canton, Mass, Watson Publishing International, 1987. $6.95 (paperback)

Wrong Diagnosis—Wrong Treatment: The Plight of the Alcoholic in America. By Joseph D. Beasley, MD. 271 pp. Durant, Okla, Creative Informatics, Inc, 1987. $12.95 (paperback)

Understanding the Alcoholic's Mind. The Nature of Craving and How to Control It. By Arnold M. Ludwig, MD. 188 pp. New York, Oxford University Press, 1988. $16.95 (hardcover)

Clinical Psychiatry. From Synopsis of Psychiatry. By Harold I. Kaplan, MD, and Benjamin J. Sadock, MD. 566 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $24.95 (paperback)

Synopsis of Psychiatry. Behavioral Sciences. Clinical Psychiatry. Fifth Edition. By Harold I. Kaplan, MD, and Benjamin J. Sadock, MD. 723 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $32.50 (paperback)

When Living Hurts. Directives for Treating Depression. By Michael D. Yapko, PhD. 240 pp. New York, Brunner/Mazel Publishers, 1988. $25.00 (hardcover)

Psychotherapy Tradecraft. The Technique and Style of Doing Therapy. By Theodore H. Blau, PhD. 320 pp. New York, Brunner/Mazel Publishers, 1988. $27.50 (hardcover)

The Good News About Depression. By Mark S. Gold, MD. 340 pp. New York, Bantam Books, 1986. $9.95 (paperback)

Annual Progress in Child Psychiatry and Child Development. Edited by Stella Chess, MD, Alexander Thomas, MD, and Margaret Hertzig, MD. 643 pp, illustrated. New York, Brunner/Mazel Publishers, 1988. $45.00 (hardcover)

Old Loyalties, New Ties. Therapeutic Strategies with Stepfamilies. By Emily B. Visher, PhD, and John S. Visher, MD. 272 pp, illustrated. New York, Brunner/Mazel Publishers, 1988. $27.50 (hardcover)

RADIOLOGY

Magnetic Resonance Imaging of the Thorax. By Miriam Sperber, MD, and Marc C. Kaiser, MD. 270 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1987. $65.00 (hardcover)

SURGERY

General Surgery: Review and Assessment. Third Edition. By Richard M. Stillman, MD. 438 pp, illustrated. East Norwalk, Conn, Appleton & Lange, 1988. $24.95 (paperback)

TOXICOLOGY

Medical Toxicology. Diagnosis and Treatment of Human Poisoning. By Matthew J. Ellenhorn, MD, and Donald G. Barceloux, MD. 1,512 pp, illustrated. New York, Elsevier Publishing Co, Inc, 1988. $175.00 (hardcover)

TRAUMA

Trauma. Edited by Kenneth L. Mattox, MD, Ernest Eugene Moore, MD, and David V. Feliciano, MD. 960 pp, illustrated. East Norwalk, Conn, Appleton & Lange, 1988. $125.00 (clothcover)

UROLOGY

Urology: Problems in Primary Care. Edited by Elroy D. Kursh, MD, Martin I. Resnick, MD. 332 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $32.95 (softcover)

News from the Department of Health and Human Services

The US Food and Drug Administration (FDA) granted approval for the National Institute of Allergy and Infectious Diseases to begin a series of clinical trials to study the effectiveness of the immunomodulating drug AS-101 as a treatment for AIDS. In addition, approval of an investigational new drug application was granted to Dr Jacob Bitran of Michael Reese Hospital (Chicago, Ill) for clinical tests with a combination of alpha interferon and zidovudine as a treatment for AIDS.

GD Searle and Co, Ortho Pharmaceutical Corp, and Syntex Corp, the three manufacturers of high-dose estrogen oral contraceptives, have agreed to FDA recommendations to discontinue the manufacture and distribution of these pills. Based on the findings of the Fertility and Maternal Health Drugs Advisory Committee that high-dose estrogen pills are no more effective than low-dose pills and that there may be an association between the dosage and the risk of blood clotting disorders, the FDA recommended that suppliers of contraceptive pills that contain more than 50 μg of estrogen withdraw those products from the market. The manufacturers will phase out distribution of high-dose pills over a six-month period, during which time physicians can switch patients to low-dose contraceptive pills that contain 30–50 μg of estrogen.

How effective are condoms in preventing HIV transmission?

The surgeon general has recommended the use of condoms as a means of protection against the spread of human immunodeficiency virus (HIV). Theoretically, an undamaged latex condom should be an effective physical barrier against any known sexually transmissible agents, note Rietmeijer et al (*JAMA* 1988; 259:1851–1853). And in vitro studies have shown that organisms such as *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, herpes simplex virus type 2, cytomegalovirus, hepatitis B virus, and HIV cannot pass through a condom.

The authors developed an in vitro model system to simulate the physical forces associated with vaginal and anorectal intercourse. They used this model to test the ability of condoms to prevent HIV transmission. Furthermore, because condoms have been associated with a 10–15% contraceptive failure rate, and because they can break or slip off, the authors sought to determine whether the addition of the spermicidal agent nonoxynol 9 to condoms could provide a second, chemical barrier against HIV transmission.

The model consisted of a hollow, formed-rubber dildo inserted into a sterile, glass cylinder that contained liquid tissue culture medium. To simulate intercourse, the dildo was pumped up and down in the cylinder. After each pumping cycle, the researchers used pipettes to withdraw 1-mL samples from the fluid in the cylinder and from the condom tip. These samples were cultured and tested for HIV.

In the first phase of testing, HIV inoculum was introduced by pipette into the condom tip. In the second phase, the condom tip was purposely ruptured. These tests were performed using condoms with and without nonoxynol 9. In addition, nonoxynol-9-containing condoms were tested with HIV inoculum in the cylinder and HIV-free medium in the condom tip. Condoms with nonoxynol 9 contained the spermicide in the condom tip as well as mixed in with the silicone lubricant that was evenly distributed over the inner and outer condom surfaces. (Standard spermicidal condoms only contain nonoxynol 9 in the lubricant.)

The results were as follows: Of the ten condoms without nonoxynol 9, all samples taken from the cylinder fluid following phase one of testing were negative for HIV. After rupture of the condom, 7/10 samples were positive. Of the ten condoms with nonoxynol 9 and with HIV inoculum inside the condom, 0/10 samples from inside the condom were positive before rupture. Of the ten condoms with nonoxynol 9 and with HIV inoculum outside the condom, 2/10 samples taken from the cylinder fluid before rupture were positive for HIV. After rupture, 0/10 samples taken from outside these condoms were positive.

From these findings, Rietmeijer et al concluded that "intact latex condoms are impermeable to HIV and that condoms containing nonoxynol 9 may inactivate HIV in case of condom slippage or breakage under conditions permitting a nonoxynol 9 concentration greater than 0.25%." An estimated nonoxynol 9 concentration of 0.04% outside the condom before rupture was sufficient to inactivate HIV in eight of ten cases.

The authors concluded that if a condom does not break or slip off during actual intercourse, it can effectively prevent most transmission of HIV. They cannot, however, predict with certainty the in vivo activity of nonoxynol 9 against HIV, particularly in the rectal area. In addition, although no adverse effects of nonoxynol 9 in rectal use have been reported, the spermicide's safety in anorectal intercourse has not been shown.

Physicians' responses to AIDS

The *Canadian Medical Association Journal* (1988; 138:736–741) sent contributing editor Dr Brian Goldman to interview internists and surgeons at San Francisco General Hospital in an attempt to better understand how physicians who treat patients with AIDS on a daily basis are responding to the risks such care entails.

Denial of risk appears to be the primary response of health care workers as a group. However, after October 1987, when officials notified the staff of San Francisco General that a nurse had become infected with HIV following an accidental needle stick injury, staff members could no longer take refuge in the safety of denial. "The announcement has sparked a running battle between surgeons and internists at the hospital, a battle that concerns fundamental questions of medical ethics."

While one internist defended the Centers for Disease

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Control (CDC) position that health care workers are at low risk of becoming infected with HIV on the job, a surgeon at the hospital contended that the CDC "soft-pedalled" the risk for health care workers. She noted the many opportunities for accidental sticks and cuts that surgeons encounter, as well as the frequent exposure to potentially contaminated blood.

Goldman quotes Dr Paul Volberding, chief of the AIDS Activities Division at the hospital, saying, "We need to accept that the surgeons are afraid and that they have some reason to be afraid. And that we should be doing everything we can to reduce their risk."

The surgeon interviewed by Goldman advocates more protective clothing and equipment as well as mandatory testing for all elective patients believed to be at high risk for HIV infection. Patients undergoing emergency surgery would be exempt from such testing. This proposal has sparked an ethical debate. On the one hand, some physicians fear a two-tiered health care system, in which HIV-infected patients would not have access to the same level of care as other patients. Should patients be denied certain procedures based on their HIV antibody status? In a similar vein, however, Goldman poses an equally difficult question: At what point does the risk to the surgeon outweigh the benefit to the patient? Some surgeons have stated that knowing the HIV status of patients would allow them to employ safer, although more time consuming, surgical techniques.

This San Francisco surgeon paints a gloomy picture for the future. "It's easy for generals who are in a 15th-floor penthouse in Paris to make the rules for the infantry. We are the ones on our bellies and on our elbows and knees trying to get to the foxhole while we're being shelled. I have the feeling that, when this is all over, a good percentage of those of us who are on the front lines may be dead. And the people who have been making the strategy and telling us what we should do will be riding in the parade."

Pediatric cardiovascular health center

The Babies Hospital division of The Presbyterian Hospital/Columbia-Presbyterian Medical Center now offers a program for children who are prone to developing heart disease. Officials hope that early identification and treatment of children at risk can help prevent premature cardiovascular disease. The Pediatric Cardiovascular Health Center will serve the populations of metropolitan New York, New Jersey, and Connecticut.

Treatment of children who are prone to early development of heart disease can prevent excessive accumulation of fatty arterial plaques. The pediatric center will stress reduction of risk factors associated with coronary artery diseases—including control of blood pressure and cholesterol levels—and will offer dietary counseling and, if needed, drug therapy.

New screening test improves early detection of bladder cancer

A new method of screening for bladder cancer, called quantitative fluorescence imaging analysis (QFIA), requires only one urine sample. The QFIA test uses a fluorescent dye to analyze the DNA in individual cells. This sensitive detection method can identify a single cancerous cell.

Measuring the amount of fluorescence after staining the cells can identify abnormal levels of DNA, which are characteristic of cancer cells. Visual monitoring of the stained cells confirms the presence of increased DNA synthesis.

This screening test (developed by CytoDiagnostics, Inc; Houston, Tex) presents no risk or discomfort to the patient and may prove more sensitive for early detection of bladder cancer than computed tomographic scanning.

Eggs found to be major source of salmonella poisoning

During the ten years from 1976 to 1986, the incidence of *Salmonella enteritidis* infection has increased throughout the United States, with a remarkable sixfold increase in the northeastern US (*JAMA* 1988; 259:2103–2107). Of the foodborne outbreaks of *S enteritidis* in this region for which a specific food source was identified, approximately 77% were attributed to Grade A eggs or foods that contained such eggs.

An important distinction from most previous salmonella outbreaks, which were attributed to cracked eggs or eggs contaminated by chicken feces, is that recent incidences of salmonella poisoning in the Northeast have been caused by uncracked eggs that have been treated with disinfectants. Researchers attribute the outbreaks to eggs that became contaminated by *S enteritidis* during ovulation. In the past, *S pullorum* had been known to exist in the ovaries of chickens for many generations, producing no symptoms of infection.

Since there is currently no program for controlling for transovarian transmission of *S enteritidis*, the only sure means of destroying the bacteria is through sufficient cooking of the egg. Consumption of raw or undercooked eggs and egg products—such as Caesar salad dressing, hollandaise sauce, and eggnog—carries the risk of *S enteritidis* infection.

MSSNY Position Paper on Physician Recredentialing

The House of Delegates of the Medical Society of the State of New York (MSSNY) adopted the following position paper on physician recredentialing at its April meeting.

The January 1988 Report of the New York State Advisory Committee on Physician Recredentialing entitled "Phase One: General Principles, Proposed Process, Recommendations" was released earlier this year. The Medical Society of the State of New York does not endorse this report or its recommendations.

Historically, quality assurance (optimal patient care) and physician competency have been the foundations upon which the policies and positions of the MSSNY have been developed. Over the past decade, the assurance of quality and maintaining of competence, a responsibility which rightly belongs within the purview of the profession (all professions), has been gradually assumed to a large extent by agencies external to the profession. Perhaps well intentioned, the regulatory mechanisms developed by these external agencies have had a deleterious effect on the delivery of medical care but have had little impact on physician clinical performance.

The MSSNY agrees with the statement, made on several occasions in the report, that the recredentialing process broadly outlined in the report "is not designed to measure medical competence." Indeed the report does little more than discuss those well known methods used to evaluate those various, individual components which when collectively are used to define knowledge and cognitive skills, not performance.

We agree with the report that there does not exist a single methodology for measuring competence and agree that employment of a combination of methodologies to measure competency would be logistically and economically unrealistic. The evaluation of competence in the health professions has not yet reached maturity. Measurement of changes in practice as a consequence of additional education, assessment of the validity of examinations and the determination of goals for competence are all necessary parts of the ongoing development of competence evaluation.

As stated in the beginning of this statement, MSSNY is committed to quality assurance and maintaining competence of health professionals. However, we do not need further government intrusion to do what already is being done. Accordingly, the MSSNY subscribes to the following recommendations of the "Health Policy Agenda for the American People."

- Health professionals are individually responsible for maintaining their competence and for participating in continuing education; all health professionals should be engaged in self-selected programs of continuing education. In the absence of other financial support, individual health professionals should be responsible for the cost of their own continuing education.
- Professional schools and health professions organizations should develop additional continuing education self-assessment programs, should prepare guides to continuing education programs to be taken by practitioners throughout their careers and should make efforts to ensure that acceptable programs of continuing education are available to practitioners.
- Health professions organizations and faculty of programs of health professions education should develop standards for competence. Such standards should be reviewed and revised periodically.
- When reliable and cost-effective means of assessing continuing competence are developed, they should be required for continued practice.

This should be done without government interference or control.

Mandatory Medicare Assignment

The House of Delegates of the Medical Society of the State of New York adopted the following resolution concerning mandatory Medicare assignment.

Resolved, That the Medical Society of the State of New York take all appropriate actions to defeat any and all legislation which would require physicians to accept Medicare assignment on a mandated basis as a condition of licensure, or through any other sanction; and be it further

Resolved, That such opposition include, at a minimum, the following five actions:

(a) immediate formulation and implementation of a program of direct and regular communication by the MSSNY, county medical societies, specialty societies and all individual physicians with individual State senators and assemblypersons for the purpose of expressing organized medicine's opposition to any such legislation, including the specific reasons upon which such opposition is based;

(b) immediate formulation and implementation of a program of regular communication by physicians, individually and organizationally, with senior citizens, individually and organizationally, which program shall fully inform senior citizens of the actual and potential negative consequences to *them* of mandatory Medicare assignment legislation;

(c) immediate procurement, when available, of the study now underway in the State of Massachusetts under the auspices of the Massachusetts Medical Society concerning the effect of mandatory Medicare assignment on physician practices and on physician availability to Medicare eligible persons and the appropriate use of such study when it is obtained in opposition to all mandatory assignment legislation in New York State;

(d) further encouragement of all physicians to join MSSNY's Voluntary Assignment Acceptance Program (VAAP) through the utilization of MSSNY publications, appropriate mailings, and medical society meetings, including hospital-medical staff meetings; and further, every effort should be made to inform the legislators of the State of New York of the existence of the VAAP Program and to disseminate this information widely to Medicare beneficiaries;

(e) MSSNY implementation of a program of communication with newspapers, radio, and television stations in New York State for the purpose of explaining the basis of organized medicine's opposition to mandatory assignment legislation; and be it further

Resolved, That MSSNY initiate all appropriate legal actions at the State court and/or Federal court level challenging any mandatory assignment law, if and when enacted, on all appropriate legal and constitutional grounds.

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Norman Nathaniel Adler, MD, Babylon. Died March 2, 1988; age 64. He was a 1948 graduate of the University of Virginia School of Medicine, Charlottesville. Dr Adler was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Stanley Amelkin, MD, Rockville Centre. Died April 17, 1988; age 58. Dr Amelkin was a 1960 graduate of Faculte di Medicine de l'Universite de Lausanne, Lausanne, Switzerland. He was a member of the American Society of Internal Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Harvey Jay Amsterdam, MD, Oyster Bay. Died March 21, 1988; age 68. Dr Amsterdam was a 1944 graduate of Tulane University School of Medicine, New Orleans. He was a Diplomate of the American Board of Family Practice and a member of the American Academy of Family Practice, the Nassau County Medical Society, and the Medical Society of the State of New York.

John Beck, MD, Palm Beach Gardens, Florida. Died February 14, 1988; age 77. He was a 1936 graduate of Faculte de Medecine de l'Universite de Geneve, Geneva, Switzerland. Dr Beck was a Diplomate of the American Board of Radiology. His memberships included the New York Roentgen Society, the Radiological Society of New York State, the Academy of Medicine, the Radiological Society of North America, Inc, the Richmond County Medical Society, and the Medical Society of the State of New York.

John Jacob Bookman, MD, New York. Died March 27, 1988; age 77. He was a 1939 graduate of New York University School of Medicine, New York. Dr Bookman was a Diplomate of the American Board of Internal Medicine and a member of the Clinical Society of the New York Diabetes Association, the New York County Medical Society, and the Medical Society of the State of New York.

Norbert Bromberg, MD, Yorktown Heights. Died April 5, 1988; age 82. He was a 1937 graduate of Jefferson Medical College of Thomas Jefferson University, Philadelphia. Dr Bromberg was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the Academy of Medicine, the American Association of Psychoanalytic Physicians, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Vincent S. Celestino, MD, Boca Raton, Florida. Died March 13, 1988; age 63. Dr Celestino was a 1955 graduate of State University of New York at Buffalo School of Medicine, Buffalo. He was a Diplomate of the American Board of Family Practice and a member of the American Academy of Family Practice, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Samuel Lewis Cohen, MD, Miami Beach, Florida. Died March 7, 1988; age 88. Dr Cohen was a 1924 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Herman Robert Cohn, MD, Bronx. Died April 1, 1988; age 79. He was a 1937 graduate of Faculte de Medecine de l'Universite de Lausanne, Lausanne, Switzerland. Dr Cohn was a Fellow of the American Academy of Family Practice, a Diplomate of the American Board of Family Practice, and a member of the Bronx County Medical Society and the Medical Society of the State of New York.

John Ruskin Dallas, MD, Southampton. Died March 19, 1988; age 82. Dr Dallas was a 1933 graduate of Georgetown University School of Medicine, Washington, DC. He was a Qualified Fellow of the International College of Surgeons. Dr Dallas was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Frank Louis De Furio, MD, Auburn. Died March 13, 1988; age 83. Dr De Furio was a 1930 graduate of State University of New York Health Science Center at Syracuse. He was a member of the Medical Society of the County of Cayuga and the Medical Society of the State of New York.

Dominic J. Dell Aquila, MD, New Rochelle. Died March 20, 1988; age 84. He was a 1928 graduate of the University of Michigan Medical School, Ann Arbor. Dr Dell Aquila was a Diplomate of the American Board of Obstetrics and Gynecology and a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Anne Mary Drislane, MD, Albany. Died April 7, 1988; age 73. Dr Drislane was a 1940 graduate of Albany Medical College of Union University, Albany. She was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics and the American Board of Psychiatry and Neurology. Her memberships included the American Psychiatric Association, the American Academy of Child Psychiatry, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Jerome Wilbur Engle, MD, Bronxville. Died March 26, 1988; age 79. Dr Engle was a 1935 graduate of the University of Michigan Medical School, Ann Arbor. He was a member of the Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Irving Jonathan Estrin, MD, New Rochelle. Died March 17, 1988; age 73. Dr Estrin was a 1940 graduate of New York University School of Medicine, New York. He was a Diplomate of the

American Board of Physical Medicine and Rehabilitation. His memberships included the Academy of Medicine, the American Rheumatism Association, the American Academy of Physical Medicine and Rehabilitation, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Stiles Dean Ezell, MD, Manchester Center, Vermont. Died February 23, 1988; age 81. Dr Ezell was a 1932 graduate of Jefferson Medical College of Thomas Jefferson University, Philadelphia. He was a member of the Medical Society of the County of Washington and the Medical Society of the State of New York.

John Leo Finnegan, MD, Fresh Meadows. Died March 20, 1988; age 87. He was a 1929 graduate of State University of New York Health Science Center at Brooklyn. Dr Finnegan was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Edward Isidore Gersh, MD, Bronx. Died March 16, 1988; age 93. Dr Gersh was a 1919 graduate of New York University School of Medicine, New York. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

George Joseph Ginandes, MD, New York. Died April 10, 1988; age 84. He was a 1927 graduate of Cornell University Medical College, New York. Dr Ginandes was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. His memberships included the Academy of Medicine, the New York Pediatric Society, the New York County Medical Society, and the Medical Society of the State of New York.

Leon Ginzburg, MD, New York. Died March 19, 1988; age 90. Dr Ginzburg was a 1920 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the Academy of Medicine, the New York State Surgical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Mor Gluck, MD, Flushing. Died April 9, 1988; age 78. Dr Gluck was a 1939 graduate of Deutsche Univerzita Medizinische Fakulta, Praha, Czechoslovakia. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Soll Goodman, MD, White Plains. Died March 23, 1988; age 75. He was a 1937 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. Dr Goodman was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the American Academy of Child Psychiatry, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Sidney Hirsch, MD, Cedarhurst. Died April 6, 1988; age 86. He was a 1926 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Hirsch was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the Academy of Medicine, the Pan American Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Robert Simon Hoffman, MD, Schenectady. Died January 20, 1988; age 79. Dr Hoffman was a 1936 graduate of Faculty of Medicine University of Edinburgh, Edinburgh, Scotland. He was a Diplomate of the American Board of Physical Medicine and Rehabilitation and a member of the American Academy of Physical Medicine and Rehabilitation, the American Geriatrics Society, the Medical Society of the County of Schenectady, and the Medical Society of the State of New York.

Vansel S. Johnson, MD, Rhinebeck. Died March 26, 1988; age 90. He was a 1925 graduate of the University of Kansas School of Medicine, Lawrence-Kansas City. Dr Johnson was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Academy of Medicine, the New York State Surgical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Nathan Krinsky, MD, Brooklyn. Died February 4, 1988; age 85. He was a 1930 graduate of Boston University School of Medicine, Boston. Dr Krinsky was a member of the Clinical Society of the New York Diabetes Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

S. H. Kritzalis, MD, New York. Died January 11, 1988; age 92. Dr Kritzalis was a 1924 graduate of Faculte Mixte de Medecine et de Pharmacie de l'Universita de Lyon, Lyon, Rhone, France. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Arthur Edward Lamb, MD, Brooklyn. Died March 7, 1988; age 87. He was a 1926 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Lamb was a Fellow of the American College of Physicians and the American College of Cardiology and a Diplomate of the American Board of Internal Medicine. He was a member of the New York State Society of Internal Medicine, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

W. Kenneth Lane, MD, New York. Died April 16, 1988; age 69. He was a 1948 graduate of Georgetown University School of Medicine, Washington, DC. Dr Lane was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

William LaVine, MD, Teaneck, New Jersey. Died March 12, 1988; age 84. He was a 1928 graduate of the State University of New York Health Science Center at Syracuse. Dr LaVine was a Fellow of the American College of Obstetricians and Gynecologists and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Herbert E. Lee, MD, Snyder. Died April 17, 1988; age 68. Dr Lee was a 1953 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Fellow of the American College of Anesthesiologists and a member of the American Society of Anesthesiologists, Inc, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Ira W. Leibner, MD, Brooklyn. Died April 7, 1988; age 70. He was a 1943 graduate of Washington University School of Medicine, St Louis, Missouri. Dr Leibner was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Arthur Linksz, MD, New York. Died March 19, 1988; age 91. He was a 1925 graduate of Medizinische Fakultaet der Christian Albrechts Universitaet, Kiel, Schleswig-Holstein, Germany. Dr Linksz was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. His memberships included the Academy of Medicine, the New York Society for Clinical Ophthalmology, the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, the New York County Medical Society, and the Medical Society of the State of New York.

Merrill David Lipsey, MD, New York. Died April 8, 1988; age 87. He was a 1925 graduate of Cornell University Medical College, New York. Dr Lipsey was a Fellow of the American College of Surgeons and a Qualified Fellow of the International College of Surgeons. He was a member of the Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Josephine A. Lockwood, MD, New York. Died March 21, 1988; age 58. Dr Lockwood was a 1959 graduate of New York Medical College, New York. She was a Fellow of the American College of Preventive Medicine and a Diplomate of the American Board of Internal Medicine. Her memberships included the Academy of Medicine, the Clinical Society, the New York Diabetes Association, the American Public Health Association, the New York County Medical Society, and the Medical Society of the State of New York.

Dante Walter Lombardi, MD, Brooklyn. Died December 29, 1987; age 64. Dr Lombardi was a 1948 graduate of Facolta di Medicina e Chirurgia dell'Universita di Napoli, Naples, Italy. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Paul J. Maloney, MD, Batavia. Died March 28, 1988; age 75. Dr Maloney was a 1937 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the Medical Society of the County of Genesee and the Medical Society of the State of New York.

John Aloysius Mangieri, MD, St Petersburg, Florida. Died December 15, 1987; age 95. Dr Mangieri was a 1920 graduate of Eclectic Medical College, Cincinnati. He was a member of the Dutchess County Medical Society and the Medical Society of the State of New York.

Michael Ralph Mazzei, MD, Staten Island. Died March 19, 1988; age 77. He was a 1939 graduate of St Louis University School of Medicine, St Louis, Missouri. Dr Mazzei was a Qualified Fellow of the International College of Surgeons and a member of the American Society of Abdominal Surgeons, the Richmond County Medical Society, and the Medical Society of the State of New York.

John G. McCauley, MD, Englishtown, New Jersey. Died March 15, 1988; age 81. Dr McCauley was a 1935 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Academy of Family Practice, the Nassau County Medical Society, and the Medical Society of the State of New York.

Edward R. Mountain, MD, Olean. Died March 12, 1988; age 77. Dr Mountain was a 1937 graduate of Yale University School of Medicine, New Haven, Connecticut. He was a Diplomate of the American Board of Otolaryngology and a member of the American Academy of Otolaryngology, the Medical Society of the County of Cattaraugus, and the Medical Society of the State of New York.

Herbert Francis Newman, MD, New York. Died March 18, 1988; age 78. He was a 1934 graduate of Harvard Medical School, Boston. Dr Newman was a Diplomate of the American Board of Surgery and a member of the New York County Medical Society and the Medical Society of the State of New York.

Pasquale J. Pagano, MD, Bronx. Died April 18, 1988; age 79. He was a 1935 graduate of Rush Medical College, Chicago. Dr Pagano was a Fellow of the American College of Chest Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York Cardiological Society, the New York State Society of Internal Medicine, the American Geriatrics Society, the American Thoracic Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

William Rufus Phillips, MD, Elmira. Died March 22, 1988; age 79. He was a 1932 graduate of Vanderbilt University School of Medicine, Nashville. Dr Phillips was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Medical Society of the County of Chemung and the Medical Society of the State of New York.

Armand E. Rainville, MD, Weston, Connecticut. Died January 9, 1988; age 87. Dr Rainville was a 1928 graduate of Tufts University School of Medicine, Boston. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Morris Meier Reschke, MD, New York. Died April 6, 1988; age 89. Dr Reschke was a 1926 graduate of Friedrich-Wilhelms-Universitaet Medizinische Fakutaet, Berlin, Germany. He was a Diplomate of the American Board of Dermatology, Inc, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Aaron D. Ruchocki, MD, Bal Harbor, Florida. Died June 22, 1987; age 77. He was a 1936 graduate of Universitaire Libre de Bruxelles, Faculte de Medicine et de Pharmacie, Bruxelles, Belgium. Dr Ruchocki was a Fellow of the American Academy of Family Practice and the American College of Allergists. His memberships included the New York Society for Physical Medicine and Rehabilitation, the American Academy of Allergy, the American Geriatrics Society, the New York County

Medical Society, and the Medical Society of the State of New York.

John Julian Scott, MD, Garden City. Died April 15, 1988; age 65. Dr Scott was a 1948 graduate of Hahnemann Medical College of Philadelphia, Philadelphia. He was a member of the Academy of Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Walter Marcel Stillger, MD, Garden City. Died March 22, 1988; age 68. Dr Stillger was a 1943 graduate of New York University School of Medicine, New York. He was a member of the American Academy of Family Practice, the Nassau County Medical Society, and the Medical Society of the State of New York.

Martha Stutzel, MD, Flushing. Died March 2, 1988; age 97. Dr Stutzel was a 1920 graduate of Medizinische Fakultaet der Universitaet, Muenchen, Bayern, Germany. She was a member of the American Medical Women's Association, the Bronx County Medical Society, and the Medical Society of the State of New York.

Edward S. Tauber, MD, New York. Died March 3, 1988; age 80. He was a 1934 graduate of Johns Hopkins University School of Medicine, Baltimore. Dr Tauber was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the Academy of Psychoanalysis, the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Ira Teicher, MD, New Hyde Park. Died April 19, 1988; age 76. Dr Teicher was a 1936 graduate of Cornell University Medical College, New York. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the New York State Surgical Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

Eduard W. Violin, MD, Yorktown Heights. Died February 25, 1988; age 91. He was a 1923 graduate of Universitaet Wien, Medizinische Fakultaet, Wien, Austria. Dr Violin was a member of the Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Morris M. Weiner, MD, Brooklyn. Died February 27, 1988; age 93. Dr Weiner was a 1918 graduate of New York University School of Medicine, New York. He was a Fellow of the American Academy of Family Practice. His memberships included the New York Cardiological Society, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Leonard A. Weinroth, MD, New York. Died March 11, 1988; age 80. Dr Weinroth was a 1935 graduate of Universite de Paris VI, Paris. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the American Medical Society for Alcoholism, the New York County Medical Society, and the Medical Society of the State of New York.

Hyman M. Weiselberg, MD, Jamaica. Died March 29, 1988; age 78. He was a 1936 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Weiselberg was a Diplomate of the American Board of Radiology and a member of the New York Roentgen Society, the Radiological Society of North America, Inc, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Leon L. Wiesel, MD, Port Washington. Died February 23, 1988; age 77. Dr Wiesel was a 1936 graduate of the State University of New York Health Science Center at Brooklyn. Dr Wiesel was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the New York State Society of Internal Medicine, the American Rheumatism Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Sabbo Woldring, MD, Schenectady. Died March 4, 1988; age 68. Dr Woldring was a 1946 graduate of Medische Faculteit Rijksuniverteit to Groningen, Groningen, Netherlands. Dr Woldring was a Fellow of the American Academy of Family Practice, a Diplomate of the American Board of Family Practice, and a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Naum Wortman, MD, Rockville Centre. Died April 7, 1988; age 74. He was a 1938 graduate of Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. Dr Wortman was a Fellow of the American Academy of Family Practice and a member of the American Geriatrics Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Benjamin Zohn, MD, Brooklyn. Died April 12, 1988; age 87. Dr Zohn was a 1925 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics and the American Board of Allergy and Immunology. His memberships included the American Academy of Allergy, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

FOR MY
PATIENTS
1/1988

TICK BITES — WHAT TO DO AND WHAT NOT TO DO

Dear Patient,

I'm sure you are hearing a lot about Lyme Disease and how it is caused by a bacteria spread through the bite of a tiny deer tick ***(Ixodes)***. Between early May and the end of August these ticks are especially dangerous and plentiful in certain areas in New York State: Suffolk County, Westchester County, Putnam County, Ulster, Dutchess, and Orange Counties.

But they are also present in other wooded areas of the state even where deer are not found. These ticks can be carried to many areas by attaching to field mice or other animals. If not treated early, Lyme Disease can seriously affect your joints, heart, or nervous system. Therefore, I urge you to keep the following facts in mind whenever you take a spring or summer stroll in the tall grass or underbrush.

THE TICK

Ixodes is an oval-shaped, eight-legged, dark red and black bug about the size of a pinhead. Unable to fly or jump, it lays in waiting in low vegetation of grasslands or wooded areas. It does not cause any pain, swelling, or itching after it silently attaches to the skin of passing animals or humans. If it bites, it gradually swells with blood of the animal or human and appears much larger in size. After several hours or days of attachment, it may fall off without you ever noticing it.

PREVENTIVE MEASURES

Like a good scout, you should be prepared for ticks whenever you walk in parks and woods between May and August. This is what you can do to protect yourself:

- Apply a good insect repellent that contains ***DEET*** (see label) to your pants, socks, shoes, and shirt sleeves according to the label directions. Also apply the repellent to the back of your neck, the back of your hands and wrists, and other exposed body parts.
- Wear a long-sleeved shirt, tape the cuffs around your wrists, and tuck the shirt into long pants. Tuck the cuffs of the pants into the socks, shoes, or boots. Don't wear open-top boots for hiking. Wear light-colored clothing which has tightly woven or hard-pressed finish. In that way, ticks can be spotted more easily and will not stick to the clothes.
- Watch for warning signs along nature trails and in parks indicating the presence of ticks in the area. Walk near the center of well-cleared paths, trails, and roads whenever possible. Carry a large, white bath towel when hiking in the woods. Drag it along the path frequently during the trip. If ticks are plentiful in the area, you will see many of them as black specks on the towel.
- Don't forget to do a body search as soon as you leave the woodlands and a more complete body inspection at home that night of all the hikers (including the dog). Then take a shower, and wash the removed clothing.

BODY SEARCH

After you, your friends (including your dog), and family members walk in the woods or overgrown fields during the tick-biting season careful body inspection should be done. Examine yourself and the others for any signs of ticks immediately. Look especially around the ankles, legs, hands, back of the head and scalp, and other parts which may have been in contact with shrubs and underbrush. Repeat the body search in the home at night under good light.

TICK REMOVAL

If you find a tick on the skin, it must be removed quickly with special precautions:

- Don't remove it with your bare fingers. Use clean, fine-tipped tweezers (such as for those used for eyebrows) to grasp it firmly around its front (mouth) part and steadily pull it off. If you have no tweezers, use facial tissue or a plastic glove for the removal.
- If possible, take a magnifying glass to examine the attachment area to see if any part of the body still remains imbedded. If so, remove that also with the tweezers. Clean the skin area of attachment with an antiseptic solution or soap and water. Wash your fingers if they were used for the removal.
- Place the removed tick—whether dead or alive—in a small, closed container. Call me or the local Health Department to find out where to bring the tick for identification. If it proves to be a tick other than ***ixodes***, it cannot be a carrier of the Lyme Disease bacteria. If it is the ***Dermacentor*** tick ("dog tick"), it may be a carrier of the Rocky Mountain Spotted Fever germ. I'll give you further advice when I learn which type of tick it is.

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

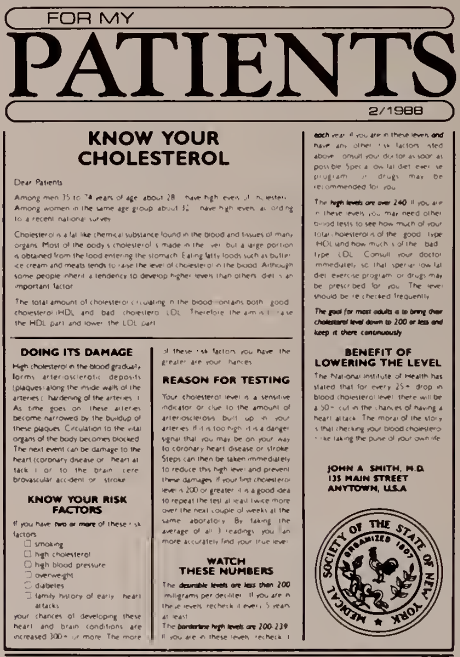
Office Handouts for Your Patients

This issue of the *New York State Journal of Medicine* contains a special additional item, "For My Patients." This feature will be run in future issues as well.

Use this new feature to keep your patients informed about timely issues concerning diseases, treatments and health maintenance. Simply make photocopies of the page and distribute them to your patients with your newsletter or other correspondence, or place them in your reception area for your patients to read and take home.

MSSNY and the *New York State Journal of Medicine* have waived the copyright for these handouts, so you may duplicate them without permission.

As an added feature, MSSNY will personalize this form and make copies for you, at a nominal charge. See the example below for how these forms would look with your name and address professionally printed on them. Use the order form below, and we'll ship the personalized forms to you as soon as possible.



FOR MY

PATIENTS

2/1988

KNOW YOUR CHOLESTEROL

Dear Patients

Among men 35 to 74 years of age about 28% have high levels of cholesterol. Among women in the same age group about 32% have high levels according to a recent national survey.

Cholesterol is a fat like chemical substance found in the blood and tissues of many organs. Most of the body's cholesterol is made in the liver but a large portion is obtained from the food entering the stomach. Eating fatty foods such as butter, ice cream and meats tends to raise the level of cholesterol in the blood. Although some people inherit a tendency to develop higher levels than others, diet is an important factor.

The total amount of cholesterol circulating in the blood contains both "good" cholesterol (HDL) and "bad" cholesterol (LDL). Therefore the aim is to raise the HDL part and lower the LDL part.

DOING ITS DAMAGE

High cholesterol in the blood gradually forms arteriosclerotic deposits (plaques) along the inside walls of the arteries ("hardening of the arteries"). As time goes on, these arteries become narrowed by the buildup of these plaques. Circulation to the vital organs of the body becomes blocked. The next event can be damage to the heart (coronary disease or "heart attack") or to the brain (cerebrovascular accident or "stroke").

KNOW YOUR RISK FACTORS

If you have **two or more** of these risk factors

- ☐ smoking
- ☐ high cholesterol
- ☐ high blood pressure
- ☐ overweight
- ☐ diabetes
- ☐ family history of early heart attacks

your chances of developing these heart and brain conditions are increased 300% or more. The more of these risk factors you have, the greater are your chances.

REASON FOR TESTING

Your cholesterol level is a sensitive indicator or clue to the amount of arteriosclerosis built up in your arteries. If it is too high it is a danger signal that you may be on your way to coronary heart disease or stroke. Steps can then be taken immediately to reduce this high level and prevent these damages. If your first cholesterol level is 200 or greater, it is a good idea to repeat the test at least twice more over the next couple of weeks at the same laboratory. By taking the average of all 3 readings, you can more accurately find your true level.

WATCH THESE NUMBERS

The ***desirable levels are less than 200*** milligrams per deciliter. If you are in these levels, recheck it every 5 years at least.

The ***borderline high levels are 200-239***. If you are in these levels, recheck it ***each*** year. If you are in these levels ***and*** have any other risk factors listed above, consult your doctor as soon as possible. Special low fat diet, exercise program, or drugs may be recommended for you.

The ***high levels are over 240***. If you are in these levels you may need other blood tests to see how much of your total cholesterol is of the "good" type (HDL) and how much is of the "bad" type (LDL). Consult your doctor immediately so that special low fat diet, exercise program or drugs may be prescribed for you. The level should be re-checked frequently.

The goal for most adults is to bring their cholesterol level down to 200 or less and keep it there continuously.

BENEFIT OF LOWERING THE LEVEL

The National Institute of Health has stated that for every 25% drop in blood cholesterol level, there will be a 50% cut in the chances of having a heart attack. The moral of the story is that checking your blood cholesterol is like taking the pulse of your own life.

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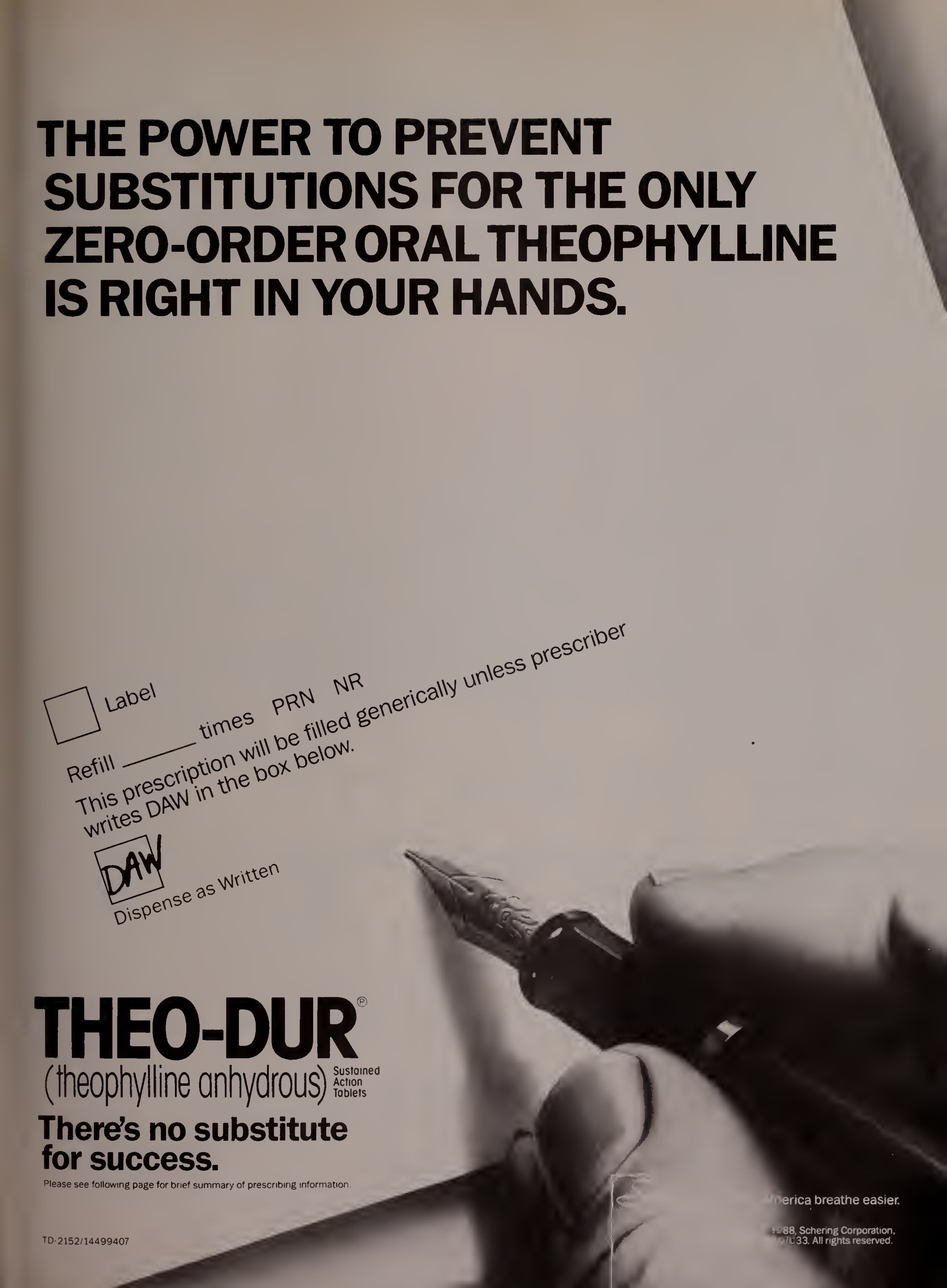

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DAW

Dispense as Written

THEO-DUR®

(theophylline anhydrous) Sustained Action Tablets

There's no substitute for success.

Please see following page for brief summary of prescribing information.

merica breathe easier.

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THEO-DUR®

THEOPHYLLINE (Anhydrous)
Sustained Action Tablets

INDICATIONS. THEO-DUR is indicated for relief and/or prevention of symptoms of asthma and for reversible bronchospasm associated with chronic bronchitis and emphysema

CONTRAINDICATIONS: THEO-DUR is contraindicated in individuals who have shown hypersensitivity to theophylline or any of the tablet components

WARNINGS: Status asthmaticus should be considered a medical emergency and is defined as that degree of bronchospasm which is not rapidly responsive to usual doses of conventional bronchodilators Optimal therapy for such patients frequently requires both *additional medication* parenterally administered. and close *monitoring*. preferably in an intensive care setting

Although increasing the dose of theophylline may bring about relief. such treatment may be associated with toxicity The likelihood of such toxicity developing increases significantly when the serum theophylline concentration exceeds 20 mcg/ml. Therefore. determination of serum theophylline levels is recommended to assure maximal benefit without excessive risk

Serum levels above 20 mcg/ml are rarely found after appropriate administration of recommended doses However. in individuals in whom theophylline plasma clearance is reduced *for any reason*. even conventional doses may result in increased serum levels and potential toxicity Reduced theophylline clearance has been documented in the following readily identifiable groups 1) patients with impaired renal or liver function. 2) patients over 55 years of age. particularly males and those with chronic lung disease. 3) those with cardiac failure from any cause. 4) neonates. and 5) those patients taking certain drugs (macrolide antibiotics and cimetidine). Decreased clearance of theophylline may be associated with either influenza immunization or active infection with influenza

Reduction of dosage and laboratory monitoring is especially appropriate in the above individuals Less serious signs of theophylline toxicity (i e nausea and restlessness) may occur frequently when initiating therapy. but are usually transient when such signs are persistent during maintenance therapy. they are often associated with serum concentrations above 20 mcg/ml. Unfortunately. however. serious side effects such as ventricular arrhythmias. convulsions or even death may appear as the first sign of toxicity without any previous warning Stated differently *serious toxicity is not reliably preceded by less severe side effects*

Many patients who require theophylline may exhibit tachycardia due to their underlying disease process so that the cause/effect relationship to elevated serum theophylline concentrations may not be appreciated

Theophylline products may cause dysrhythmia and/or worsen pre-existing arrhythmias and any significant change in rate and/or rhythm warrants monitoring and further investigation

The occurrence of arrhythmias and sudden death (with histological evidence of necrosis of the myocardium) has been recorded in laboratory animals (minipigs. rodents and dogs) when theophylline and beta agonists were administered concomitantly. although not when either was administered alone The significance of these findings when applied to human usage is currently unknown

PRECAUTIONS: THEO-DUR TABLETS SHOULD NOT BE CHEWED OR CRUSHED

General: Theophylline half-life is shorter in smokers than in non-smokers Therefore. smokers may require larger or more frequent doses Morphine and curare should be used with caution in patients with airway obstruction as they may suppress respiration and stimulate histamine release Alternative drugs should be used when possible. Theophylline should not be administered concurrently with other xanthine medications. Use with caution in patients with severe cardiac disease. severe hypoxemia. hypertension. hyperthyroidism. acute myocardial injury. cor pulmonale. congestive heart failure. liver disease. in the elderly (especially males) and in neonates. In particular. great caution should be used in giving theophylline to patients with congestive heart failure Frequently. such patients have markedly prolonged theophylline serum levels with theophylline persisting in serum for long periods following discontinuation of the drug Individuals who are rapid metabolizers of theophylline. such as the young. smokers. and some non-smoking adults. may not be suitable candidates for once-daily dosing. These individuals will generally need to be dosed at 12 hour or sometimes 8 hour intervals Such patients may exhibit symptoms of bronchospasm near the end of a dosing interval. or may have wider peak-to-trough differences than desired

Use theophylline cautiously in patients with history of peptic ulcer Theophylline may occasionally act as a local irritant to the G I tract although gastrointestinal symptoms are more commonly centrally mediated and associated with serum drug concentrations over 20 mcg/ml

Information for Patients: The physician should reinforce the importance of taking only the prescribed dose and time interval between doses THEO-DUR tablets should not be chewed or crushed When dosing THEO-DUR on a once daily (q24h) basis. tablets should be taken whole and not split. As with any controlled-release theophylline product. the patient should alert the physician if symptoms occur repeatedly. especially near the end of the dosing interval.

DRUG INTERACTIONS: **Drug-Drug**: Toxic synergism with ephedrine has been documented and may occur with some other sympathomimetic bronchodilators In addition. the following drug interactions have been demonstrated

Drug	Effect
Theophylline with lithium carbonate	Increased excretion of lithium carbonate
Theophylline with propranolol	Antagonism of propranolol effect
Theophylline with cimetidine	Increased theophylline blood levels
Theophylline with troleandomycin. erythromycin	Increased theophylline blood levels

Drug-Food. *THEO-DUR 100 mg Sustained Action Tablets* have not been adequately studied to determine whether their bioavailability is altered when given with food Available data suggest that drug administration at the time of food ingestion may influence the absorption characteristics of theophylline controlled-release products resulting in serum values different from those found after administration in the fasting state

A drug-food effect if any. would likely have its greatest clinical significance when high theophylline serum levels are being maintained and/or when large single doses (greater than 13 mg/kg or 900 mg) of a controlled-release theophylline product are given

THEO-DUR (200. 300. and 450 mg) Sustained Action Tablets The rate and extent of absorption of theophylline from THEO-DUR 200 mg 300 mg. and 450 mg tablets when administered fasting or immediately after a moderately high fat content breakfast is similar

Drug-Laboratory Test Interactions: When plasma levels of theophylline are measured by spectrophotometric methods. coffee. tea. cola beverages. chocolate. and acetaminophen contribute falsely high values

Carcinogenesis, Mutagenesis, and Impairment of Fertility: Long-term animal studies have not been performed to evaluate the carcinogenic potential mutagenic potential. or the effect on fertility of xanthine compounds

Pregnancy: Category C—Animal reproduction studies have not been conducted with theophylline It is not known whether theophylline can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Xanthines should be given to a pregnant woman only if clearly needed

Nursing Mothers: It has been reported that theophylline distributes readily into breast milk and may cause adverse effects in the infant Caution must be used if prescribing xanthine to a mother who is nursing. taking into account the risk-benefit of this therapy

Pediatric Use: Safety and effectiveness of THEO-DUR administered

1 Every 24 hours in children under 12 years of age have not been established
2 Every 12 hours in children under 6 years of age. have not been established

ADVERSE REACTIONS: The most consistent adverse reactions are usually due to overdose and are

1 *Gastrointestinal* nausea. vomiting. epigastric pain. hematemesis. diarrhea
2 *Central nervous system* headaches. irritability. restlessness. insomnia. reflex hyperexcitability. muscle twitching. clonic and tonic generalized convulsions
3 *Cardiovascular* palpitation. tachycardia extrasystoles. flushing. hypotension. circulatory failure ventricular arrhythmias
4 *Respiratory* tachypnea
5 *Renal* albuminuria. increased excretion of renal tubular and red blood cells. potentiation of diuresis
6 *Other* rash hyperglycemia and inappropriate ADH syndrome

OVERDOSAGE: **Management**: If potential oral overdose is established and seizure has not occurred

A Induce vomiting
B Administer a cathartic (this is particularly important if sustained-release preparations have been taken)
C Administer activated charcoal

If patient is having a seizure

A Establish an airway
B Administer oxygen
C Treat the seizure with intravenous diazepam. 0.1 to 0.3 mg/kg up to 10 mg
D Monitor vital signs maintain blood pressure and provide adequate hydration

Post Seizure Coma:

A Maintain airway and oxygenation
B If a result of oral medication follow above recommendations to prevent absorption of the drug but intubation and lavage will have to be performed instead of inducing emesis and the cathartic and charcoal will need to be introduced via a large bore gastric lavage tube
C Continue to provide full supportive care and adequate hydration while waiting for drug to be metabolized In general the drug is metabolized sufficiently rapid so as not to warrant consideration of dialysis. however if serum levels exceed 50 mcg/ml charcoal hemoperfusion may be indicated

CAUTION Federal law prohibits dispensing without prescription For full prescribing information see package insert

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(*continued from p 9A*)

fice of Continuing Medical Education, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6675.

SOUTH CAROLINA

August 4–7. **Otolaryngology for the Practicing Pediatrician.** 6 Cat 1 Credits. Mariner's Inn, Hilton Head Island. Contact: Debbie Shealy, South Carolina Chapter American Academy of Pediatrics, PO Box 11188, Columbia, SC 29211. Tel: (803) 798-6207.

TEXAS

August 28–Sept 2. **XIII World Conference on Health Education.** Houston. Contact: Dr Judith Ottoson, Executive Director, United States Host Committee, Inc, PO Box 20186, Suite 902, Houston, TX 77225. Tel: (713) 792-8540.

WEST VIRGINIA

August 5–7. **Venous Insufficiency of the Lower Extremity.** Davis. *August 12–14.* **Physician Update on Cancer, AIDS and Liver Disease.** Davis. *August 19–21.* **Advances in Cardiology.** Davis. *August 26–28.* **Anesthesia Update.** Davis. Contact: Frederick A. Reichle, MD, Presbyterian-University of Pennsylvania Medical Center, 39th and Market Sts, Philadelphia, PA 19104. Tel: (215) 387-3685.

AROUND THE WORLD

CARIBBEAN ISLANDS

August 8–12. **Family Medicine Review—Summer in St Maarten.** 26 Cat 1 Credits. Mullet Bay Resort and Casino, The Netherlands Antilles. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773. ☐

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8 mEq (600 mg)

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*Based on worldwide sales data on file, CIBA Pharmaceutical Company.
Capsule or tablet slow-release potassium chloride preparations should be reserved for patients who cannot tolerate, refuse to take, or have compliance problems with liquid or effervescent potassium preparations because of reports of intestinal and gastric ulceration and bleeding with slow-release KCl preparations.

Before prescribing, please consult Brief Prescribing Information on next page.

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For good reasons

- ☐ **It works**—a 12-year record of efficacy[1]
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- ☐ **It's comparable to 10 mEq**—in low-dosage supplementation[3]†
- ☐ **It's economical**—less expensive than all other leading KCl slow-release supplements on a per tablet cost to the patient[1]

Slow-K®
potassium chloride
slow-release tablets 8 mEq (600 mg)

For patients who can't or won't tolerate liquid KCl.

*The most common adverse reactions to potassium salts are gastrointestinal side effects.

†Pooled mean serum potassium following oral administration of 30 mEq K-Tab compared to 24 mEq Slow-K in diuretic-treated hypertensives (n = 20) over 8 weeks.

C I B A

References: **1**. Data on file, CIBA Pharmaceutical Company. **2**. Skoutakis VA Acchiardo SR, Wojciechowski NJ, et al: Liquid and solid potassium chloride Bioavailability and safety *Pharmacotherapy* 1980:4(6):392-397. **3**. Skoutakis VA, Carter CA, Acchiardo SR Therapeutic assessment of Slow-K and K-Tab potassium chloride formulations in hypertensive patients treated with thiazide diuretics. *Drug Intell Clin Pharm* 1987:21 436-440

Slow-K®
potassium chloride USP
Slow-Release Tablets
8 mEq (600 mg)

BRIEF SUMMARY (FOR FULL PRESCRIBING INFORMATION SEE PACKAGE INSERT)

INDICATIONS AND USAGE

BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS.

1 For therapeutic use in patients with hypokalemia with or without metabolic alkalosis, in digitalis intoxication and in patients with hypokalemic familial periodic paralysis.

2 For prevention of potassium depletion when the dietary intake of potassium is inadequate in the following conditions patients receiving digitalis and diuretics for congestive heart failure, hepatic cirrhosis with ascites, states of aldosterone excess with normal renal function, potassium-losing nephropathy; and certain diarrheal states

3 The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern Serum potassium should be checked periodically however and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases In more severe cases supplementation with potassium salts may be indicated

CONTRAINDICATIONS

Potassium supplements are contraindicated in patients with hyperkalemia, since a further increase in serum potassium concentration in such patients can produce cardiac arrest Hyperkalemia may complicate any of the following conditions chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns adrenal insufficiency or the administration of a potassium-sparing diuretic (e g spironolactone triamterene) (see OVERDOSAGE)

All solid dosage forms of potassium supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract In these instances, potassium supplementation should be with a liquid preparation Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to an enlarged left atrium

WARNINGS

Hyperkalemia (See OVERDOSAGE).

In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally Potentially fatal hyperkalemia can develop rapidly and be asymptomatic

The use of potassium salts in patients with chronic renal disease or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment

Interaction With Potassium-Sparing Diuretics

Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e g , spironolactone or triamterene), since the simultaneous administration of these agents can produce severe hyperkalemia

Gastrointestinal Lesions

Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage, or perforation Slow-K is a wax-matrix tablet formulated to provide a controlled rate of release of potassium chloride and thus to minimize the possibility of a high local concentration of potassium ion near the bowel wall While the reported frequency of small-bowel lesions is much less with wax-matrix tablets (less than one per 100,000 patient-years) than with enteric-coated potassium chloride tablets (40-50 per 100,000 patient-years) cases associated with wax-matrix tablets have been reported both in foreign countries and in the United States In addition, perhaps because the wax-matrix preparations are not enteric-coated and release potassium in the stomach, there have been reports of upper gastrointestinal bleeding associated with these products. The total number of gastrointestinal lesions remains approximately one per 100,000 patient-years Slow-K should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs.

Metabolic Acidosis

Hypokalemia in patients with metabolic *acidosis* should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, or potassium acetate

PRECAUTIONS

General:

The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis *per* se can produce hypokalemia in the absence of a deficit in total body potassium, while acute acidosis *per* se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium

Information for Patients

Physicians should consider reminding the patient of the following

To take each dose without crushing, chewing, or sucking the tablets

To take this medicine only as directed. This is especially important if the patient is also taking both diuretics and digitalis preparations

To check with the physician if there is trouble swallowing tablets or if the tablets seem to stick in the throat

To check with the doctor at once if tarry stools or other evidence of gastrointestinal bleeding is noticed

Laboratory Tests

Regular serum potassium determinations are recommended In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis

Drug Interactions

Potassium-sparing diuretics see WARNINGS

Carcinogenesis, Mutagenesis, Impairment of Fertility

Long-term carcinogenicity studies in animals have not been performed

Pregnancy Category C

Animal reproduction studies have not been conducted with Slow-K It is also not known whether Slow-K can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Slow-K should be given to a pregnant woman only if clearly needed

Nursing Mothers

The normal potassium ion content of human milk is about 13 mEq/L It is not known if Slow-K has an effect on this content Caution should be exercised when Slow-K is administered to a nursing woman

Pediatric Use

Safety and effectiveness in children have not been established

ADVERSE REACTIONS

One of the most severe adverse effects is hyperkalemia (see CONTRAINDICATIONS, WARNINGS, and OVERDOSAGE) There also have been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see CONTRAINDICATIONS and WARNINGS), other factors known to be associated with such conditions were present in many of these patients

The most common adverse reactions to oral potassium salts are nausea, vomiting abdominal discomfort, and diarrhea These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose

Skin rash has been reported rarely

OVERDOSAGE

The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see CONTRAINDICATIONS and WARNINGS) It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration (6.5-8 0 mEq/L) and characteristic electrocardiographic changes (peaking of T waves loss of P wave, depression of S-T segment, and prolongation of the Q-T interval) Late manifestations include muscle paralysis and cardiovascular collapse from cardiac arrest (9-12 mEq/L).

Treatment measures for hyperkalemia include the following (1) elimination of foods and medications containing potassium and of potassium-sparing diuretics, (2) intravenous administration of 300-500 ml/hr of 10% dextrose solution containing 10-20 units of insulin per 1,000 ml, (3) correction of acidosis, if present, with intravenous sodium bicarbonate, (4) use of exchange resins, hemodialysis, or peritoneal dialysis.

In treating hyperkalemia in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity

DOSAGE AND ADMINISTRATION

The usual dietary intake of potassium by the average adult is 40-80 mEq per day Potassium depletion sufficient to cause hypokalemia usually requires the loss of 200 or more mEq of potassium from the total body store Dosage must be adjusted to the individual needs of each patient but is typically in the range of 20 mEq per day for the prevention of hypokalemia to 40-100 mEq or more per day for the treatment of potassium depletion Large numbers of tablets should be given in divided doses

Note: Slow-K slow-release tablets must be swallowed whole and never crushed, chewed, or sucked

HOW SUPPLIED

Tablets — 600 mg of potassium chloride (equivalent to 8 mEq) round, buff colored, sugar-coated (imprinted Slow-K)

Bottles of 100	NDC 0083-0165-30
Bottles of 1000	NDC 0083-0165-40
Consumer Pack — One Unit	
12 Bottles — 100 tablets each	NDC 0083-0165-65
Accu-Pak® Unit Dose (Blister pack)	
Box of 100 (strips of 10)	NDC 0083-0165-32

Do not store above 86°F (30°C) Protect from moisture Protect from light

Dispense in tight, light-resistant container (USP).

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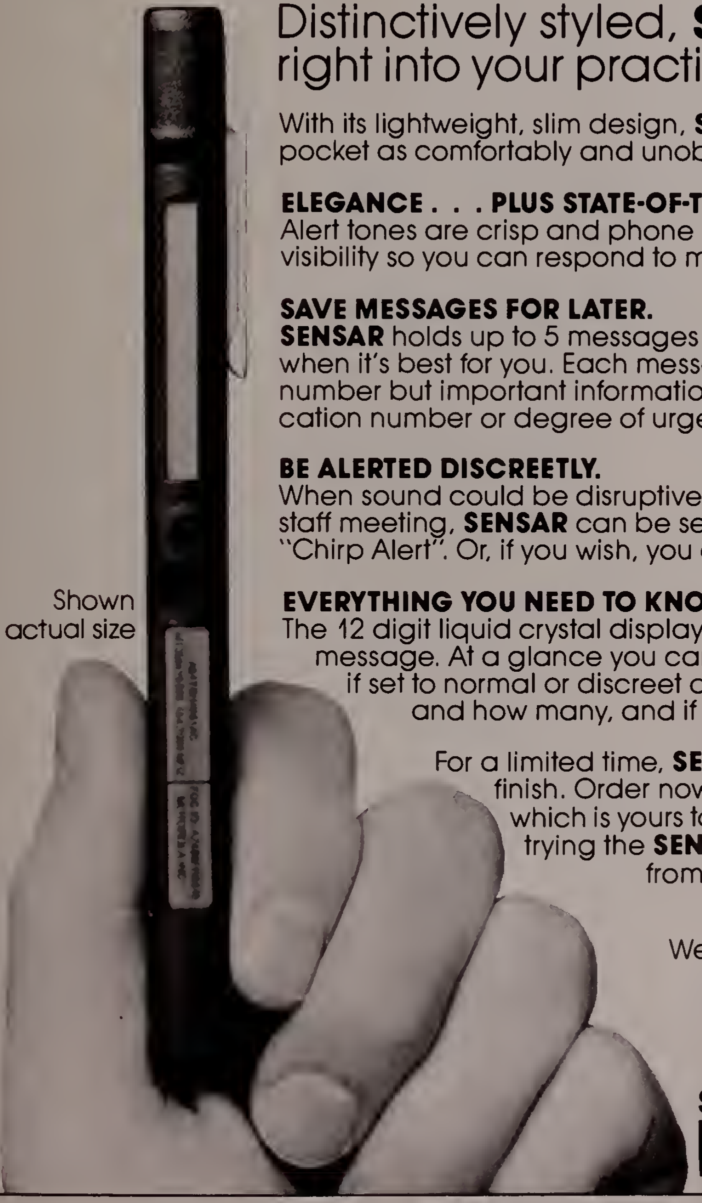

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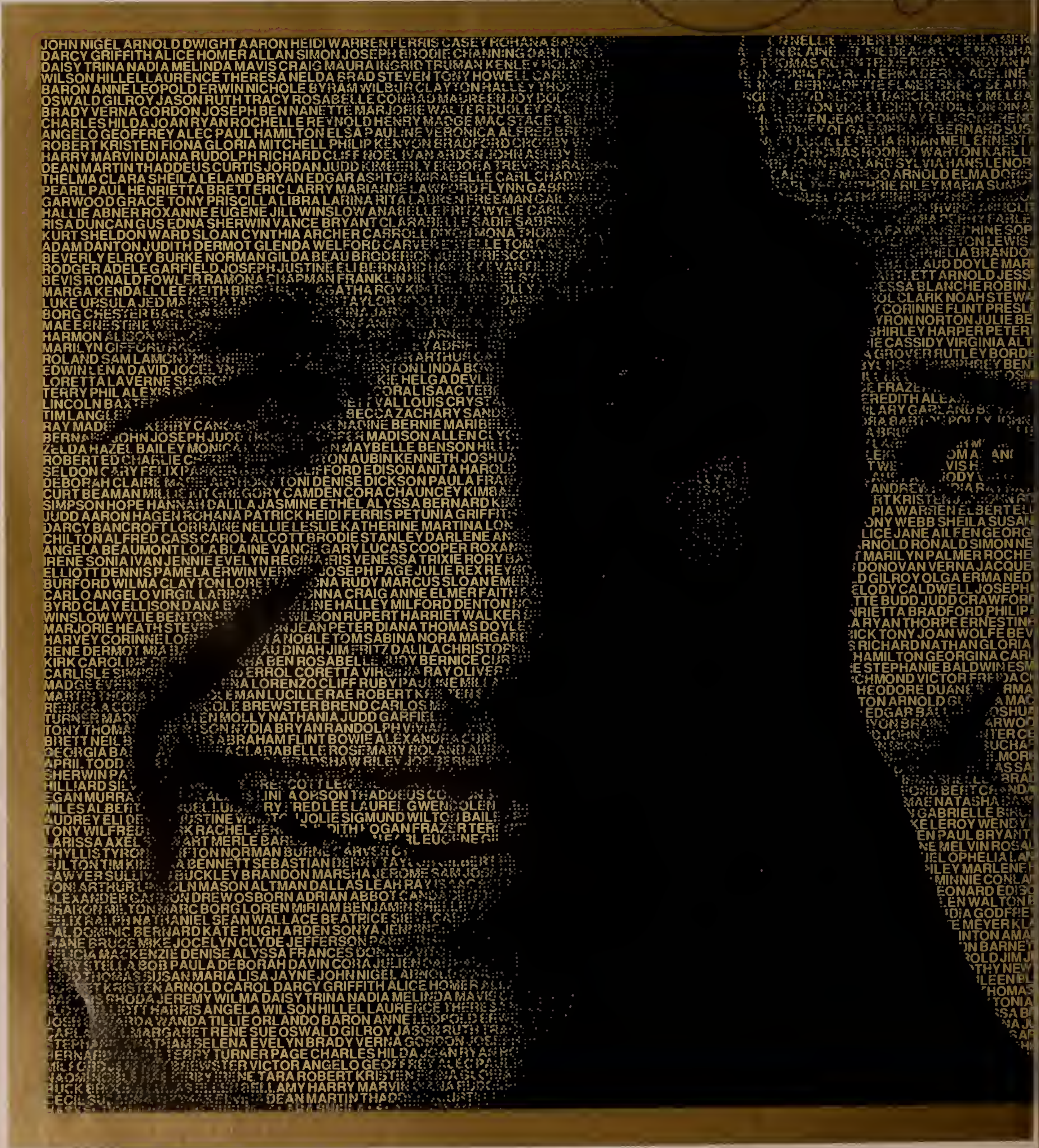
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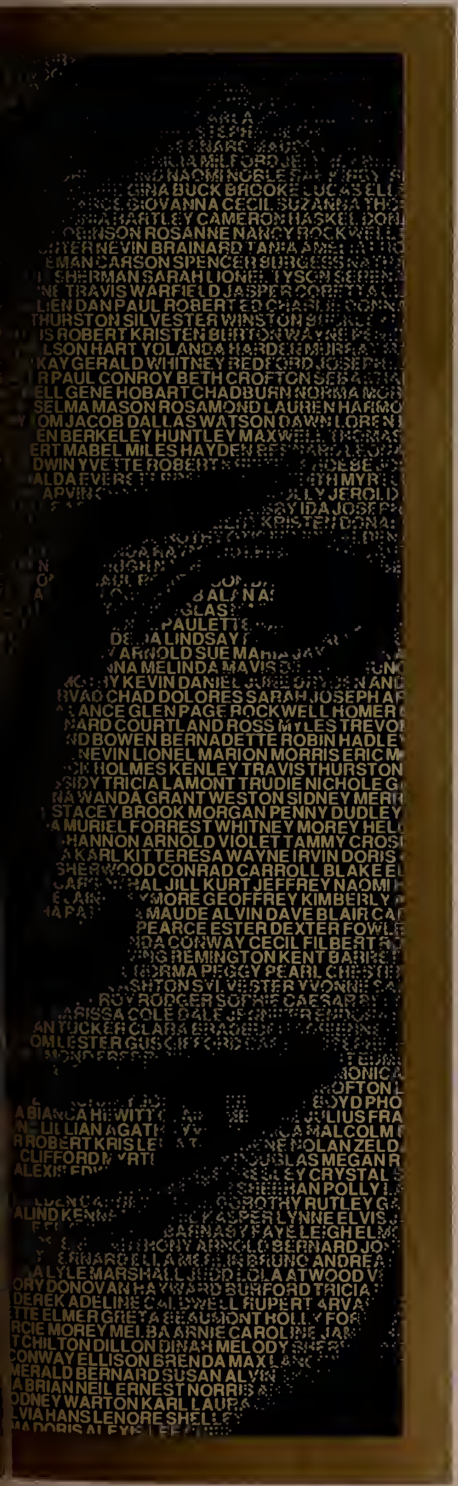

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In an unprecedented program,[1] over 9,000 physicians nationwide profiled 234,505 patients receiving INDERAL LA for *hypertension*. In a subsequent survey, 4,120 of these participating physicians gave us their views on the performance of INDERAL LA.

The consensus†:

Nearly three out of four physicians responding to the survey consider INDERAL LA their preferred beta blocker for hypertension—preferring to prescribe it before ACE inhibitors, calcium channel blockers, and alpha blockers. Forty-nine percent of these physicians also preferred to prescribe INDERAL LA before diuretics.

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(PROPRANOLOL HCl)

LONG ACTING CAPSULES
60, 80, 120, 160 mg

The one you know best keeps looking better

Please see next page for brief summary of prescribing information.

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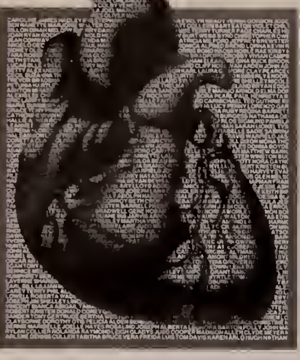

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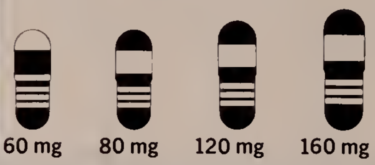

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride. INDERAL LA is available as 60 mg, 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure, and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first-degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected. Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL (propranolol HCl) is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Caution should be exercised when patients receiving a beta blocker are administered a calcium-channel-blocking drug, especially intravenous verapamil, for both agents may depress myocardial contractility or atrioventricular conduction. On rare occasions, the concomitant intravenous use of a beta blocker and verapamil has resulted in serious adverse reactions, especially in patients with severe cardiomyopathy, congestive heart failure, or recent myocardial infarction.

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol.

Ethanol slows the rate of absorption of propranolol.

Phenytoin, *phenobarbitone*, and *rifampin* accelerate propranolol clearance.

Chlorpromazine, when used concomitantly with propranolol, results in increased plasma levels of both drugs.

Antipyrine and *lidocaine* have reduced clearance when used concomitantly with propranolol.

Thyroxine may result in a lower than expected T_3 concentration when used concomitantly with propranolol.

Cimetidine decreases the hepatic metabolism of propranolol, delaying elimination and increasing blood levels.

Theophylline clearance is reduced when used concomitantly with propranolol.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS: INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type.

Central Nervous System: Light-headedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; vivid dreams; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. For immediate formulations, fatigue, lethargy, and vivid dreams appear dose related.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL Tablets to INDERAL LA Capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg-for-mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary, especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—*Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three- to seven-day intervals until optimal response is obtained. Although individual patients may respond at any dosage level, the average optimal dosage appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE—*Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimal migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximal dose, INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of these capsules is a registered trademark of Wyeth-Ayerst Laboratories.

Reference:

1. Data on file, Wyeth-Ayerst Laboratories.

†Based on response of 4,120 participating physicians to questionnaire survey.

7297/188

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Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

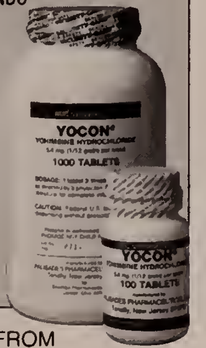
Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85



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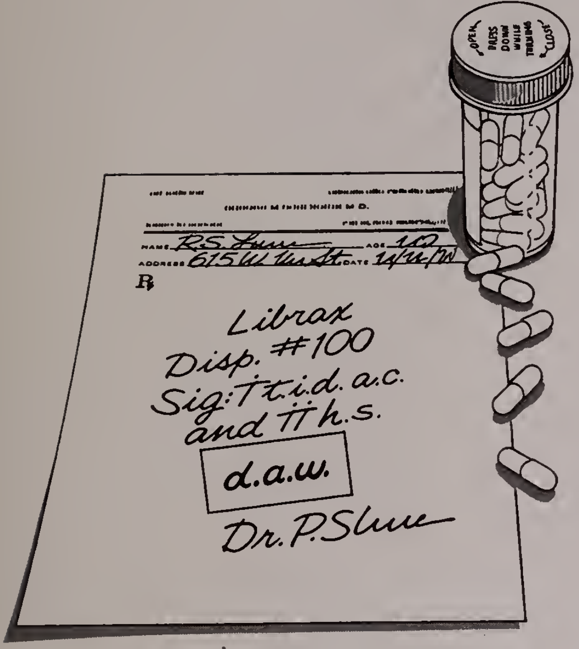

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Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

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Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

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Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975, 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

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1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med*, 141:293-300, 1981.

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Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS:
Special Risk Patients: As with any narcotic analgesic agent, VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture. The usual precautions should be observed and the possibility of respiratory depression should be kept in mind.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly
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The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone.
The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
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Labor and Delivery: As with all narcotics, administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
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Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS:
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients. The antiemetic phenothiazines are useful in suppressing these effects; however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia. Prolonged administration of VICODIN may produce constipation.
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AROUND THE STATE

ALBANY

Sept 22. **Sports Medicine Update: Prevention and Treatment of Fall Sports Injuries.** Desmond Americana. *Sept 28.* **Psychiatry Teaching Day.** Capital District Psychiatric Center. *Sept 28.* **Disorders of Higher Incidence in Women.** Capital District Psychiatric Center. Contact: Diane M. Martin, Albany Medical College, Office of Continuing Medical Education, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

BRONX

Sept 28. **The Real Miami Vice—The Forensic Experience in Dade County, Florida, 1978–88.** Lehman College, Bronx. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 120 Gold Pl, Malverne, NY 11565. Tel: (516) 887-4691.

COOPERSTOWN

Sept 14. **Focus on General Surgery.** 3 Cat I Credits. Basset Hall Conference Center. *Sept 19–20.* **1988 Spine Review Conference.** 13 Cat I Credits. The Otesaga Hotel. Contact: Charlotte Hoag, Medical Education, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326. Tel: (607) 547-3764.

MANHATTAN

Sept 16–17. **The First Annual Combined Manhattan Eye, Ear & Throat Hospital-Edward S. Harkness Eye Institute Ophthalmology Seminar.** Contact: Martha Klapp, Manhattan Eye, Ear & Throat Hospital, 210 East 64th St, New York 10021. Tel: (212) 605-3762.

Sept 29–30. **11th Annual Current Concerns in Adolescent Medicine. A Practical Approach to Current Problems in Adolescents.** New York Hilton. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Schneider Children's Hospital of Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Sept 29–Oct 1. **Viral Oncogenesis and Cell Differentiation: The Contributions of Charlotte Friend.** Sheraton Centre. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

NEW HYDE PARK

Sept 10. **Surgery Oral Board Review Course and Practice Exam.** 8 Cat I Credits. Learning Disabilities Center, Schneider's Children's Hospital of Long Island Jewish Medical Center, New Hyde Park. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

ROCHESTER

Sept 22. **Diabetes: Emerging Concepts and Challenges.** Rochester Hilton. Contact: Nancy E. Fay, RPA-C, Professional Education Committee Co-chairman, American Diabetes Association, PO Box 1037, Syracuse, NY 13201. Tel: (315) 735-0591.

SOUTHAMPTON

Sept 12. **Brand Drugs vs Generic Drugs.** 1 Cat I Credit. Southampton Hospital. Contact: Charlotte Henry-Sullivan, Medical Staff Secretary, Southampton Hospital, 240 Meeting House Lane, Southampton, NY 11968. Tel: (516) 283-2600.

AROUND THE NATION

CALIFORNIA

Sept 9–11. **Fourth Annual Sonography Update and Review Course.** Hanalei Hotel, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

Sept 16–17. **AIDS and the Oncologist.** 13 Cat I Credits. Hotel Nikko, San Francisco. Contact: Extended Pro-

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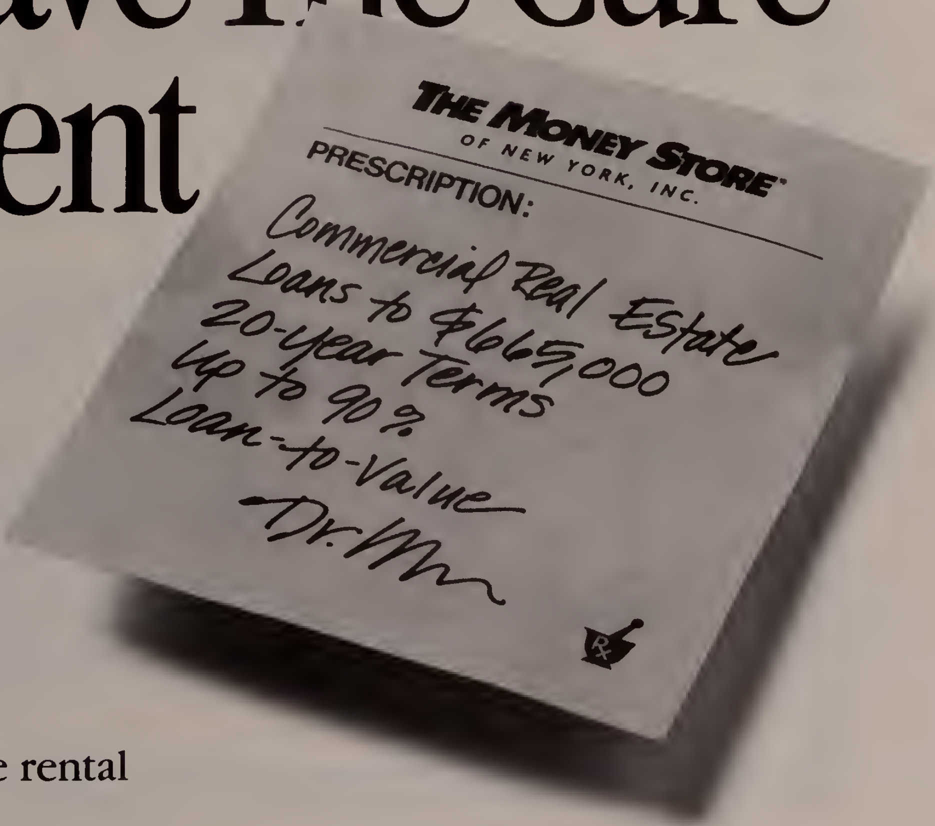

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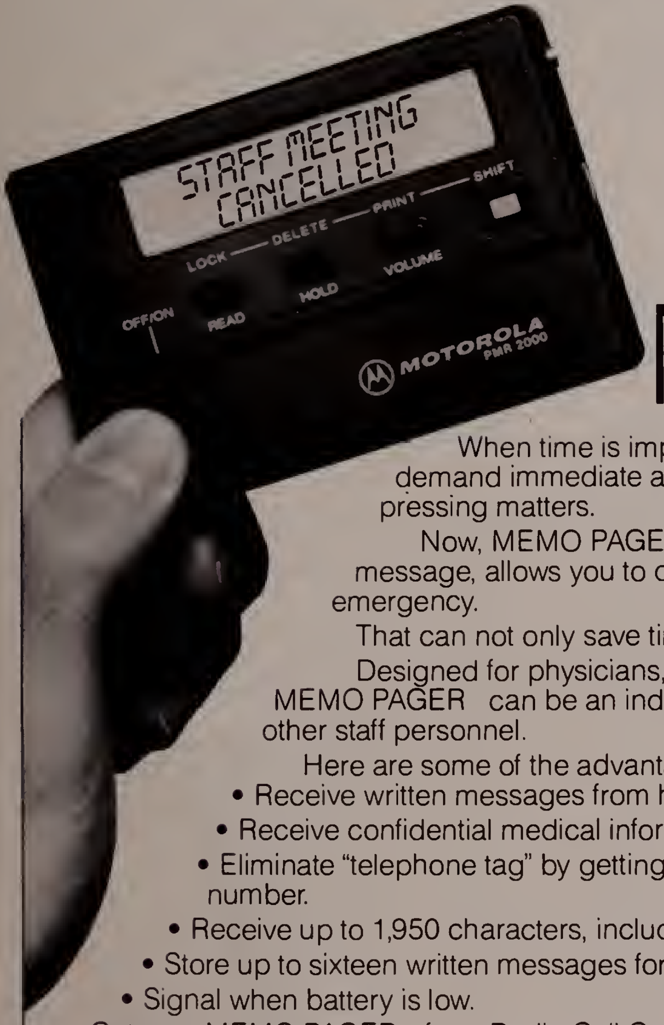

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Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect There was a dose related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high dose males compared to placebo Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups The rate of hepatic carcinoma in the high dose animals was within the historical control limits seen for the strain of mice used The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement compared to concurrent controls, and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive, and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery is not considered evidence of a carcinogenic potential for Axid.

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Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo Among the more common adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common in the nizatidine group A variety of less common events was also reported; it was not possible to determine whether these were caused by nizatidine.

Axid® (nizatidine, Lilly)

Hepatic—Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients possibly or probably related to nizatidine In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L), and in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients All abnormalities were reversible after discontinuation of Axid

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Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal Intravenous LD_{50} values in the rat and mouse were 301 mg/kg and 232 mg/kg respectively

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NEW YORK STATE
JOURNAL OF MEDICINE

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COMMENTARIES

The management of accidental severe hypothermia

The term "submersion hypothermia" is applied when cooling follows acute asphyxia.[1,2] The risk of brain damage is great. Nevertheless, the rapid cooling process in children, in addition to the presence of the mammalian diving reflex, may account for the surprisingly high survival rate. The mammalian diving reflex occurs when facial cooling triggers apnea and circulatory shunting to the heart and brain. This primitive reflex is present in children but not in adults. It may account for numerous reports of children surviving ten to 40 minutes of submersion.[2-4]

The case report of submersion hypothermia in this issue represents that of the oldest survivor of submersion hypothermia without neurologic sequelae.[5] At age 14, the individual's physiologic mechanisms are most similar to those of an adult. Therefore, as the authors discuss, it is unlikely that the mammalian diving reflex explains this patient's survival. Paradoxically, survival is related to a severe hypothermic event concomitant to submersion. This may best be exemplified by the relative outcomes of the two patients discussed in the case report; survival of the hypothermic victim but nonsurvival of the patient whose submersion event was not accompanied by severe hypothermia.

The authors draw attention to two important points in the resuscitation of the submersion hypothermic victim which are equally applicable to any severe hypothermia victim. First and foremost, there are no indicators to appropriately prognosticate survivability from this traumatic insult.[6-8] Secondly, rewarming should never precede control and/or stabilization of cardiopulmonary function.[9-11] These points were originally indicated through less sophisticated early clinical studies.[12-15] Subsequent laboratory investigation by Moss et al[16] on an animal model for accidental severe hypothermia clarified and strengthened these principles. The following is relevant to the management of victims of accidental severe hypothermia.

Cardiopulmonary Resuscitation (CPR). Much of the ability to survive the traumatic insult of severe hypothermia is determined before the resuscitation phase. The hypothermia time is uncontrolled and unavailable to the clinician who manages the resuscitation. The balance between systemic perfusion and metabolism is delicate during injury.[17,18] The cytoprotection of systemic hypothermia will protect against the decreasing cardiovascular function, but there are limits.[12,19] Many of these limits are imposed by the premorbid physiologic state of the patients which primarily encompasses respiratory and cardiovascular compromise.[6,14,20] Even in the best of circumstances, myocardial performance (and therefore systemic perfusion) will cease, as either ventricular fibrillation or asystole occurs, when body temperature falls below 20° Centigrade.[21,22] Before this extreme state, the patient may appear lifeless, with clinicians unable to differentiate between the salvageable and the unsalvageable.[6,7]

It is important to remember, however, that death cannot be determined until body heat has been restored.[6,7] Damage to vital organs during the period of hypothermia may not be determined until increasing body temperature, and an associated increase in metabolic function and need, place functional demands upon organ systems that cannot be met.[17,18] Therefore, all severely hypothermic patients, regardless of extremis, should undergo vigorous resuscitative efforts.

Initial resuscitative efforts for severely hypothermic patients should follow the emergency cardiac care guidelines as they relate to basic life support concepts and implementation upon persons who present with symptoms of cardiopulmonary arrest.[23] These efforts may begin in the emergency room and continue in the intensive care unit. When respiratory failure is present, an airway must be achieved with discretion. Masked ventilation should be performed before intubation. The associated incidence of induced ventricular fibrillation with the stimulus of intubation appears to be dramatically reduced when appropriate oxygenation is achieved.[19,24,25] Even so, manipulation should always be kept to a minimum.[26,27] If asystole or ventricular fibrillation is present, then the need for perfusion support is clear. In their absence, systemic perfusion should be examined by analysis of arterial blood pressure, urine output, and arterial blood gases. Hypothermic persons requiring perfusion support appear to be adequately supported by indirect cardiac compression, either manual or mechanical.[16] Results of numerous reports of successful treatment, in addition to the results of studies on animals by one investigator, support this position. In the rare person for whom indirect cardiac compression is ineffective, direct cardiac compression by left anterolateral thoracotomy may be performed. If partial cardiac bypass is to be used for rewarming, then this system will replace cardiac compression as soon as the patient is moved to the operating room and vascular canulation is performed. Chemical and electrical cardioversion may be attempted for ventricular fibrillation, but the probability of success within the severely hypothermic temperature range is unlikely.[28,29]

Routine administration of steroids and antibiotics is not warranted. Their specific use should be dictated by either the premorbid history or the resultant workup.[13,15,25] A Foley catheter, arterial line, peripheral venous catheter, electrocardiogram leads, and central or pulmonary artery catheter, or both, should all be placed as soon as possible. During cardiac arrest, however, the placement of a pulmonary artery catheter by flow direction will not be possible. Although recordings of pulmonary artery pressure have been useful in an animal model, the lack of similar studies in humans, combined with the knowledge that the catheter may induce ventricular arrhythmia, may preclude its use under these circumstances. In the absence of a pulmonary artery catheter, intravascular measurements can be inferred from the recordings of central venous pressure. Measurements of central venous pressure will be useful in guiding appropriate intravascular replacement with physiologic solutions, the rewarming of which may hasten the systemic rewarming process. Routine blood and urine tests should be performed, including serum amylase, with particular attention to and correction of any abnormalities in serum pH, hemoglobin, potassium, or glucose concentration levels. Lastly, external evidence of trauma should be searched for, with temporary measures such as pressure bandaging and splinting applied.

Rewarming. Rewarming measures should begin only after cardiopulmonary resuscitation is effectively underway. Increasing body temperature before this will only increase metabolic demands without increasing supply, leading to an increased likelihood of infarction.[9-11] In addition, individual rewarming methods may induce physiologic effects related to the mode of heat exchange.[9,14,18] Although it now appears that these effects need not preclude the successful use of a variety of rewarming methods, success depends on both understanding these phenomena and keeping them to a minimum, which are skills executed at a variety of competence levels depending on the institution.[16]

Active external rewarming with the fluid circulated blanket requires more time than core rewarming by partial cardiac bypass or peritoneal dialysis.[16,21] The occurrence of peripheral vasodilatation leads to the intermittent return of sequestered, cold, acidotic blood to the central circulation.[30,31] Associated changes in left ventricular filling volume and arterial pH must be detected through more frequent measurements of arterial blood gases and direct or indirect measurements of left ventricular filling volumes; through measurements of pulmonary artery or central venous catheter pressure; and with compensation through intravenous infusion of fluid and sodium bicarbonate.[16] These changes should be closely observed even well above severe hypothermia range, as these same shifts of fluid have occurred when body temperature is above 30°C. Rhythm disturbances should also be continuously monitored, the recurrence of ventricular fibrillation after successful cardioversion in association with the previous fluid shifts having been noted. In these instances, immediate infusion of sodium bicarbonate and repeated cardioversion are necessary for the continuance of a successful resuscitation. The increased rewarming time associated with active external rewarming with the fluid circulated blanket will also necessitate longer periods of cardiopulmonary resuscitation (CPR). Although CPR was successfully performed for these longer periods using a mechanical device in an animal model, prolonged manual CPR by clinicians may be exhaustive and lead to suboptimum perfusion support.

Partial cardiac bypass rewarming provides concomitant perfusion support throughout the rewarming period and obviates the need for the burdensome performance of manual CPR. However, researchers in one study found that after the initial rewarming cycle, core temperature dramatically declined, necessitating an average of two rewarming cycles per subject.[16] Similar effects have been described with clinical resuscitations.[32] This may be due to the delayed return of peripheral cold blood as the vascular compartments gradually equalize in temperature. The significance of this is two-fold. To ignore this phenomenon would set the stage for the transfer of a presumably rewarmed patient to the intensive care unit, only to find the patient hypothermic upon arrival. Also, after adding the total time required for several rewarming cycles, partial cardiac bypass rewarming may be no faster than peritoneal dialysis (a finding recorded by some researchers).[16] Additional modifications to multiple rewarming cycles, aimed at avoiding this delayed hypothermia, may include prolonging the rewarming cycle, or using concurrent external rewarming. Both methods could help thermally equilibrate peripheral and core vascular compartments.

Peritoneal dialysis rewarming can be as rapid as partial cardiac bypass rewarming and four times as rapid as active external rewarming.[16] In addition, because peritoneal dialysis is a core rewarming method, significant arterial pH and fluid shifts remain absent (precluded through the avoidance of peripheral vasodilatation seen with external rewarming), allowing a more liberal monitoring regimen than that applied during active external rewarming. Rapid rewarming has been achieved using dialysate warmed to 55°C and two peritoneal dialysis catheters to maintain a continuous flow.[16] When dialysate of 55°C was allowed to flow from a six-foot intravenous pole, through six feet of intravenous tubing, intraperitoneal temperature did not exceed 45°C. Therefore, heat exchange can be optimized if the clinician carefully monitors either intraperitoneal temperature or dialysate temperature just before its entrance into the peritoneal cavity. The resulting increased rate of rewarming and decreased CPR time may have practical significance as previously discussed.

In conclusion, Dr Herz and his colleagues are to be applauded for their successful resuscitation of a 14-year-old girl suffering from submersion hypothermia. Appropriate knowledge as to the aggressiveness and timing of cardiopulmonary support relative to rewarming was paramount to her survival. We hope this is an understanding reached by more and more physicians who are likely to see patients suffering similar traumatic events.

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The changing environment of medicine

Some 22 years ago I began working in a hospital, mostly out of the necessity to acquire even a modest income, but also in the interest of finding something out about myself. The 60s were a time of social turmoil, and toward the end of that decade I had developed a certain social consciousness. I had a feeling that I needed to accomplish something, anything that might be considered of value to society. Not that I was a socialist, a democrat, or a communist by philosophy. I just wanted to do something that would be regarded as worthwhile by someone. Working in the hospital provided an ideal opportunity for just such an endeavor. Not being unduly influenced by the anti-establishment attitudes of my peers, I still thought of doctors as a variety of societal holy men and venerated their art. I also thought of hospitals as great institutions that provided for the physical welfare of people in need.

Working in a hospital then was great fun! Everyone seemed to enjoy what they were doing, and despite the fact that I knew very little about anything, they always took the time to teach me. No matter how ignorant I proved myself to be, I can't remember an instance when I was degraded for it. There was a sense of comradery and a willingness to pitch in and help each other take care of the tasks that needed to be done to make the patients better. Care was also taken to provide for the comfort of those who could not be made better. It was also a time when there seemed to be a lot of new and better things happening in the hospital. With the advent of a new technology, or a new treatment, or some new procedure, everyone was more enthusiastic and eager to learn, and eager to help others learn, too. What better atmosphere could a young, searching individual ask for?

It was no small wonder that all of this encouraged me to think of how I might relate to things on a more involved and permanent scale. My experiences as an orderly were enlightening and satisfying, but they gave rise to a feeling inside that I had not previously known. Something told me I would like to become a physician and enjoy the satisfaction and sense of worth I found in the doctors at that time. I was fascinated by the gratification that most of them seemed to experience, and awed by the amount of knowledge they possessed. Being a young, impressionable person, I was also interested in the life style they enjoyed.

Shortly after I began emptying bedpans and helping nurses turn incapacitated patients in their beds, I developed an image of myself crafted after a great man in my life—my own family doctor. Here was a man of immense stature, both to me and to everyone who knew him. He was always kind, gentle, and accessible, and he knew more about the world than anyone else I had ever known. Yet in spite of his stature, he accepted other people as equally important in the cosmos and made one feel at ease. This was the kind of man I wanted to be, and the only way I could conceive of achieving this goal was to become a physician—it was an image I could not transfer to any other profession or occupation. Despite a previously dismal academic experience, and the pessimistic counseling of several significant people, I set about acquiring the necessary scholastic background and was eventually accepted into medical school in the Fall of 1975. I began my medical studies with the awareness that things were not going to be the same for this time-honored profession in the future as they were when I first developed my dream. Having worked in the hospital environment to help pay for my education and sustenance, I began to become concerned with the eventual outcome of this change as it might affect my professional career. I was not prepared for the enormity of the change that would occur, or the devastating impact it would have on medical practice.

Needless to say, I was not the only one interested in gaining admission to medical school and working for the good of my fellow man. I was, however, one of those individuals who still held the art of medicine in high esteem. I was somewhat amazed to hear the pronouncements of some of my fellow students about how rotten some doctors who expected to earn a good living were. It seemed to me that they made out fairly well, but they also did one hell of a lot of work, and spent long hours doing it after many years of learning what it was they were doing. The financial recompense didn't seem all that unreasonable. If one was interested in becoming a barefoot doctor in a developing country, there was nothing wrong with that, but there seemed no reason to berate those who wanted to live well in an industrialized society. My only objection was to those who chose to study the art solely for the purpose of earning a good living and enjoying the prestige. They had apparently missed out on the fun I'd had, and seemed not to appreciate practicing an art that could bring such self-actualization as to make the money mundane.

Not only did I detect a changing attitude in the students, but the government and the press seemed to be conspiring to make my life miserable. Suddenly, every time I picked up a newspaper or magazine there was another article about how some government agency had determined that someone in medicine was making far too much money, and producing less than desirable results in the process. I began to hear conversations about economics on the hospital wards, mixed in with the pearls about the practice of good medicine. As time went by, these conversations took on more and more prominence, and I wondered why we didn't spend as much time talking about the ethical dilemmas we faced every day. But the articles and television news stories continued, and the conversations became more bitter, and I came to realize that I was entering a profession that was under attack. I also became painfully aware of the attitudinal changes that this attack engendered in those who work in health care.

Somewhere between pre-med and residency, the hospital lost its charm. Now it was a place where one went to work. The simple question became a bother, or an unwelcome criticism. If you didn't know something, it meant you were dumb. Personnel had become consumed by mountains of new paperwork and were struggling to take care of the patients and find the time to write everything down in duplicate. The task of mounting a good offense against illness and the discomfort it produces had been transformed into a task of documenting a defense against myriad agencies looking over one's shoulder. Even worse, the sense of cooperation and comradery in our work had been perverted into protecting ourselves against the potential accusations of our comrades in arms. This is no way to fight a war.

On every front it seems that the value of health care has eroded in our society. The government, insurers, and employers all say we spend too much money. The lawyers, feeding on the inaccurate findings of others, claim we cause harm in the process. They want their share of the too much money we spend. The patients now have a cacophony of agencies, reviewers, and insurers between them and the providers, and a cadre of consumer alternatives that oftentimes represent sheer quackery. Physicians are now competitive with each other and the hospitals, and all too often are willing to sell their expertise to lawyers or to consumers to whom they present themselves as the latest and greatest. Everyone concentrates on the money, the criticism, and the competition.

Somewhere along the line we seem to have forgotten that we in the United States enjoy the finest health care in the world. We lose sight of the fact that many countries send their doctors here to learn how to do it better. We have lost sight of the fact that we can now offer people a healthier and longer life than we could ever make possible before. We seem to have forgotten the fun of everybody working together to do something that everyone thought was good. Perhaps it's not too late to recapture that feeling. I hope not.

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Renal transplantation experience

A historical review and analysis

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ABSTRACT. The results of the first 323 kidney transplants performed at Buffalo General Hospital are presented. The renal transplant program at The Buffalo General Hospital advanced from two transplants in 1964 to 33 transplants in 1984. Over the ten-year period from 1973 to 1983, 70 patients received renal allografts from living related donors, and 170 received cadaveric transplants. One-year patient and graft survival rates for recipients of living related transplants were 97% and 84%, respectively; for cadaver transplants, the figures were 82% and 62%, respectively. An analysis of survival curves shows that patient and graft survival experience is in keeping with the results from other major transplant centers. There was no correlation between patient survival and the transplant rate at the center. This study reflects the kidney transplantation experience at a small center, and confirms the observation that such a center can achieve patient and graft survival rates that are equivalent to those of larger transplant centers.

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The object of renal transplantation is to provide those who are suffering from end-stage renal failure with an allografted vascularized kidney that will function and maintain health for a long time. Kidney transplantation was first successfully performed in 1954 between identical twins. Shortly thereafter, it was shown to be feasible between nonidentical siblings following modification of the recipient's immune response by irradiation.[1] During the last three decades, developments in surgical techniques and increased expertise in immune modulation have had a major impact on both the safety and efficacy of this procedure. Kidney transplantation has now emerged as the treatment of choice for end-stage renal failure. Approximately 7,000 transplants are now performed annually in the United States.[2]

The survival of transplant recipients is equal to or exceeds that of patients treated with dialysis. Transplant recipients may also enjoy a degree of rehabilitation and a quality of life that are attainable with no other form of therapy. Successful transplantation depends on careful preparation of the recipient and donor, surgical technique, and optimal manipulation of the immune response to prevent graft rejection.

In this paper, we examine the renal transplant experience at The Buffalo General Hospital, in an attempt to document that a small transplant center can achieve acceptable allograft survival, while having patient survival rates comparable to those of larger transplant programs.

Patients and Methods

From November 1964 to December 31, 1984, 323 renal transplants from living related donors and cadaver donors were performed at The Buffalo General Hospital, Buffalo, NY. The results were tabulated and retrospectively analyzed. At least one year of follow-up information was available on all patients. There were 128 female (40%) and 195 male (60%) patients, ranging in age from eight to 55 years.

The clinical experience in transplantation was divided into two time periods based on the significant differences in philosophies regarding medical and surgical care of renal transplant patients that prevailed. The transplants performed from 1964 to 1972 were considered separately from transplants performed after 1972.

The 323 renal transplants were performed in 297 patients (195 male and 128 female). Two transplants were performed in 1964, while 33 transplants were carried out in 1984, with the maximum number performed in a single year being 37. Of the 323 transplants, 101 were from living related donor sources, 222 were from cadaver donors, and none were from living unrelated donors. Forty percent of patients who received transplants (128/323) presently have functioning grafts. At the time of transplantation 6.2% of these patients were children.

From 1964 through 1972, 50 transplants were performed. The results, without exclusion and 100% follow-up during this early, pioneering phase of the program, are summarized in Table I. Graft and patient survival were separately evaluated on all living related and cadaver transplants carried out since January 1973, with a minimum of one year follow-up.

The immunologic criterion for cadaver kidney placement was a negative crossmatch between donor and recipient using fresh and stored recipient serum. Since 1979, antiglobulin crossmatching has been used in addition to the Amos-modified standard National Institutes of Health crossmatch. Although the criterion for cadaver kidney selection was negative crossmatching, many cadaver kidney recipients shared one or two HLA antigens with the donor. Recipients of living related kidneys all shared at least one haplotype with the donor. Since 1979, mixed lymphocyte culture also has been used to confirm haplotype sharing. All liv-

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TABLE I. Renal Transplant Experience at The Buffalo General Hospital, 1964–1972

	Number
Kidney donor source	
Living related	24
Cadaver	26
Total	50
Patient status	
Alive	
with living related graft	12
with cadaveric graft	4
without graft	1
with new graft after 1972	5
Died	28
Total	50

ing related recipients had negative crossmatching with their donors.

The immunosuppressive medications used were prednisone and azathioprine. Standard maintenance regimens of azathioprine and prednisone were used consistently, with a three-day intravenous steroid pulse administered for episodes of acute rejection (1 g/day for three days, repeated once if the episode was not reversed). A maximum of three such pulses was used during the first three months after transplantation. Since 1973, a more rapid tapering of steroids has also been used, with a maximum of 20 to 30 mg of prednisone per day given at three months, and 10 to 20 mg per day at one year after the transplant. More recently, cyclosporine has been used as an alternative immunosuppressive agent in an attempt to improve graft survival and reduce the complications related to conventional immunosuppression.

Since 1973, the rate of sepsis was lowered by instituting several modifications in patient management, reducing immunosuppressive therapy, and improving prophylaxis and diagnosis of infections—especially those in surgical incisions. These refinements include intraoperative antibiotics, routine use of ultrasonogram and renal scan, as well as substitution of needle biopsy for open renal biopsy. All transplant recipients who underwent any surgery were given a single intravenous bolus of high-dose broad-spectrum antibiotics on induction of anesthesia. Subsequently, specific antibiotics were administered only as indicated.

In selected patients with documented remote or active peptic ulcer disease, an acid-reducing procedure was done before transplantation to lessen the potential for postoperative steroid-induced gastrointestinal hemorrhage. All patients were treated with cimetidine and antacids during and after their hospital stay. Recently, ranitidine therapy was started as an alternative to cimetidine, especially in those patients whose immune systems were suppressed by cyclosporine. Splenectomy has not been carried out in the preparation of transplant recipients since the analysis of pooled data has shown no significant beneficial effect on graft survival.[3] Definite indications for pretransplant bilateral nephrectomy were pyelonephritis, structural abnormalities of the urinary tract, renin-mediated hypertension, and polycystic kidneys. These conditions have led to urinary infection or significant hematuria.

Graft and patient survival curves were calculated by actuarial methods.[4] Graft loss was calculated on the basis of the date of transplant nephrectomy or, in cases of retained failed grafts, the date that the patient was returned to maintenance dialysis. Graft loss also includes death of a patient from any cause. The data on patient survival include all deaths that occurred within one year after transplant nephrectomy and reinstitution of maintenance dialysis. They also include any subsequent deaths that may have had any relationship to the transplant.

The rehabilitation status of all patients who survived five years or longer with functioning grafts was systematically reviewed and assessed. Pertinent data of patient status at five years posttransplantation were derived from the patient, family members, medical records, and staff caring for the patient at that time. Patients were considered to have been rehabilitated if they were able to lead active lives both physically and psychologically. This included tasks identical to or perceived as the equivalent of those in which they had engaged prior to the onset of renal failure.

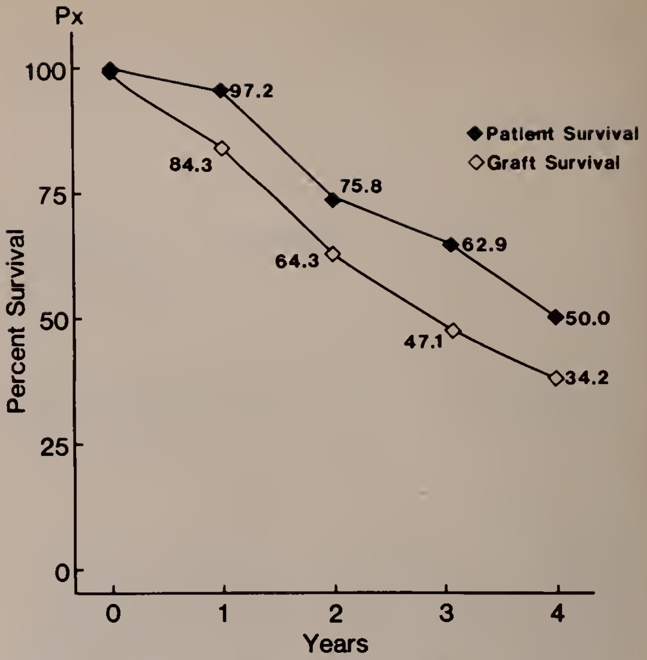

FIGURE 1. Patient and graft survival in living related recipients.

RESULTS

Figure 1 demonstrates graft and patient survival following transplants from living related donor sources. In this group, 97% one-year patient survival and 84% one-year graft survival rates were achieved.

One-year graft and patient survival rates of 173 cadaver transplants performed since 1973 are shown in Figure 2. In patients who

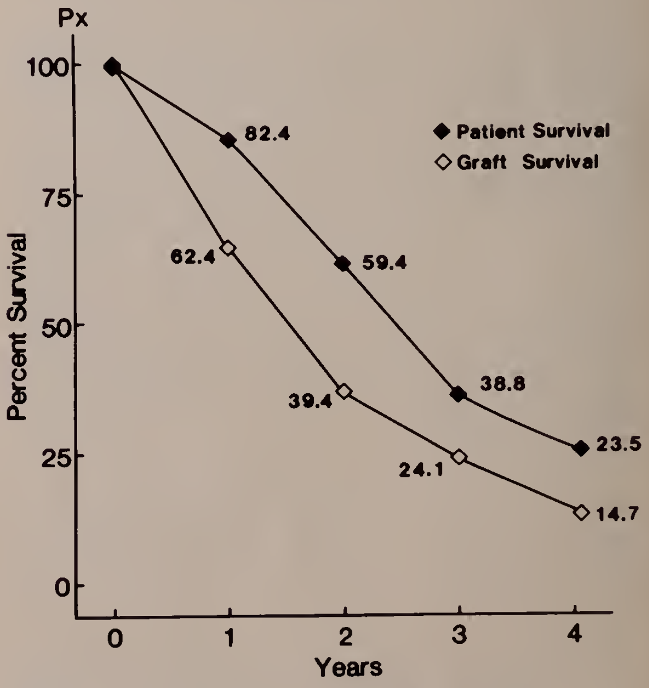

FIGURE 2. Patient and graft survival in cadaver recipients.

received a cadaver kidney transplant, the one-year patient and allograft survival rates were 82% and 62%, respectively. Overall patient survival rates among recipients of living related and cadaver grafts since 1973 were 87% and 69%, respectively.

Assessment of the degree of rehabilitation in patients with long term functioning grafts was evaluated in 40 recipients who lived five years or longer with functioning grafts. Functioning grafts in these patients represented 23 living related grafts and 17 cadaver grafts. There were no cases of retransplant among those five-year surviving kidneys.

The longest survivor with a functioning living related graft (almost 15 years) was 47 years old at the time of transplantation. This recipient has normal renal function and has been fully rehabilitated without medical problems. The longest survivor of cadaveric transplantation (almost 14 years) was 17 years of age at the time of transplantation. This recipient also has normal renal function.

The causes of death less than one year after graft placement were sepsis, gastrointestinal hemorrhage, encephalopathy, pulmonary embolus, and ischemic colitis. There were only two fatal cytomegalovirus (CMV) infections and only one *Herpes simplex* infection. Complications occurred in 240 patients (including recipients of 170 cadaveric and 70 living related transplants) and included sepsis, gastrointestinal hemorrhage, cerebrovascular accident, renal artery stenosis, myocardial infarction, and pulmonary embolus. These were managed with surgery, endoscopy, or medical treatment.

DISCUSSION

The field of renal transplantation encompasses a unique integration of surgical and immunologic disciplines that can now result in total rehabilitation and a return to normal life style for most patients with end-stage renal disease. Prior to the emergence of renal transplantation and dialysis therapy, all patients with end-stage renal failure died of their illness.[5]

During the ten-year period 1973 to 1983, one-year patient survival rates at The Buffalo General Hospital have been 97% and 82% after living related and cadaver transplantation, respectively. Similar results have been reported by two other small transplant centers.[6,7] The patient and graft survival rates do not differ significantly from the results determined by the Standards Committee of the American Society of Transplant Surgeons and the World Transplant Registry (Table II).[8,9]

In this institution, transplant surgeons, nephrologists, and general surgery residents participated in the care of transplantation patients. The care necessary for the successful management of renal transplant recipients can be difficult or impossible to give when the transplant service does not exist as a separate clinical entity. Even small numbers of patients require an inordinate amount of time and thought. However, as this report illustrates, acceptable results can be obtained on a smaller scale by close collaboration betweeen skilled transplant surgeons and nephrologists committed to high quality patient care in transplantation.

TABLE II. A Comparison of Patient and Graft Survivals After One Year

	Living Related		Cadaver	
	Patient	Graft	Patient	Graft
The Buffalo General Hospital	97	84	82	62
Standards Committee of the American Society of Transplant Surgeons[8]	95	78	88	55
World Transplant Registry[9]	85	70	70	50

Renal transplantation requires a specialized form of treatment, and it is reasonable to assume that experience with more patients should enhance the expertise of the involved clinicians and thereby optimize the therapeutic outcome. We believe that the results of complex surgical therapy such as renal transplantation are dependent on the skill and experience of the attending physicians and surgeons rather than the absolute number of cases.

This study not only reflects the renal transplantation experience in this institution, but also confirms the observation that patient and graft survival rates can be the same in small and large transplant centers. It also indicates that there is no correlation between center activity and patient survival.

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Awareness of the New York State law banning the sale of tobacco to minors

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ABSTRACT. In New York State it is illegal to sell tobacco products to individuals under the age of 18 years. In most communities, compliance with this law is poor. We surveyed 79 business establishments in the Buffalo, NY, area in which tobacco was sold to determine awareness of the law prohibiting the sale of tobacco to minors. In 40% of the businesses surveyed, owners/store managers either did not know of the law or were misinformed of the legal age for purchasing tobacco products. Only 23 stores (29%) claimed to have a copy of the law posted on the premises as required by the law. Strategies for increasing the percentage of businesses that comply with the law are outlined. In addition, legislative initiatives are identified that could further strengthen efforts to prevent children from using tobacco.

(NY State J Med 1988; 88:418–419)

Tobacco use was responsible for approximately 16% of all deaths in New York State in 1986.[1] The medical care costs associated with treating persons with diseases caused by tobacco in New York State totaled $1.7 billion in 1985.[1] There are more than four million cigarette smokers in New York State, 250,000 of whom are under the age of 18 years. Most adult cigarette smokers report first experimenting with smoking as teenagers.[2] Unfortunately, many of those who start smoking as teenagers become dependent on tobacco use and, despite repeated attempts at stopping, continue to smoke throughout their lives.

In an effort to prevent children from using tobacco, 38 states have enacted laws to restrict the sale of tobacco to minors.[3] In 1965, New York State passed a law banning the sale of cigarettes to persons under age 18 years (Penal Law, Section 260.20(5)).[3] This law was amended in 1983 to include a ban on the sale of chewing tobacco and powdered tobacco. The New York State law also requires businesses that sell tobacco to post a sign stating: "SALE OF CIGARETTES OR OTHER TOBACCO PRODUCTS TO PERSONS UNDER EIGHTEEN YEARS OF AGE IS PROHIBITED BY LAW" (General Business Law, Section 399-e).[3] Selling tobacco to a minor is a class B misdemeanor, with penalties including imprisonment and/or fine. Failure to post a sign is punishable by a fine of up to $100 for the first offense and up to $250 for all subsequent violations.

Despite the good intentions of laws banning the sale of tobacco products to minors, evidence indicates that such laws are almost never enforced. For example, when we investigated enforcement of the law in the City of Buffalo, representatives of the Criminal Records Department could not recall a case in which a business was fined for selling tobacco to a minor. In a recent test of the efficacy of the Massachusetts law prohibiting the sale of tobacco to minors, an 11-year-old girl unaccompanied by an adult was able to purchase cigarettes in 75 of 100 stores visited.[4] Only four of the stores had the law posted where it could be seen by customers. A follow-up telephone survey of 80 of the stores revealed that 36% of respondents were either unaware of the law or could not correctly identify the legal age for purchase. Awareness of the law and refusal to sell cigarettes to a minor was greater in those stores that reported having a sign posted about the law.

This paper reports on awareness of the New York State law prohibiting tobacco sales to minors among businesses that sell tobacco in the Western New York area. Findings from this study are used to suggest ways to increase compliance with the current law. In addition, legislative initiatives are identified that could further strengthen efforts to protect children from tobacco use.

METHODS

The study population included 88 businesses located in the Buffalo, NY, area. The following types of businesses were surveyed: supermarkets, convenience food stores, and drugstores. Businesses were randomly selected out of the Buffalo Area Telephone Directory Yellow Pages. For each business selected, an attempt was made to contact the owner or store manager to request his or her participation in a survey about the store's policy concerning the sale of tobacco products to minors. Interviewers identified themselves as representatives of Roswell Park Memorial Institute, a cancer hospital and research center in Buffalo. Of the 88 stores contacted, two refused to participate in the survey and seven indicated that they did not sell tobacco products.

In businesses in which tobacco was sold, the owner/store manager was asked the following questions: Does your store have a policy about how old a person must be to buy cigarettes? Are you aware of any laws concerning the sale of tobacco products to children? Is there a sign posted anywhere on your premises pertaining to the sale of tobacco products to minors? Those who claimed to be aware of the law were asked to identify the legal age for purchasing cigarettes.

RESULTS

Among the 79 businesses surveyed, 59 respondents (73.4%) claimed awareness of the law. However, only 47 respondents

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TABLE I. Awareness of the Law Prohibiting the Sale of Tobacco to Minors by Type of Business

	Supermarket (N = 13)	Convenience Food Store (N = 36)	Drugstore (N = 30)	Total (N = 79)
Aware of the law	84.6%	66.7%	76.7%	73.4%
(95% confidence interval)	(64.2–100)	(51.1–83.2)	(61.2–92.3)	(63.4–83.2)
Aware of the legal age	69.2%	52.8%	63.3%	59.5%
(95% confidence interval)	(43.1–95.3)	(36.3–69.3)	(45.8–80.7)	(48.5–70.5)
Post the law*	84.6%	13.9%	23.3%	29.1%
(95% confidence interval)	(64.2–100)	(2.5–25.3)	(7.8–38.8)	(19.1–39.1)

* $P < 0.01$; supermarkets > convenience food stores and drugstores.

(59.5%) correctly identified the legal age for purchasing tobacco. Twenty-three of the businesses surveyed (29.1%) reported having a copy of the law posted on the premises. As shown in Table I, posting of the law varied by type of store. Eleven of the 13 supermarkets surveyed reported having a sign posted about the law, compared to five of 36 convenience food stores and seven of 30 drugstores. Convenience food stores and drugstores that were part of a chain were three times more likely to have the law posted than nonchain stores (29% versus 9%).

DISCUSSION

Given what we know about the addictive potential of tobacco use and its danger to health, efforts to limit access to tobacco products by children are clearly warranted. However, the mere existence of a law prohibiting the sale of tobacco to minors does not guarantee that it will be enforced.[4] Forty percent of the businesses surveyed in this study either did not know of the law or were misinformed of the legal age for purchasing tobacco products. Fewer than one third of the businesses reported compliance with the requirement that the law be posted in a conspicuous place in the store.

Educating tobacco vendors about the law prohibiting the sale of tobacco products to minors is an obvious first step needed in gaining their compliance with the law. Making copies of the law available for posting in stores will increase the likelihood that businesses will post the law. Presently, businesses are responsible for making their own signs. A sign warning that it is illegal to sell tobacco to persons under 18 years of age, conspicuously displayed, may discourage some teenagers from attempting to purchase tobacco.

Even with an increased educational effort, compliance with the law will likely continue to be poor, until enforcement efforts are increased and penalties levied that act as a deterrent to noncompliance. To date, enforcement of the law banning the sale of tobacco to minors has been almost nonexistent in the area surveyed. Enforcement of the law banning the sale of tobacco to minors is the responsibility of local law enforcement agencies. Unless community concern is raised about the issue of selling tobacco products to minors, it is not likely that law enforcement officials will expend resources for enforcement efforts. It may be necessary to convince law enforcement officials of the need to enforce the law, perhaps by publicizing how easy it is for a teenager to purchase tobacco. For example, youth groups such as the Boy Scouts and Girl Scouts could be recruited to monitor periodically a store's compliance with the law. Alternatively, a survey could be conducted in the schools to identify where children are able to purchase tobacco products. The local school board or parent-teacher association could initiate efforts to convince major offenders of the law to improve compliance or threaten to promote a boycott of their business.

New laws should be enacted to further strengthen efforts to prevent children from gaining access to tobacco products. The free distribution of tobacco samples, which is legal in New York State, should be prohibited. While distribution of tobacco to minors is against the law, children frequently obtain free samples. Banning the distribution of free samples is the only feasible way to be sure that minors are not given free tobacco samples. Vending machines are an important source of tobacco products for minors. Despite the fact that vending machines have a sign affixed which states that it is illegal to sell tobacco products to persons under 18 years of age, children frequently obtain tobacco products from vending machines in violation of the law. Because the sale of tobacco products to minors from vending machines cannot be closely supervised, tobacco product vending machines, like alcohol vending machines, should be prohibited. Finally, vendors of tobacco products should be licensed in New York State. Licensure would provide an opportunity to educate vendors about tobacco laws and give public officials the opportunity to revoke the licenses of businesses that repeatedly fail to comply with the law. The threat of losing one's license to sell tobacco products may serve as a more effective deterrent to violating laws that prohibit the sale of tobacco to minors than the small monetary fines currently imposed.

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Comparative efficacy of two dopamine agonists, pergolide and lergotrile, in Parkinson disease

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ABSTRACT. Pergolide is a dopamine agonist that will soon be available for the treatment of Parkinson disease (PD). Pergolide is a congener of an older agonist: lergotrile. Lergotrile, an effective antiparkinson drug, was withdrawn from the market because it resulted in hepatotoxicity in up to one third of patients. Pergolide differs from lergotrile in the presence of a propyl group on the N6 portion of the ergoline molecule. In animal models of PD, this substitution increases the antiparkinsonian effect. Pergolide also lacks lergotrile's cyano and halogen groups. These groups may be responsible for lergotrile's hepatotoxicity.

The activity of pergolide and lergotrile was compared in a retrospective analysis of 12 patients with advanced PD who were no longer satisfactorily responding to levodopa. Lergotrile or pergolide was added to the levodopa regimen. Pergolide was at least as effective as lergotrile. Unlike lergotrile, pergolide was not hepatotoxic. The mean dose of lergotrile was 50.4 mg. The mean dose of pergolide was 2.2 mg. This study demonstrates that a drug's structure can be modified in a rational manner with the new drug retaining the old drug's efficacy but not its toxicity.

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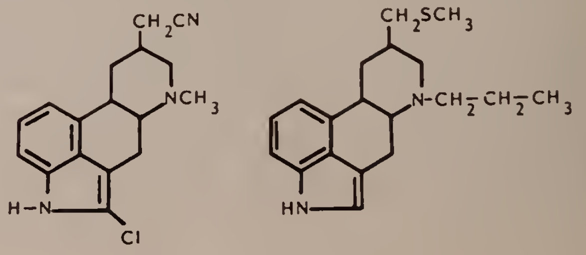
Parkinson disease (PD) is characterized by degeneration of the nigrostriatal dopamine-containing neurons. The effectiveness of levodopa in PD depends upon the ability of the remaining nigrostriatal neurons to synthesize dopamine from levodopa, and stimulate the striatal dopamine receptors. There are two populations of striatal dopamine receptors: the D_1 receptors that are linked to adenyl cyclase, and the D_2 receptors that are not. In time, the response to levodopa decreases and it becomes associated with a variety of fluctuations in performance. The decreased response to levodopa and the response fluctuations may be due to continued degeneration of the nigrostriatal dopamine neurons or to decreased sensitivity of the striatal dopamine receptors. Patients whose response to levodopa has decreased may respond to dopamine agonists, drugs that directly stimulate the striatal dopamine receptors. Stimulation of the D_2 striatal receptors is largely responsible for the antiparkinson effect. Stimulation of the D_1 receptors may also have an antiparkinson effect.

Lergotrile is a synthetic ergoline that is effective in PD.[1-4] Lergotrile stimulates the D_2 receptors.[5,6] However, approximately one third of patients treated with lergotrile develop hepatotoxicity.[3,4,7] Pergolide is one of several congeners of lergotrile (Fig 1). In animal models it has been demonstrated that the antiparkinson efficacy of the ergolines is related to the length of the alkyl chain at the N6 portion of the molecule. Pergolide, which contains a propyl group, is more potent on a milligram per milligram basis than lergotrile, which contains a methyl group. In addition, as it was thought that lergotrile's hepatotoxicity was related either to the presence of the cyano group or to the halogen, the congener selected for testing had to be free of both. Pergolide fulfilled these criteria and has been shown to be an effective antiparkinson drug.[10-13]

There are no studies comparing patients who were treated with lergotrile with patients who were treated with pergolide. There are also no studies comparing the effects of lergotrile and pergolide on the liver. If patients who developed hepatotoxicity on lergotrile did not develop hepatotoxicity on pergolide, this would affirm the theory that the cyano group or halogen was responsible for the hepatotoxicity. As pergolide will soon be approved by the Food and Drug Administration (FDA) for the treatment of PD, we decided to review our experience with both drugs.

METHODS

Between 1975 and 1977, 53 patients with advanced PD were treated with lergotrile.[3] Lergotrile was added to the levodopa regimen until toxicity developed, as manifested by mental changes, dyskinesias, and/or hepatotoxicity, or until an arbitrary maximum of 150 mg per day was reached. At monthly intervals a blind observer assessed rigidity, tremor, bradykinesia, postural stability, and gait on the NYU Disability Scale, where "0" represents no disability and "100" represents maximal disability. Patients were evaluated during "on" and "off" periods.



LERGOTRILE PERGOLIDE

FIGURE 1. Molecular structures of lergotrile and pergolide.

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TABLE I. Comparative Efficacy of Lergotrile/Pergolide in 12 Patients

Drug	Mean Patient Age in Years (Range)	Mean Duration of PD in Years (Range)	Diurnal Fluctuation (No. of Patients)	Mean Dosage of Levodopa (mg Sinemet®)	Mean Levodopa Duration in Years (Range)
Lergotrile	61.3 (49–72)	9.9 (7–16)	11	1,113 (750–1,500)	6.4 (8–13)
Pergolide	64 (50–75)	13 (9–18)	12	1,100 (500–1,500)	9.7 (8–13)

Between 1979 and 1982, 59 patients were treated with pergolide.[11] Pergolide was added to levodopa until mental changes, dyskinesias, and/or toxicity developed, or an arbitrary maximum of 5.0 mg per day was reached. Patients were assessed in a manner similar to those on lergotrile.

Among the 53 lergotrile-treated patients, 12 were later treated with pergolide. The results of treatment with lergotrile were compared to treatment with pergolide in these 12 patients using the paired t test. Data on these patients are summarized in Table I. Eleven of the patients had diurnal response fluctuations consisting mainly of "wearing off" phenomena. In one patient, response fluctuations developed at the time pergolide was started. At the time pergolide was begun the patients were 2.7 years older, had had PD 3.1 years longer, and were on levodopa 3.3 years longer. In general, patients were worse at the time they started pergolide (stage 3.3 ± 0.02) than at the time they started lergotrile (stage 2.7 ± 0.1.)

Results

The results of treatment are summarized in Table II. Seven patients improved on lergotrile at least one stage in the "on" period, and one patient improved at least one stage in the "off" period. The mean duration of lergotrile treatment was 9.8 months, the range, 3–33 months. Eight patients on pergolide improved at least one stage in the "on" period and seven improved at least one stage in the "off" period. The mean duration of pergolide treatment was 14.4 months, the range, 0.5–26 months. Lergotrile was discontinued in seven patients because of adverse effects including hepatotoxicity (six patients) and dyskinesias (one patient). Lergotrile was discontinued in five patients because of lack of efficacy. Pergolide was discontinued in six patients because of adverse effects including confusion (one patient), dyskinesias (three patients), and orthostatic hypotension (two patients).

Discussion

In animal models pergolide is more potent and longer acting than lergotrile. This may be related to the substitution of the propyl group in pergolide for the methyl group in lergotrile at the N6 portion of the molecule. Alternatively, pergolide may be more potent and longer acting than lergotrile because it stimulates both the D_1 and the D_2 receptors. Because pergolide and lergotrile were not compared directly in humans, the most that can be said is that the drugs have a similar antiparkinson effect. However, it is noteworthy that pergolide resulted in improvement in more patients than did lergotrile at a time when the patients had more advanced disease. It is encouraging that the decreased efficacy in patients on lergotrile did not preclude a good response to pergolide. The decrease in efficacy that occurs with chronic therapy may be related to desensitization of the striatal receptors by the agonist, a process known as "down regulation,"[9-13] or it may be related to disease progression.[9-12] The response to pergolide at a time when patients were no longer responding to lergotrile suggests that part of "down regulation" is drug related, and using a different agonist restores responsiveness. Finally, patients who developed hepatotoxicity on lergotrile did not develop hepatotoxicity on pergolide, confirming the rationale for choosing pergolide.

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TABLE II. Results of Treatment

		"On"		"Off"	
		NYU Scale	Stage	NYU Scale	Stage
Mean lergotrile dose (range) 50.4 mg (30–100)	Before	26.0[*] (±2.4)[†]	2.7 (±0.1)	55.4 (±5.8)	3.9 (±0.2)
	After	20.5 (±2.9)	2.0 (±0.2)	55.0 (±5.9)	3.9 (±0.3)
	Change	−21% $p < 0.05$	−18.5% $p < 0.05$	0 NS[‡]	−3% NS
Mean pergolide dose (range) 2.2 mg (0.1–3.5)	Before	39.9 (±3.2)	3.3 (±0.2)	70.0 (±3.3)	4.8 (±0.1)
	After	25.0 (±4.4)	2.7 (±0.3)	57.2 (±3.1)	4.2 (±0.2)
	Change	−37% $p < 0.01$	−18% $p < 0.01$	−18% $p < 0.01$	−12.5% $p < 0.05$

[*] Mean.
[†] Standard error of the mean.
[‡] NS, not significant.

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New York City Department of Health HIV Counseling Program

The New York City Department of Health provides free and anonymous counseling and human immunodeficiency virus (HIV) antibody testing at six sites in New York City. Physicians may advise patients who desire information and referral regarding anonymous HIV antibody counseling and testing to call the department's AIDS Hotline number:

718-485-8111

The early diagnosis of glomerulopathy through the detection of subclinical proteinuria: Microalbuminuria

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For more than 150 years, since Richard Bright's description of glomerulonephritis (Bright's disease), significant proteinuria (>250 mg/day)[1-4] has remained the most reliable, reproducible, and easily detectable clinical sign of renal disease. Proteinuria is transient in most acute, self-limited glomerulo-tubulo-interstitial nephropathies. Conversely, glomerulopathies with a relentless course display persistent proteinuria. Urinary excretion of protein is, however, a normal phenomenon common to most mammalian species.

CLINICAL (OVERT) PROTEINURIA

In humans, daily proteinuria does not exceed 200–250 mg, of which less than 50% is constituted by albumin. In health and in disease as well, a larger proportion of the 24-hour urinary protein is excreted while the subjects are up and about, indicating some influence of physical activity perhaps mediated by hormonal variations on the glomerular filtration rate. Due to its high concentration in plasma (4 g/dL) and its low molecular weight and size (67 kD and <35Å radius, respectively), albumin generates and maintains oncotic pressure in the plasma. Massive urinary losses of protein, defined as 3.5 g/m^2 body surface area/day for adults and 60–100 mg/kg body weight/day for young children, may surpass the body's capability to synthesize new material, resulting in hypoalbuminemia and the nephrotic syndrome. This seemingly simple mechanism, however, has become highly controversial. Actually, liver synthesis of albumin may be greater than urinary excretion, implying the existence of additional processes, ie, extraurinary losses and increased albumin catabolism, contributing to produce and/or maintain hypoalbuminemia.[5,6] Clinically, then, it becomes of paramount importance to evaluate albuminuria because, independent of etiologic and pathogenetic phenomena, it is a gauge of the activity of renal disease and reflects the organism's compartmental distribution of fluids.

Normally, permselectivity properties of the glomerular capillary wall (GCW) result in an almost deproteinized plasma ultrafiltrate,[7] ie, macromolecules may be selectively retained or transported through the GCW depending on their net electrical charge and a critical dimension. Most native, circulating, plasma proteins, such as albumin, are endowed with a low or anionic pI and are repelled through electrical impedance by the glomerular polyanion, a glycoprotein-rich, highly negatively charged matrix that coats the GCW.[8-11] On the other hand, proteins of sizes larger than albumin are excluded.[7,8] Despite these anatomic and physiochemical constraints to leakage, minute quantities of albumin and other higher molecular weight proteins slip through nonselectively into the urine. Therefore, processes that alter, deplete, or neutralize the glomerular polyanions induce significant proteinuria.[9-11]

Ultrastructurally, the GCW, from its inner aspect to the urinary space, consists of a triple layer, namely, the endothelium, the basal membrane, and the epithelium. The actual plasma filtration process begins in the endothelial fenestrae, and continues through the lamina densa of the glomerular basement membrane and across the slit pores and membranes of the epithelial cell, so that the filtrate may reach the urinary space and the tubule, where most protein is reabsorbed. Increments of a fraction of a milligram of protein per mL of glomerular filtrate above normal in the 180 liters of plasma filtered daily may overload the tubular resorptive capability. In this setting, tubules cannot return all protein back to the circulation, resulting in overt proteinuria. Biochemical, structural, and functional alterations of the GCW polyanions and filtering structures and the glomerular filtration rate, respectively, will increase protein excretion, particularly albumin, as occurs in the idiopathic nephrotic syndrome, insulin dependent diabetes, and other diseases.[9-13] Once the glomerular damage is established, and depending on its severity, it tends to be progressive with appearance of heavier molecular weight proteins, such as immunoglobulin G, in the urine.

The development of significant and persistent proteinuria in a disease previously manifested by other isolated urinary abnormalities, such as hematuria, may be considered an ominous sign. Similarly, proteinuria with or with-

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out hematuria most certainly indicates the establishment of serious renal involvement in a systemic disease such as diabetes mellitus (insulin dependent or insulin independent), systemic lupus erythematosus, and other vasculitides. About 45% of people with insulin dependent diabetes progress to end-stage renal disease within a few years after the onset of proteinuria.[14-20] Implicit in this statement is the concept that detection of significant proteinuria in insulin dependent diabetes represents a late finding. In the proteinuric stage, diabetic glomerulopathy may already be irreversibly established; chronic dialysis, and renal and possibly pancreatic transplants may be necessary as ultimate life sustaining procedures. As a clinician, one would like to determine well in advance which patients with insulin dependent diabetes may become proteinuric, and to be able to provide some form of treatment whereby development of end-stage renal disease is delayed or prevented.

MICROALBUMINURIA

Determination of minuscule amounts of urinary albumin has long been performed in the research laboratory setting. However, it was only in 1982 that the term microalbuminuria was coined and the concept introduced into clinical medicine.[21] Measurement of microalbuminuria, defined as abnormally elevated albumin excretion without clinical proteinuria, has added a new dimension to the evaluation of patients with insulin dependent diabetes by providing the means to identify high risk individuals. This new and important marker of insulin dependent diabetic nephropathy allows for earlier determination of the patient's clinical status and more accurate therapeutic decisions.

Urine specimens collected to test for microalbuminuria should be obtained during basal conditions, after patients have remained recumbent overnight, particularly those patients who suffer from insulin dependent diabetes who seem highly susceptible to the proteinuric effect of physical exercise. At least three determinations should be performed and the sterility of the sample guaranteed.[19,22-26] Normoalbuminuria (2.5–25 mg/24 hours or 3–18 μg/min) and microalbuminuria (26–150 mg/24 hours or 20–100 μg/min) are both Albustix® negative, whereas clinical (overt) proteinuria or macroalbuminuria is readily detectable by the Albustix® test ($\geq$200 mg/24 h or $\geq$180 μg/min). Albumin excretion can be estimated more accurately using the ratio of urinary albumin to urinary creatinine concentrations (albumin/creatinine, mg/mg; $<$0.1 is considered normal; 0.1 to 1.0 is slightly increased; $>$1.0 to 10 is moderate, and $>$10 indicates heavy proteinuria).[27] Measurement of the albumin/creatinine ratio can also be performed on a single random urine sample; this simple and convenient way to monitor albuminuria in large patient populations has demonstrated a high correlation with the 24-hour urinary albumin losses.[28]

Patients who will develop overt proteinuria demonstrate progressively increasing albumin excretion rates, from 11% in the normoalbuminuria category, to 22% (microalbuminuria), to greater than 50% (clinical albuminuria). Four percent of normoalbuminuric insulin dependent diabetics are at risk of having glomerulopathy, whereas 80% of those with a urinary excretion of 50–200 mg of albumin per day are at risk of progressing to end-stage renal disease.[19,20] Thus, the clinical importance of microalbuminuria resides in its ability to signal the early transition from functional and perhaps structural integrity to an intermediate, reversible situation, which, given the proper set of circumstances, will become irreversibly progressive.[13,23]

TECHNIQUES FOR MEASURING MICROALBUMINURIA

At the present time, microalbuminuria is measured by a variety of techniques,[29-31] two of which are most acceptable for clinical purposes: the immunocolorimetric[32] and the chemical methods.[33] In its present form the immunologic test for microalbuminuria is relatively inexpensive, and easy and safe to perform in a short period of time with reproducible results. The technology is readily available to any general chemistry or immunology laboratory. The enzyme linked immunosorbent assay (ELISA) is highly sensitive and is accurate at albumin concentrations as low as 3 μg/L of urine in timed specimens obtained overnight. The ELISA technique consists of "a solid phase single immunologic sandwich" whereby microtiter plates are coated and incubated with (goat) antiserum to human albumin to which the antigen (albumin, provided by the urine) is added; in a subsequent step, the plates are reincubated with a second peroxidase-labelled-(goat) antibody to human albumin.[30] The interaction between the enzyme (peroxidase) and a substrate produces a color reaction, the absorption of which is determined in a microtiter plate reader (at 450–486 nm) and is expressed in μg of albumin per liter of urine or mg per 24h. Sensitivity of the assay is high, with an optimum level between 5 and 300 μg/L (urine or plasma); precision profiles indicate estimated errors of less than 18% over this range, and of less than 8% between 25 and 300 μg/L. The results compare favorably with and are as good as or better than those obtained by radioimmunoassay and the turbidimetric procedure.[30] Because the reagents are used in high dilutions, 2 mL of antiserum could provide about 50,000 duplicate determinations.

One of the chemical methods, the Microalbutest® tablet[33] (based on the protein error of pH indicators), although less sensitive than the ELISA procedure, detects albumin at a much lower level than Albustix® but within the intermediate and highest discriminatory levels of the immunologic technique (70–100 mg/24 h, respectively).[19,33] This test, not yet available in the United States, is easy to perform and can be used as a bedside or office screening determination in nonproteinuric insulin dependent diabetics.

IMPLICATIONS OF TESTING FOR MICROALBUMINURIA

The enactment of Section 299 I of Public Law 92-603, known as the Social Security Amendments of 1972, allowed for Medicare coverage for individuals afflicted with end-stage renal disease effective July 1, 1973. Since then, the percentage of the population requiring dialysis and/or transplantation has grown at a steady 6.5% per year (from 15,417 patients in 1978 to 21,100 in 1983, or 92/million population). The prevalence of end-stage renal disease in the US is about 58,000 patients, with a mortality rate of about 11,000 per year according to data released in 1984.[34] Worldwide, particularly in the United States and Japan, the incidence rate of end-stage renal disease has reached 83 patients per million per year.

Within the United States, New York State has a nonadjusted (based on demographics) incidence rate of 85 per million, ranking ninth among all other states (the race, age, and sex-adjusted incidence is 78 per million or 17th among the states).[34] Notably, 75% of the 21,100 individuals in this predicament suffer from three major groups of illnesses: primary hypertensive disease, diabetic nephropathy, and glomerulopathies. Of this total, diabetic renal disease constitutes about 22% per year (4,620 patients or 19 per million). By age group, the largest annual increase in the incidence of end-stage renal disease occurs among individuals less than 25 years of age and 65 years or over—12.1% and 8.2% per year, respectively. The incidence of diabetes mellitus-related end-stage renal disease increased the most (compared to hypertension and glomerulopathies) from 1978 to 1983, jumping from 12 per million (2,730 patients) to 24 per million (5,460 patients), respectively.[34] The cost of programs for diabetic patients with end-stage renal disease amounts to about $300 million per year, which is nearly 17% higher than the cost of such programs for individuals without diabetes. Poorer therapeutic outcome of both dialysis and transplantation, and more frequent and prolonged hospitalizations, constitute the basis of the greater expenses incurred by diabetics.[34]

The use of a clinical test for early detection of diabetic patients who require better metabolic, blood pressure, and glomerular filtration rate control because of their high risk of progressive renal disease is highly desirable, reasonable, and beneficial. Such a test may be feasible, but it must also be cost effective. The benefits, measured in terms of significant delay of end-stage renal disease (by five to ten years or more), will spare much suffering; morbidity and mortality may be considerably decreased, and large sums of money can be saved and diverted to improve other aspects of end-stage renal disease programs. Comprehensive diabetic management, including normalization of metabolic abnormalities and of blood pressure, eradication of infections, adequate diet and physical activities, cessation of smoking, and perhaps the use of antiplatelet aggregation medications, may result in normalization of glomerular hyperfiltration and in a change back from microalbuminuria to normoalbuminuria.[35-40] Thus, the potential impact the intelligent and generalized use of microalbuminuria detection programs may have in diabetes, primarily for insulin dependent patients, is impressive. Moreover, kidney biopsy performed early in individuals at high risk of developing end-stage renal disease may enhance the knowledge of why and how functional abnormalities arise in this and perhaps in other nephropathies. Whether the microalbuminuria test is applicable to nondiabetic renal diseases, in which determination of covert albuminuria may carry the same significance as in insulin dependent diabetics, has not yet been ascertained. The data needed to answer this question are scanty at this point in time,[41] although it is evident that they can be easily obtained.

CONCLUSIONS

We have at hand a highly useful clinical tool to detect the subset of insulin dependent diabetic patients who will develop renal disease. Realization of its potential will be much more complicated. Physicians need to adopt a protocol for microalbuminuria screening of at least patients with insulin dependent and independent diabetes and other renal diseases. The presence of persistent microalbuminuria should stimulate sensitive counseling of these patients about their increased risk of nephropathy and a plan to improve and maintain glycemic control or to start other early therapeutic strategies. The magnitude of problems associated with end-stage renal disease in diabetic nephropathy and other glomerulopathies warrants greater allocation of medical resources to these high risk patients, their families, and their communities.

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EARLY DIAGNOSIS IN CHRONIC RENAL DISEASE

Accurate data concerning the incidence of chronic renal diseases are lacking. These disorders often are insidious in onset and pursue their course with few clinical characteristics, usually escaping detection until an advanced stage is reached. It is difficult to correlate individual clinical and laboratory findings with the renal pathologic lesions. Diverse etiologic agents may produce similar renal pathologic conditions, and different lesions may follow the same causative agent. Opinion remains divided concerning the relationship or continuity between acute glomerulonephritis and both chronic nephritis and the nephrotic syndrome. At an advanced stage, precise differential diagnosis may be impossible for the clinician and pathologist alike. It is not surprising that the clinical diagnosis of these conditions is often less than precise and that reporting at death is misleading. . . .

. . . Persisting proteinuria is the invariable denominator of renal disease. Most diagnostic methods are not applicable as routine screening methods. An effective screening method must be simple and generally applicable; it must be accurate, sensitive, and specific. The sulfosalicylic acid or similar standard qualitative tests for urinary protein meet these requirements. To detect traces of protein, urines should be of good specific gravity and if cloudy must be centrifuged. Urines collected after standing or activity are required. Twenty-four-hour urine collections are the least useful. Traces of urine protein (3 to 5 mg. per 100 ml.) are significant. Paper strip colorimetric methods are not even claimed to be sensitive below 10 mg. per 100 ml. and may prove inaccurate and even misleading. . . .

The increasing mortality from chronic pyelonephritis has brought sharply into focus the need for greater attention to the early diagnosis of chronic renal disease. It appears that the true incidence of chronic renal disease has been grossly underestimated. Although renal infections have increased absolutely and relatively in frequency, other renal disorders including chronic glomerulonephritis remain important clinical problems. . . .

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(*NY State J Med* 1961; 61:2766-2771)

Near-drowning in children: Management and outcome

DEBORAH K. RASCH, MD, TREVOR G. POLLARD, MD

Drowning is the cause of approximately 10% of all accidental deaths that occur each year. This problem is one of the most common emergencies physicians may be required to handle, regardless of their practice location. The annual morbidity and mortality from drowning among children and adolescents ranges from 8,000 to 12,000 per year. Boys account for nearly five times as many deaths as girls. Among boys, mortality peaks between 15 and 19 years of age whereas among girls mortality peaks between one and four years of age.[1] In 1977, 12.7% of all drownings occurred in swimming pools. More than 50% of pool drownings occurred among children under ten years of age, and the majority of these were under age four.

Neurologic morbidity in the form of chronic dependency for personal care after surviving the near-drowning episode varies between 0% and 21%. Several factors have emerged which appear to be important historical points with regard to improved outcome. These factors include time of submersion, water temperature, need for cardiopulmonary resuscitation (CPR), presence of spontaneous respiration, age of the patient, pH above 6.9 to 7.0, and the effectiveness of resuscitation and stabilization during the initial 12 to 24 hours.[2-4] With this background, the pathophysiology, management, and prognosis of the child who presents with near-drowning are reviewed.

PATHOPHYSIOLOGY

Drowning or near-drowning usually involves asphyxia and fluid aspiration. The introduction of even small amounts of fluid into the airways produces reflex closure of the glottis. In about 10% of drownings this laryngospasm persists, resulting in death from obstruction without actual aspiration.[1] In the majority of cases, however, increasing asphyxia causes the spasm to subside and the victim is able to gasp. With gasping there is flooding of the lungs with water, which is often contaminated with gastric contents. Theoretical differences between fresh and saltwater drowning exist. However, in the clinical situation many of these differences are subtle or may have been resolved by the time the victim arrives in the emergency center. Nevertheless, these abnormalities related to fresh water and salt water drowning should be identified and treated when present.

Fresh water drowning classically produces the following abnormalities: hemodilution (due to the aspiration and swallowing of large volumes of hypotonic fluid), hypervolemia (usually transient), hyperkalemia, hemoglobinuria (hypokalemia may also occur during hemodilution or with use of diuretics), and decreased serum sodium, chloride, and calcium concentrations.

Salt water drowning, on the other hand, produces hemoconcentration, hypovolemia (due to swallowed or aspirated hypertonic sea water producing a gradient for the translocation of plasma water into the alveoli and gastrointestinal tract), hypokalemia, particularly if diuretics are given, and increased serum sodium, chloride, and calcium concentrations.

Regardless of whether fresh water or salt water aspiration is involved, laboratory tests show decreased PaO_2 (arterial O_2), acidosis, leukocytosis, and decreased urinary output.

Acidosis is due to hypoxemia and/or peripheral hypoperfusion. Hypoperfusion may occur secondary to a generalized decrease in cardiac output or from changes in regional blood flow by the body's homeostatic mechanisms in an attempt to conserve perfusion to the heart, brain, and kidneys. Leukocytosis is due to demargination of white blood cells secondary to stress. Decreased urinary output is associated with albuminuria and hematuria by dipstick testing. If no red blood cells are seen on microscopic examination of the urine, this would suggest hemolysis and hemoglobinuria.

Radiographs reveal various forms of pulmonary pathology. Initially, patchy infiltrates suggestive of aspirated foodstuff, atelectasis, or evidence of diffuse pulmonary edema may be seen (Fig 1). Later developments include consolidation suggestive of pneumonitis or bronchopneumonia. In severe cases, there may be empyema or pulmonary abscess where food particles have been aspirated (Fig 2).

Pulmonary edema tends to be more extensive in salt water aspirations. In salt water drowning, intravascular water enters the alveoli along the concentration gradient created by the high osmolality of water aspirated into the alveolus. As a result, ventilation-perfusion abnormalities may persist longer after sea water (salt water) has been aspirated. However, most victims of near-drowning rarely aspirate only pure water, but rather aspirate a mixture that is contaminated with foodstuff. Therefore, the distinction between salt water and fresh water drowning may

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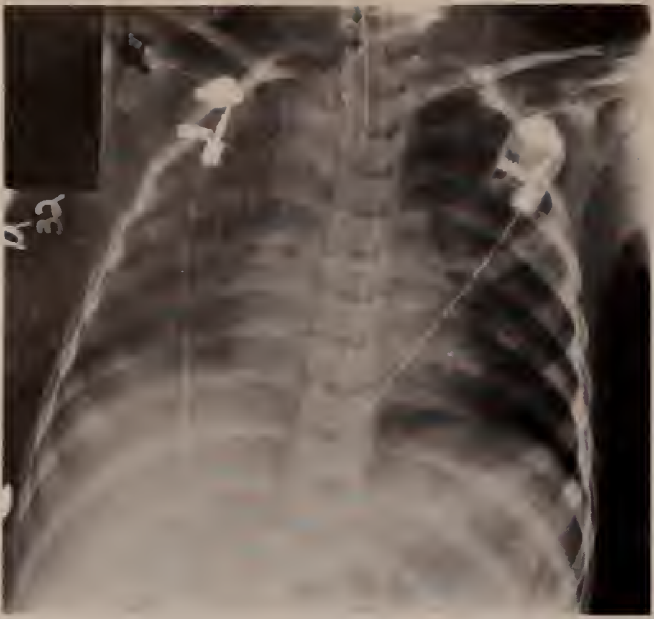

FIGURE 1. Chest film showing diffuse pulmonary edema. These are the early changes in near-drowning.

not be apparent due to secondary chemical or bacterial pneumonitis. This pneumonitis may complicate either type of near-drowning and is longer lasting than the abnormalities associated with the simple ventilation-perfusion mismatch due to the water itself.

Aspirated fluid soon creates an inflammatory alveolitis accompanied by capillary congestion and hyaline membrane formation. This exudate fills the alveoli and creates a high alveolar-arterial oxygen gradient. Lung compliance is greatly reduced by the presence of intra-alveolar and interstitial pulmonary edema, which is frequently hemorrhagic. Also, bronchospasm of terminal airways tends to augment ventilatory obstruction already present due to aspirated liquids. Later developments include organization of these hyaline membranes, which can cause prolonged aberrations in pulmonary function up to six to 12 months after the initial injury.

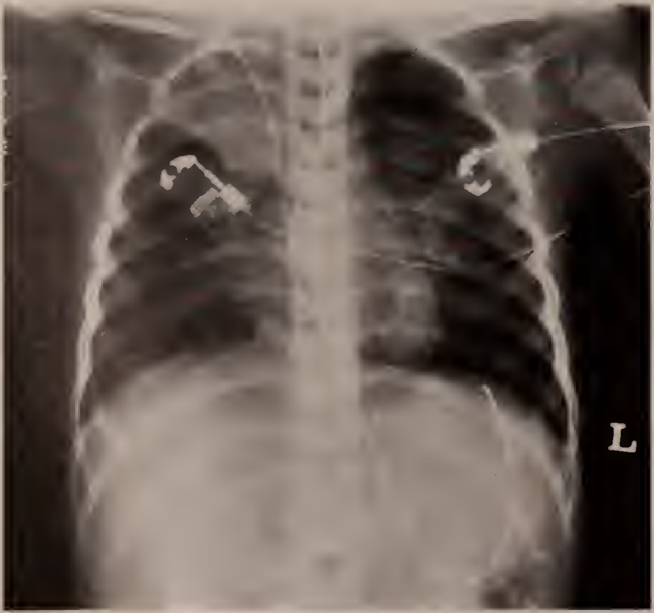

FIGURE 2. Chest film of same patient three days later showing right upper lobe collapse. Foodstuff was obtained from the right upper and right middle lobe bronchus using fiberoptic bronchoscopy.

MANAGEMENT

Speed in initiation of aggressive management is of primary importance, since irreversible central nervous system damage usually begins within three to four minutes of an asphyxial event. The key to preventing more serious central nervous system damage is rapid restoration of breathing. There should be maintenance of PaO_2 above 100 torr, correction of acidosis, and stabilization of cardiovascular function. The longer hypoxemia persists, the greater the neuronal injury and subsequent cerebral edema.

On the scene of drowning, basic cardiac life support should be instituted immediately. These resuscitative efforts should be continued until the transport team arrives to assume responsibility for the patient. The airway should be cleared of any particulate matter and the adequacy of air exchange by the patient assessed. Diving accidents with resultant neck fracture may be the cause of the near-drowning event in an older child. Thus, particular care should be taken to support the head and cervical spine while giving cardiorespiratory resuscitation. Mouth-to-mouth resuscitation should be instituted if spontaneous breathing is absent or inadequate. Closed chest cardiac massage should be begun if no pulse can be felt. Some authors recommend palpating the carotid artery for up to one full minute prior to beginning closed chest massage, especially in cold water immersions. This hesitancy in starting external cardiac massage is because of the significant risk of inducing ventricular fibrillation with CPR.

In the emergency department, no matter how alert a child appears, cardiopulmonary support should continue while an initial assessment of the patient is carried out. Pure oxygen should be administered to the patient either by face mask, ambu bag, or endotracheal tube until blood gas test results are available. Initial laboratory tests for all victims of near-drowning should include arterial blood gases, chest radiograph, complete blood count, and serum electrolytes.

Children who present to the emergency department in respiratory failure, or those who are comatose, should be endotracheally intubated. Gastric contents should be evacuated to prevent aspiration of swallowed water and foodstuff, and a nasogastric tube left in place. Often the child will have increased muscle tone or trismus, making visualization of the larynx difficult, even traumatic. In this situation, the child may be given a nondepolarizing muscle relaxant, such as pancuronium (0.15 mg/kg), vecuronium (0.15 mg/kg), or atracurium (0.4 mg/kg), in addition to sedation with diazepam (0.25–0.3 mg/kg) or thiopental sodium (3–4 mg/kg). Use of these drugs for muscle relaxation and sedation may allow a much smoother introduction of the endotracheal tube into the trachea.

The use of depolarizing muscle relaxants (such as succinylcholine chloride) should be avoided when possible since these agents may raise intracranial pressure and serum potassium levels. If muscle relaxants are used, the child should be gently ventilated with bag and mask prior to intubation. Cricoid pressure can be applied (Fig 3) to help prevent aspiration of gastric contents.

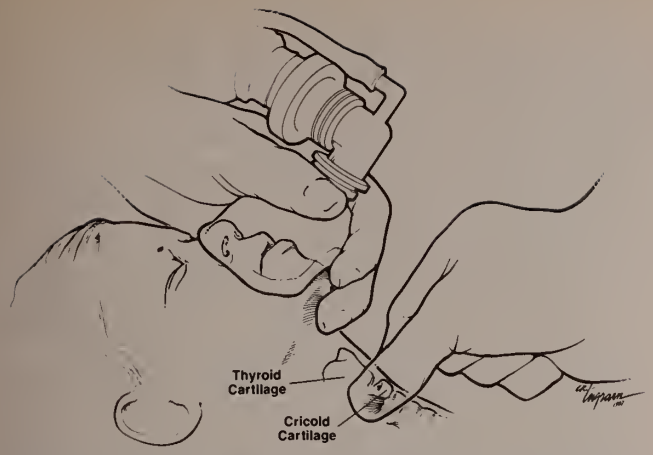

FIGURE 3. Anatomic sketch showing technique of the Sellick maneuver. Firm pressure is held over cricoid cartilage by thumb and forefinger. The cricoid is the only completely cartilagenous ring of the trachea so that firm pressure effectively occludes the esophagus and helps prevent passive regurgitation of gastric contents before tracheal intubation is accomplished.

Most children who undergo near-drowning will be overhydrated due to aspirated or swallowed water. Conservative fluid administration should be guided by right-sided and/or left-sided cardiac filling pressures. Hypotension may persist after reversal of hypoxemia and hypercarbia in the face of a low central venous pressure (<3–4 torr). In these cases, isotonic fluids, such as Ringer's Lactate or Plasmanate™, may be used to augment circulating blood volume. Most often, however, decreased blood pressure will be due to myocardial failure from hypoxemia. The use of cardiac inotropes would be preferable to fluid administration for support of arterial pressure and cardiac output in these instances, because giving excess fluid may promote cerebral edema. Dopamine is the primary drug of choice because it also conserves renal blood flow (Table I).

If the child is anemic, packed red blood cells may be given. The potential risks of transfusion must be balanced against the expected benefits. However, when dealing with regional or global cerebral ischemia, it has been shown that a hematocrit in the 28% to 32% range improves oxygen delivery to tissues due to a lower viscosity of the circulating blood. Acidosis should be treated primarily with hyperventilation to a PCO_2 of between 25 and 30 torr, support of cardiac output with judicious use of fluids, vasopressors, or both, and correction of hypoxemia. Use of bicarbonate should be reserved for those patients with a pH below 7.15 after the preceding measures have been instituted. This precaution is based on the fact that bicarbonate is very hyperosmolar. There are 1,000 mosm/L in the pediatric concentration of 5.4%. This high osmolarity adds to blood viscosity and may adversely affect cerebral blood flow.

With regard to hyperventilation, cerebral blood flow is linearly related to PaCO_2 between values of 20 to 80 torr. Acute reduction of PaCO_2 to a level of 25 to 30 torr may reduce intracranial pressure by reducing blood flow.[5] However, care must be taken when using more vigorous hyperventilation (producing levels of PaCO_2 of less than 25 torr). Such hyperventilation may reduce cerebral blood flow to levels inadequate to provide sufficient oxygen to the already injured brain cells.

TABLE I. Outline for Emergency Room Management of Near-Drowning Victim Based on Neurologic Presentation

	Neurologic Presentation	
Awake	Blunted	Comatose
Laboratory: Chest film Complete blood count Arterial blood gases	Laboratory: Chest film Complete blood count Arterial blood gases Electrolytes	Laboratory: Chest film Complete blood count Arterial blood gases Electrolytes
Oxygen	Oxygen with or without intubation	Endotracheal intubation, hyperventilation
Admit for observation	Admit to intensive care unit	Provide cardiovascular support Fluid restriction Diuretics Steroids ± Temperature control Paralysis Antibiotics ±
Discharge after 24 hours if stable	Careful observation Try to prevent second hypoxic episode At risk for cerebral edema—restrict fluids	Better prognosis: pH >6.9–7.0 No CPR needed Spontaneous respirations in emergency room Younger age Awake or blunted Decreased time of submersion Decreased water temperature

Fluid restriction to one-half to two-thirds of maintenance requirements may help prevent further brain swelling. Restriction of fluid intake may also reduce the complications associated with the syndrome of inappropriate antidiuretic hormone secretion (SIADH). With fluid restriction, however, urine output may decrease to 0.25 or 0.5 mL/kg/h. This range of urine production is acceptable, and is compatible with adequate renal perfusion. However, the best method for guiding fluid administration and for assessing the need for diuretics is to evaluate urine output in conjunction with several factors, such as peripheral perfusion, central venous pressure (CVP) readings (normal being 2–4 mm Hg in a supine, sedate child), and findings on neurologic examination.

If muscle relaxants are required to prevent posturing or to improve ventilatory status, continuous monitoring of the electroencephalogram (EEG) may be carried out, using a more simplified display of frontal and temporal lobe output (PSA-1® Life Scan®). These instruments permit detection of changes in neurologic condition that may not otherwise be appreciated in a child paralyzed by muscle relaxant drugs. The primary caretakers must be familiar with the use of these EEG monitors, which provide general information about cerebral function and are useful primarily for trend monitoring.

Another method being studied to help guide fluid and diuretic therapy in head-injured patients is the measurement of jugular bulb venous oxygen saturations. These determinations are obtained from a catheter inserted into the internal jugular vein and passed cephalad.[6] Oxygen saturations are followed periodically. Decreases below normal in venous saturation, or increases in arterial to jugular venous oxygen saturation differences, are interpreted as indicating inadequate cerebral blood flow. Use of jugular bulb venous oxygen saturations has not been studied extensively in children. Thus, it is unclear whether this monitor may favorably influence outcome for the pediatric patient in the years to come.

Methylprednisolone (4–8 mg/kg/day) or dexamethasone (1.0–1.5 mg/kg/day), given in divided doses intravenously every six hours, has been recommended to treat both pulmonary complications (such as pneumonitis) and cerebral edema. However, recent studies have shown that the use of steroids to treat hypoxic cerebral edema does not reliably produce an improvement in outcome and may hinder pulmonary healing.[7] Routine use of antibiotics is also controversial. If pulmonary aspiration of gastric contents is known to be present at the time of admission, many authors favor the use of antibiotics, especially since these patients have pulmonary infiltrates and are usually febrile. The drug of choice is penicillin, given at a dosage of 100,000–150,000 units/kg/day divided in four doses. Volume overload and cerebral edema may be ameliorated by the use of diuretics such as furosemide (0.5–1.0 mg/kg intravenously) or mannitol (0.25–1.0 gm/kg intravenously).

Intracranial pressure (ICP) monitoring may be carried out by use of a ventriculostomy or by the placement of a subarachnoid bolt. These techniques have been used as guides to hyperventilation and fluid therapy in patients with closed head injury, brain tumors, and hypoxic encephalopathy.[8–11] But for patients with hypoxic encephalopathy, several studies showed that, even if intracranial pressure was controlled at less than 10 to 15 mm Hg, outcome was not favorably affected. This finding is in contrast to the definite benefit achieved in patients with head injury or brain tumors in whom the ICP is controlled.[9,11] If an ICP monitor is used, the patient should be placed on prophylactic antibiotics to protect against staphylococcal infection of the cerebrospinal fluid. Limiting the use of these monitors to less than five days will reduce infectious complications.

In an attempt to improve outcome in the comatose near-drowning patient, Conn and associates[12] proposed a program for treating these children which they termed HYPER therapy. Their therapy is focused upon the five most common clinical states that these patients exhibit. These are hyperhydration, hyperventilation, hyperpyrexia, hyperexcitability, and hyperridigity.

The relative severity of these findings, according to the Toronto protocol, would then dictate use of corresponding measures for supportive care of the patient at risk for central nervous system sequelae (Table II).

Most of the measures listed in Table II have been widely accepted as useful. However, the use of barbiturate coma and deliberate hypothermia instituted after the drowning event remains controversial.[7,12–15] Barbiturate coma has not been shown to improve outcome significantly.[13,14] Pentobarbital and thiopental, which are the two barbiturates most studied, are both potent myocardial depressants in the dosages required to induce barbiturate coma. This effect on myocardial performance may reduce cardiac output, which in turn may further reduce cerebral blood flow.

TABLE II. The HYPER Therapy Program for Treatment of the Comatose Near-Drowning Child

Hyperhydration

1. Fluid restriction to one-half daily maintenance.
2. Diuretics: mannitol, 0.25–1.0 g/kg; furosemide, 0.5–1.0 mg/kg.
3. Therapy guided by intracranial pressure (≤ 15), central venous pressure (2–4 mm Hg), or pulmonary capillary wedge pressure (≤ 2) (cardiac index, 2.0–2.5 L/min/m^2).

Hyperventilation

1. $PaCO_2$ between 25 and 30 mm Hg, raise PaO_2 to above 150 mm Hg.
2. Positive end-expiratory pressure to lower fraction of inspired oxygen, which would cause lower pulmonary risk of O_2 toxicity and barotraumas.
3. Rotobed, frequent position changes (this decreases the risk of major ventilation/perfusion abnormalities due to atelectasis from prolonged neuromuscular paralysis).

Hyperpyrexia

1. Acetaminophen (10 mg/kg) to prevent temperature rise.
2. Cooling blanket (keep temperature of blanket at 30 ± 1°C).
3. Muscle relaxants to prevent shivering.

Hyperexcitability

1. Perform electroencephalogram early during admission.
2. Anticonvulsants (diphenylhydantoin is the drug of choice because it does not affect neurologic examination and inhibits secretion of antidiuretic hormone.
3. Barbiturate coma (daily measurement of serum barbiturate levels should be obtained to achieve the goal of 75–100 mg/L): phenobarbitone dose on day 1 is 50 mg/kg, and on day 2 it is 25 mg/kg.
4. Steroids: dexamethasone (loading dose, 0.2 mg/kg and maintenance dose, 0.1 mg/kg).

Hyperrigidity

1. Neuromuscular paralysis.

Calcium may reverse some of the adverse effects of barbiturates on cardiac function, but, unfortunately, calcium may be detrimental to the patient with hypoxic encephalopathy.[16,17] There is evidence to suggest that permanent neurologic injury due to hypoxia is related to abnormal fluxes of calcium across membranes in the central nervous system. Some authors have suggested that calcium channel blockers may be of benefit in the management of hypoxic encephalopathy.[16,17] However, the best results were obtained by pretreatment of animals with these drugs, before hypoxemia occurred, which is not possible in clinical practice. In addition, the side effects of calcium channel blockers, such as arterial vasodilation, may decrease arterial blood pressure and cerebral blood flow. Elevation of the intracranial pressure has also been reported due to cerebral vasodilation by the drug. Therefore, more investigation is required before these agents may be considered useful in the treatment of cerebral edema.[17]

The deliberate induction of hypothermia after the injury has occurred has not been shown to improve outcome. Furthermore, such hypothermia may make the patient more susceptible to sepsis due to secondary leukopenia.[18] But temperature control within normal limits does reduce cerebral metabolism and reduces injury.[7]

OUTCOME

Many papers have been published regarding outcome in near-drowning victims, but the results are difficult to interpret. Accurate, controlled, reliable data are badly needed for a number of reasons. This information could help clinicians in formulating methods and determining intensity of needed intervention. Clinical results could assist caregivers in counseling families about outcome expectations, and could allow realistic anticipatory guidance for families of survivors and for the victims' primary medical caretakers.

Many variables have been utilized in the past to predict outcome (Table III). The variables (prognostic factors) used most often can be grouped into three categories: neurologic status, cardiopulmonary status, and epidemiologic factors, the first variable, neurologic status, being the most useful. Orlowski[19] devised a list of five unfavorable prognostic indicators involving all three categories: age under three years; estimated maximum submersion time more than five minutes; with no attempt at resuscitation for at least ten minutes after rescue; pupils fixed and dilated when patient is seen in the emergency department; and initial arterial pH less than 7.1. Each indicator present was assigned one point; children with a score of more than two points were found to have a 5% chance of full recovery, while a score of less than two points correlated with a 90% chance of full recovery.

The Glasgow coma scale (Table IV) has been extensively utilized as a predictor of outcome in cases of near-drowning. Dean and Kaufman[20] found that 80% of patients with a Glasgow coma scale (GCS) score of under five either died or had permanent neurologic sequelae. Frewen et al[21] found that the Glasgow coma scale correlated with outcome as well as or better than the Orlowski score. This was believed to occur in part because two of the Orlowski score variables were history-dependent, whereas the Glasgow coma scale relies on more objective signs. In this study five out of six patients with a Glasgow coma score of three either died or were neurologically damaged, while all children with a score higher than eight had good recoveries.

TABLE III. Prognostic Factors Useful in Assessing the Near-Drowning Child

Factors Associated with Good Outcome	Percent
Glasgow coma score >5 in ICU	100
Orlowski score <2	90
Decerebrate in ER, improving, with cardiopulmonary stability	82
Time to first gasp	? ("excellent")
Brain O_2 extraction normal or greater	66
Factors Associated with Poor Outcome	**Percent**
Glasgow coma score <3 in ICU	67% dead, 33% damaged
Orlowski score >2 in hospital	95% dead or damaged
Flaccid in ER	91% dead, 9% damaged
Severely abnormal electroencephalogram	100% dead or damaged
Sustained, late increased intracranial pressure greater than 20 mm Hg	100% dead or vegetative
Apneic and fixed, dilated pupils in ICU (barring iatrogenic causes)	100% dead or vegetative
Apneic, no cardiac output in ER	100% dead or vegetative
Decreased brain O_2 extraction	100% dead

TABLE IV. Criteria for Patient Assessment According to the Glasgow Coma Scale

Eye Opening	
1	None
2	Response to pain
3	Response to speech
4	Spontaneous
Motor response	
1	None
2	Abnormal extension
3	Abnormal flexion
4	Withdrawal from pain
5	Localization of pain
6	Obedience to commands
Verbal response	
1	None
2	Incomprehensible
3	Inappropriate
4	Confused conversation
5	Oriented

Allman et al[18] used the Glasgow coma score in sequential assessments of patients to predict the outcome of severe near-drowning. Of 69 patients presenting to an emergency room (ER) with a GCS of three (flaccid), 37 had not improved by the time they arrived in the intensive care unit (ICU). Of those 37, 26 died and 11 suffered severe neurologic damage. Of 24 patients with a GCS of four or five in the ICU, 50% survived intact, while 25% died and 25% remained in a persistent vegetative state. Children with a Glasgow coma score of higher than five on arrival in the ICU all survived neurologically intact.

Turner and Levin[22] have devised a system of neurologic evaluation based on behavioral planes, classified as alert, obtunded, decorticate, decerebrate, or flaccid. Using these criteria, they follow changes in neurologic status over time to predict outcome. They found that of 36 patients who ranged from alert to decerebrate on their initial ER evaluation, 86% were discharged with normal neurologic function. However, out of 11 patients determined to be flaccid on initial exam, ten died and the 11th suffered severe neurologic sequelae. Orlowski[23] suggested that these prognosticators may be a useful alternative to his earlier system.

Finally Oakes et al[24] proposed yet another neurologic indicator of outcome. This prognostic indicator is based on the observation that all their near-drowning patients with a "severely abnormal" electroencephalogram (EEG) either died or remained comatose. Their criteria for a "severely abnormal" EEG were not described. Therefore, their results are not easily applied to patients in the clinical setting.

Another frequently studied neurologic prognostic factor in near-drowning victims is increased intracranial pressure (ICP). Nussbaum and Galent[11] noted that ICP and cerebral perfusion pressure (CPP) were valid predictors of survival after near-drowning, but these parameters did not predict whether there would be a good neurologic outcome. Frewen et al[21] and Sarnaik et al[25] found that successful control of increased intracranial pressure and cerebral perfusion pressure did not guarantee a normal outcome. Also, a late sustained elevation of ICP (for longer than 72 hours post-immersion) was thought to be a reliable predictor of a poor outcome. It was believed that such an elevation in ICP may be a sign of a profound primary neurologic insult, rather than being a secondary insult.

Bohn et al[26] showed that while barbiturate coma may help control elevations in intracranial pressure, improved neurologic outcome could not be proven. Further, some nonsurvivors of near-drowning whose intracranial pressure was normal showed cerebral edema and uncal herniation at autopsy. Similarly, Allman et al[18] monitored intracranial pressure in the majority of near-drowning victims with Glasgow coma scores of less than six, and initiated aggressive measures to control ICP. Nonetheless, they found that prevention of persistent ICP elevations of over 20 mm Hg while the patient was receiving therapy did not necessarily signify a good outcome.

Cardiopulmonary function has been used in attempts to predict outcome in near-drowning. The status of cardiopulmonary function is reflected in the vital signs recorded at the rescue site and in the initial examination in the emergency room, and by oxygen utilization in the setting of the intensive care unit. Pearn[27] listed ten prognostic guides, of which five directly reflected cardiopulmonary function. Of those five, the time to first spontaneous respiratory effort (gasp) was the best indicator of improved neurologic outcome. Children whose first gasp was within 20 minutes of rescue had an excellent prognosis, while those who were still apneic (with fixed and dilated pupils) in the ICU had a poor prognosis. These observations confirmed Orlowski's findings.[19] But of five near-drowning patients presenting to an emergency room with continued apnea and pulselessness, one died during the initial hospitalization, and four were discharged.[28] Of those four, two were vegetative, one had severe neurologic sequelae, and the fourth expired secondary to an obstructed tracheostomy.

Status of the patient on arrival to the emergency room was reviewed by Weinberg[29] who found that 33 out of 100 near-drowning victims were comatose on arrival to the ER. Of those patients, 22 were apneic and had no cardiac output, having either asystole or ventricular fibrillation. All 22 either died or suffered severe brain damage. Alternatively, of the remaining 11 patients who presented comatose but with an adequate cardiac output, 82% had a good outcome. Nussbaum and Galent[11] reported a rate of intact neurologic survival (37%) in initially comatose near-drowning victims. But Bell et al[30] observed a rate of intact neurologic survival of only 14% in a similar patient population. Controversy exists as to the reasons for these seemingly disparate rates,[31] probably reflecting difficulties inherent in studies on prognosis of near-drowning.

Another indicator of cardiopulmonary function that has been studied in children is the cross-brain oxygen consumption, or the arteriovenous O_2 difference of the cerebral circulation. Frewen et al[32] found that survival of brain injured children, of whom four out of six were near-drowning victims, could be predicted by a normal or greater than normal O_2 extraction by the brain, as measured in the jugular bulb. All the nonsurvivors had a less than normal extraction of O_2 by the brain. All the survivors, three out of six near-drowning victims, were either neurologically normal (two patients) or had only mild, improving neurologic sequelae (one patient).

Initial arterial pH and partial pressures of gases were not consistently useful prognostic indicators of outcome in cases of near-drowning.[19,27]

Other proposed prognostic indicators in near-drowning, of variable significance, include neutropenia[26] and several epidemiologic factors such as the following: fresh water as opposed to surf,[27] whether CPR was required at the presenting hospital,[20,24] whether required CPR was actually administered,[19] level of training of the resuscitator,[9] duration of submersion time, and age of the patient.[19,20]

It is clear that cold water submersion has less mortality and a lower morbidity rate than warm water submersion.[32]

It is apparent in the above discussion that we do not have a system for outcome prediction that is 100% reliable in near-drowning cases. Problems with past and currently used systems are numerous. Most of the findings are based on retrospectively obtained data, from chart reviews, and from historically derived information. Subjectivity is difficult to eliminate in such studies: immersion times and temperatures, durations of resuscitation, etc, are frequently estimated and not measured. Also, medical histories are often emotionally influenced, inaccurate, and difficult to verify.

Few of the studies of survival controlled for patient temperature during evaluation. Neurologic function is decreased with hypothermia alone. Thus, attempts at outcome prediction based on early information, obtained either at the site of immersion or in the emergency room, may be premature. Definitions of entities such as "abnormal EEG" and "good outcome" are, more often than not, lacking in clarity and precision. Finally, the most difficult aspect of predicting outcome is the knowledge that occasionally a patient will survive intact, despite the most dire presenting symptoms, defying all the odds.

In conclusion, every near-drowning victim must be vigorously and aggressively resuscitated as early as possible, regardless of initial presentation. Our current ability to predict outcome (outlined in Table III) seems to be most useful for realistic parental counseling about likely outcome, preparing parents for the worst without removing hope. In addition, some data may be helpful in predicting which survivors may need closer follow-up by their primary medical caretakers, looking for subtle neurologic deficits resulting from a prior near-drowning episode.

At present, we do not think that any of the prognostic indicators, considered either alone or in combination with others, can be used to justify withholding initial conventional resuscitation. But use of these prognostic indicators may be useful in determining which patients might be candidates for controversial or experimental therapeutic regimens.

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See also pp 411-413 and 434-436.

FROM THE LIBRARY

RESCUE BREATHING AND CARDIAC MASSAGE: FIELD EXPERIENCES OF THE CITY OF NEW YORK FIRE DEPARTMENT

. . . New York City firemen are always being called for emergency resuscitation in a variety of injuries. Because of the promising results in smoke exposure cases our medical department has felt that the best approach to the restoration of breathing was rescue breathing. We therefore continued this method until January, 1962, when, in addition to the method previously described, we added external cardiac massage. . . .

We took up this method in conjunction with rescue breathing after careful education of every fireman. First, we trained a key group of men and taught them the anatomy, physiology, and methodology of external cardiac massage. We also showed them the moving picture film *Life in Your Hands*. These men, in turn, visited the firehouses and gave individual instruction to all members of the department. The author gave a one-half hour lecture on this subject on WUHF, the New York City television station, and the live talk was taped. It is now being televised at regular intervals and is received in all the firehouses. Following the television broadcast, the men are put through the paces of rescue breathing and external cardiac massage.

Since our initial publication we have collected data on 444 cases of resuscitation; of these, 331 were rescue-breathing cases, and 113 were cases of rescue breathing and external cardiac massage. Rescue breathing consists of mouth-to-mouth or mouth-to-mask resuscitation, with or without the use of a resuscitator supplying 100 percent oxygen. External cardiac massage is always accompanied by rescue breathing.

. . . In victims of drowning accidents, breathing was restored by rescue breathing in a fair proportion of cases. The added procedure of external cardiac massage had disappointing results when compared to the results of rescue breathing alone. . . .

GAMLIEL SALAND, MD
(*NY State J Med* 1963; 63:3521-3523)

Management of submersion hypothermia: Successful resuscitation of a 14-year-old girl

BURTON L. HERZ, MD; FREDERICK A. COVILLE, MD; CYNTHIA A. KOCSIS, MD

Near-drowning is a common problem in the United States and constitutes a significant therapeutic challenge in cardiopulmonary resuscitation.[1] Submersion in frigid waters presents a second insult related to severe hypothermia, with survival and recovery being uncommon.[2] However, a hypothermic event may paradoxically allow for organism preservation during the submersion asphyxiation time. Failure to understand the pathophysiology of submersion hypothermia may lead to unattempted or poorly performed resuscitation and subsequent death.[3]

This report reviews the successful resuscitation and complete recovery, without neurologic sequellae, of a 14-year-old girl following prolonged submersion. Initial control of cardiopulmonary resuscitation followed by expeditious but controlled core rewarming with multiple active core rewarming techniques is discussed.

CASE REPORT

A 14-year-old girl weighing 35 kg arrived at the Coney Island Hospital Emergency Room (ER) at 9:50 PM. At 9:42 PM she had been removed from a van submerged for 35 minutes in 25 feet of cold (35°F) salt water. It was possible to document the submersion time because the van struck a fire alarm box before entering the water and the impact triggered the alarm at 9:07 PM. Witnesses at the scene stated that the van sank immediately upon hitting the water. The fire department responded within minutes with conventional fire fighting equipment but personnel were unable to see well enough to effect rescue.

The Police and Fire Department Submersion Rescue Team was nearby when summoned and responded promptly. With proper lighting and other equipment, they were able to see the van lying passenger side up with the window opened. Submersion was confirmed as a rescue team failed to identify an air pocket.

The patient was the first victim extracted from the van. She and one other victim were brought to our facility in separate ambulances. The three other victims were taken to another nearby hospital.

On removal from the van, the patient was unresponsive and had no pulse or spontaneous respirations. Her pupils were fixed and dilated. Intubation, intravenous access, and cardiopulmonary resuscitation (CPR) were performed in the field.

On the patient's admission to the ER, an electrocardiogram revealed cardiac asystole. A chest radiogram revealed pulmonary edema. Initial blood gas analysis on 100% oxygen revealed a pH of 7.01, a P_{CO_2} of 42 torr, and a P_{O_2} of 18 torr (corrected for temperature). The patient's core temperature measured 82°F by esophageal temperature probe. The profound metabolic acidosis was treated with a total of 489.5 mEq of sodium bicarbonate. After several minutes, sinus bradycardia began, and the blood pressure was 70/0 mm Hg. The patient was started on an isoproterenol drip at 2 mg/min and a dopamine drip at 11 mg/kg/min to achieve a cardiac rate of 90 and a systolic blood pressure of 100 mm Hg.

Following stabilization of the cardiopulmonary course, rewarming was begun using warmed blankets, nasogastric irrigation, and peritoneal lavage through a Tenchkoff catheter inserted subumbilically. Gastric lavage proceeded at a rate of 200 cc every 4–5 minutes and peritoneal lavage was accomplished at a rate of 2 liters every 20 minutes with a dwell time of ten minutes. All intravenous and irrigation fluids were prewarmed to 100°F. The inspired O_2 was warmed to 99°F through a cascade humidifier with a volume of 6 L/min.

The patient's temperature was permitted to rise at a rate of 2°F per hour. When the esophageal probe recorded a temperature of 96°F, the pulse rate was 80 beats/min and the blood pressure was 130/70 mm Hg, without pharmacologic support.

The patient was placed on a volume cycled respirator with positive end-expiratory pressure (PEEP) of 15 cm H_2O, after which the Pa_{O_2} improved to 196 mm Hg with a fraction of inspired oxygen (Fi_{O_2}) of 1.0. The pH was 7.16 and the P_{CO_2} was 55 mm Hg. After six hours, the patient's pH was corrected to 7.31 and her Pa_{O_2} was 88 mm Hg, with an Fi_{O_2} of 0.5 and a PEEP of 15 cm H_2O.

Two hours after admission generalized seizures developed, lasting one hour. The seizures were controlled with 20 mg/kg of phenobarbital over a one-hour period and 20 mg of dexamethasone intravenously. Two hours later the patient was placed on an antiseizure regimen of phenytoin, 100 mg every six hours, and the phenobarbital was discontinued. Dexamethasone was continued at 4 mg intravenously every six hours for two days.

Within 24 hours after admission spontaneous respirations were noted. The patient was responsive to deep pain and her pupils were reactive to light. A computed tomographic scan was normal. An electroencephalogram showed slow function with no focal abnormalities. The patient was extubated 72 hours after admission.

On the fifth post-resuscitative day the patient's movements became more purposeful and she began to speak. During the next seven days she made remarkable progress in higher cerebral function, and she was discharged 25 days after admission. Physical therapy was continued on an outpatient basis.

Three months after the accident, the patient still exhibited some minor deficits in fine coordination as well as some residual amnesia of the events surrounding the accident. Otherwise, she was neurologically normal and functioning at an intellectual level appropriate for her age. One year after the accident, all fine coordination deficits had reversed and she was able to resume all her previous activities.

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DISCUSSION

After careful review of the literature, we believe this patient is probably one of the oldest survivors, without neurologic deficit, of prolonged submersion hypothermia (35 minutes). In view of the rapid recovery in her PaO_2 and the abrupt clearing of her chest film, asphyxia rather than near-drowning is probably the insult she sustained. The asyphyxia could have been a result of several mechanisms. The most common mechanism seen in 95% of adults is laryngeal spasm. Another mechanism more commonly seen in children is the mammalian diving reflex. This reflex occurs when facial cooling triggers apnea and circulation is shunted to the heart and brain preferentially.[4] This primitive reflex is present in young children but not usually in young adults. However, the presence of an active mammalian diving reflex in a 14-year-old is a conjecture worthy of consideration.

The pathophysiologic effects of hypothermia paradoxically allow for protective maintenance of the organism.[5] Severe hypothermia allows for the dramatic reduction in arterial partial pressure of oxygen and a markedly reduced cardiac output. This phenomenon is paramount to the survival of the organ and organism during hypothermia.

The treatment of both victims who were taken to our hospital was similar once they arrived in the ER. The one patient who survived was small-framed, with minimal body fat, and was noted to be severely hypothermic. The nonsurviving patient had a much larger frame with more adipose tissue, and was normothermic when first seen. The difference between survival and nonsurvival in the two patients may be related to the protective effect exerted by severe hypothermia.

In extreme hypothermia, where core temperatures fall below 68°F, the patient may appear lifeless and it may be clinically impossible to differentiate between the salvageable and the unsalvageable. Death cannot be determined until the patient is normothermic. Therefore, all extremely hypothermic victims should undergo vigorous resuscitative techniques, with CPR being started before rewarming is attempted.[4] A poor understanding of these physiologic principles following severe hypothermic immersion could lead to an unsuccessful resuscitation. This would be due either to failure to attempt resuscitation of a patient who appears to be dead or to a poor understanding of the balance required between the rate of rewarming and the increase in metabolic rate. Increases in metabolic demand seen with rewarming should not outpace cardiopulmonary resuscitation, to avoid organ infarction through relative hypoxia.

In severe hypothermia, with core temperatures less than 89°F, many patients present in cardiac arrest secondary to ventricular fibrillation.[6] Our patient presented in asystole. Although asystole is seen less frequently, it has been argued that it is the resuscitation efforts and myocardial stimulation that create ventricular fibrillation.[4]

Another situation requiring extreme caution occurs with patients who have decreased yet present respiratory efforts as well as severe bradycardia. These patients may in fact be in an adequate state of perfusion relative to metabolic needs and will not benefit if rewarming precedes stabilization of the cardiopulmonary function.

Extreme caution must be exercised during resuscitation so that the commonly seen metabolic acidosis is not corrected too rapidly with intravenous bicarbonate. Otherwise, refractory terminal ventricular fibrillation may result.[7] This form of ventricular fibrillation has been reported to be unresponsive to electrical or pharmacologic cardioversion unless the core temperature is brought back to 82°F.[8] Repeated attempts at defibrillation before 82°F is reached may result in myocardial damage.[7] Passive rewarming may prevent further heat loss but is of little value when used alone at temperatures below 82°F. Rewarming, however, should not precede cardiopulmonary stabilization. Until a good cardiac output is achieved, rapid rewarming may be detrimental to organ survival.[11]

Active shell rewarming has been demonstrated to achieve survival similar to core rewarming in an experimental model. However, the added length of time required for rewarming and therefore resuscitation are pitfalls for complications, such as afterdrop in core temperature and intermittent phases of hypotension related to returned acidotic products from the peripheral venous pool.

Active core warming was accomplished in this case using four recommended techniques[4,8]: Intravenous fluids were warmed to 100°F. The flow rates and total volume were titrated using appropriate invasive cardiac monitoring to maintain adequate intravascular volume and adequate perfusion as the patient was warmed. Intragastric lavage with normal saline at a temperature of 100°F was also used. A high rate of flow is possible, but caution should be exercised to prevent aspiration. Although not used in the treatment of this patient, rectal lavage with warm fluid can also be used. A warmer attached to the ventilator was used to bring the gas mixture to 99°F. This method primarily prevents further reduction in body heat. Originally devised for field use, this technique results in an insufficient rate of rewarming if used alone and has been reported to have a high mortality if used without other rewarming techniques in cases of severe hypothermia.[4] Peritoneal lavage was used with rapid infusion (6L/h) of fluids at 100°F with a minimal dwell time (ten minutes). Rates of up to 12 liters per hour can be achieved using two peritoneal dialysis catheters if conditions warrant it.

Other core rewarming techniques include chest irrigation and cardiopulmonary bypass. Chest irrigation may be performed by tube thoracostomy bilaterally or when open thoracotomy for direct cardiac massage becomes necessary. Partial cardiac bypass using groin canulation is technically more difficult than open thoracotomy for chest irrigation but easier than open thoractomy with right atrial canulation. In addition, partial cardiac bypass has the advantage of being the fastest heat exchange method known, and it can also support the failing myocardium. However, rewarming by peritoneal lavage after CPR is begun will probably find the widest acceptance and be most readily applied for treatment of accidental severe hypothermia.

The successful resuscitation of the submersion hypothermia victim is most affected by the aggressiveness and timing of resuscitation of cardiopulmonary function. We believe that peritoneal lavage offers the best method of heat exchange with easy performance and is available in almost all emergency centers. This would obviate the need for transfer to a tertiary center. Transportation to a tertiary center may result in inadequate performance of CPR in transit while rewarming is occurring through passive and/or active means.

The old adage still holds true: "The patient is not dead until he is warm and dead."

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See also pp 411–413 and 427–433.

Amebic abscess of the liver

A report of ten cases

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Although amebiasis is primarily considered a tropical disease, its prevalence among the untravelled population in the United States is as high as 5%.[1] In areas with a multiethnic population, the prevalence is probably higher.[2] Liver abscess is the most serious complication of amebiasis, occurring in about 1% of patients,[3] and may prove to be fatal if an early diagnosis is not made. A suspicious medical history and serologic tests appear to be the most important factors in making an early diagnosis. In this study, we report our experience with amebic liver abscesses, with emphasis on diagnosis and treatment.

CASE REPORTS

We reviewed the charts of patients admitted to the City Hospital Center at Elmhurst, Queens, NY, who were discharged with a diagnosis of liver abscesses between January 1977 and July 1987. Out of a total of 22 patients with the diagnosis of liver abscess, ten had hepatic amebic abscesses. The diagnosis of amebic abscess was based on a history of recent travel abroad, ethnic background, results of serologic studies, imaging studies of the liver, and response to antiamebic therapy. In one patient in whom serologic tests were not done, the diagnosis of amebic abscess was made at an exploratory laparotomy. In another patient in whom the serologic findings were negative, the diagnosis was based on the response to antiamebic therapy and the nature of the aspirated material.

Age, Sex, Background. There were ten patients who were diagnosed as having amebic liver abscesses, of whom nine were men and one was a woman. The age range was from 23 to 54 years. Nine of the ten patients were immigrants who had been living in the United States for periods ranging from three months to ten years (Table I). Countries of origin were Colombia, Cuba, Ecuador, Peru, India, and the United States. One patient admitted to homosexual practices, and another was an intravenous drug abuser.

Symptoms and Signs. The symptoms included pain in the right upper quadrant or epigastric pain, nausea, vomiting, chills, weight loss, anorexia, and fever. Fever was the most predominant symptom, occurring in all ten patients, followed by abdominal pain in nine patients. Three patients had a palpable abdominal mass. One had clinical jaundice. Of the ten patients, the correct diagnosis was only suspected in one patient at the time of admission.

Laboratory Data. Mild anemia (hematocrit < 35%) was noted in four patients. Leukocytosis (white blood cell count > 10,000/mm^3) with predominantly polymorphonuclear forms was present in all ten patients. Eosinophilia was not noted in any of the ten patients. All patients had a normal prothrombin time. Of the liver function tests, the alkaline phosphatase was elevated (range, 124–531 IU) in eight of the patients in whom it was tested. Elevations in aspartate aminotransferase (AST) ranged from 32 to 269 IU and alanine aminotransferase (ALT) from 9 to 241 IU (Table II). Stool for culture and ova and parasite tests were only sent for analysis from one patient, and these findings were negative.

Serology. Serologic tests were performed in all but one patient. The tests included counterimmunoelectrophoresis (CIE), enzyme immunoassay, and radioimmunoassay. The details of the results are outlined in Table III. The CIE was positive in seven of

TABLE I. Ethnic Origin, Age, Sex, and Duration of Stay of Patients

Patient	Origin	Age (yr)	Sex	Duration of Stay in US at Time of Admission
1	Peru	46	M	Three years
2	Colombia	41	M	Three months
3	India	23	F	Six months
4	Colombia	25	M	Three and a half years
5	Ecuador	54	M	Four months
6	Cuba	30	M	(not available)
7	United States	33	M	Trip to Mexico four years prior to admission
8	Colombia	72	M	> Ten years
9	India	38	M	Six years
10	India	44	M	One year

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TABLE II. Significant Laboratory Findings

	No.	%
Leukocytosis (> 10,000/mm^3)	10/10	100
Eosinophilia	0/10	0
Alkaline phosphatase elevation	8/8	100
(> two-fold elevation)	6/8	75
Serum glutamic oxaloacetic transaminase (> two-fold)	3/10	30
Serum glutamic pyruvic transaminase (> two-fold)	2/10	20

the eight patients in whom it was done; one was a false negative.

Imaging Studies. Most abdominal films were nonspecific, but the chest films showed elevated right hemidiaphragms and pleural effusions in three patients. Nine patients underwent ultrasonography that suggested an abscess. Seven of these nine patients showed involvement of the right lobe of the liver. Of all five patients who had liver-spleen scans, the right lobe of the liver was involved in four patients and the left lobe in one. On isotopic scans, these abscesses were cold, space-occupying lesions without diffuse parenchymal changes. Only five patients had computed tomographic (CT) scans of the abdomen and all CT scans showed evidence of liver abscesses—four occurring in the right lobe and one in the left lobe. In summation, nine patients had involvement of the right lobe, and two had involvement of the left lobe. Nine of the ten patients had solitary lesions, while in one (patient #2), circumscribed foci were located in more than one lobe.

Treatment. All patients received medical therapy that consisted of metronidazole, 750 mg orally three times a day for ten days. In addition, five patients had treatment with iodoquinol, 650 mg orally three times a day for 20 days. All patients responded to therapy, with resolution of the abscess in each case. Drainage of the abscess was performed in three cases. In one case, an open surgical drainage was done at exploratory laparotomy. In two other cases, CT-guided percutaneous aspiration of the abscess was carried out because of fear of impending rupture. One patient had a secondary infection with *Streptococcus milleri*.

DISCUSSION

In order to diagnose an amebic abscess, a high index of suspicion is necessary. Early diagnosis is important because an amebic abscess can be easily and effectively treated. Although an amebic abscess occurs mainly in the immigrant population (nine out of the ten patients described here were immigrants), it can occur in the untravelled American population. One study reported that 8% of patients had no history of travel abroad.[4] Predominance among male patients is an accepted feature.[4] Nine out of the ten patients described in this report were men.

Fever was the chief complaint in all these patients (100%) followed by abdominal pain in the right upper quadrant in nine of the ten (90%). Other studies[4,5] have noted similar features, but fever was found to a lesser degree—in about 75% of patients. Diarrhea was not a part of the presenting symptoms of these patients, but two other studies have noted diarrhea to be present in 14–40% of patients.[4,5] A history of previous intestinal amebiasis is obtained in only 4–14% of patients.[4,6] Clinical jaundice may or may not be present.[4-6] Tenderness in the right upper quadrant was present in 90% of these patients. Other studies have quoted figures ranging from 64% to 85%.[5,6]

A correct admitting diagnosis is made in less than 20% of patients.[6] An initial diagnosis of amebic abscess was made in only one patient in this group (10%).

TABLE III. Serology

	Tests*			
Patient	CIE	IHA	EIA	RIA
1	0	0	0	0
2	0	0	+	0
3	+	0	+	0
4	+	0	+	0
5	+	0	0	0
6	+	0	0	0
7	+	0	0	0
8	−	0	0	0
9	+	+	0	0
10	0	0	0	+

* CIE, counterimmunoelectrophoresis; IHA, indirect hemagglutination; EIA, enzyme-linked immunoabsorbent assay; RIA, radioimmunoassay; 0, not done; −, negative; +, positive.

With regard to the routine laboratory studies, elevation of alkaline phosphatase appears to be the most consistently noted abnormality, being elevated in the majority of patients. Six out of eight patients in whom it was estimated showed a greater than two-fold elevation in alkaline phosphatase. Minimal elevations in serum glutamic oxaloacetic transaminase and serum glutamic pyruvic transaminase may be seen, but they are usually not more than twice the normal values. Leukocytosis is usually present, but eosinophilia is not a feature of this disease. Hypoalbuminemia may occasionally be seen.[6]

A variety of imaging studies can be used to locate the lesion—ultrasound, liver scan, and CT scan. Ultrasonography will distinguish solid tumors from abscesses or cysts, but cannot distinguish between the latter two conditions. A liver scan is useful in differentiating a cyst from an abscess. Although all three modalities will aid in the diagnosis, the general consensus appears to be that an ultrasound examination combined with liver scanning should be sufficient to make the diagnosis.[4,6,7] No additional information is obtained by resorting to a CT scan. The abscess is solitary in about 70% of cases and is usually located in the right lobe[4]; 90% of the patients studied had solitary lesions, with 90% of these lesions situated in the right lobe.

Serology is a vital diagnostic tool in confirming the diagnosis of amebic liver abscess. The list of available serologic tests includes indirect hemagglutination, indirect .immunofluorescence, counterimmunoelectrophoresis, immunoelectrophoresis, gel diffusion precipitin tests, complement fixation, latex agglutination, and enzyme-linked immunoabsorbent assay (ELISA).[8-12] Serologic tests are positive only in patients with invasive conditions such as amebic colitis or amebic liver abscess. Asymptomatic carriers usually test negative.[12] The sensitivity of these tests ranges from 95% to 99% in the presence of a liver abscess.[9] The indirect hemagglutination test is extremely sensitive, and a negative result (< 1:128) means that the invasive amebiasis never occurred. The indirect hemagglutination test may remain positive for up to 20 years[12] after an invasive infection and is, therefore, not suitable for diagnosis, especially in an endemic area. It appears more suited for epidemiologic studies. Counterimmunoelectrophoresis has the advantage that it can be performed quickly (30–60 minutes). Gel diffusion tests usually become negative six months after the infection and are, therefore, more suited for the diagnosis of acute conditions.[12]

To aspirate or not to aspirate is a major question. The majority of patients do not require aspiration.[2,13,14] Aspiration is recommended only in those patients with impending rupture or those who fail to improve after five days of chemotherapy.[15] Mortality for cases after rupture is 11%,[14] whereas the mortality in an uncomplicated liver abscess is 3%.[16] The aspirate is usually chocolate-colored, and it is extremely rare to find ameba in the aspirate. Failure to demonstrate the organism in the fluid, however, does not negate the diagnosis.

The preferred treatment is metronidazole, 750 mg three times a day for ten days, followed by iodoquinol, 650 mg three times a day for 20 days.Chloroquine, when used alone in a dose of 500 mg daily for ten weeks, has also been found to be effective.[16] Most patients respond within 48 to 72 hours.

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Pneumomediastinum induced by inhalation of alkaloidal cocaine

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The incidence of illicit cocaine use has risen to epidemic levels, particularly the use of the volatile, alkaloidal form of the drug known as "crack." Many of the medical complications of cocaine use have been previously described.[1] We report another case of a recently recognized complication of alkaloidal cocaine abuse, pneumomediastinum.

CASE REPORT

A 27-year-old man without known history of pulmonary disease presented with pleuritic chest pain, sore throat, and odynophagia. He admitted to several hours of alkaloidal cocaine inhalation coupled with strenuous Valsalva maneuvers. Physical examination was notable for left neck crepitus and Hamman's sign ("crunching" heart sounds over the precordium). A chest roentgenogram revealed pneumomediastinum and subcutaneous emphysema in the neck and precervical spine areas (Fig 1). Flexible laryngoscopy, barium swallow test, spirometry, lung volume analysis, and single breath diffusing capacity for carbon monoxide were all within normal limits. The patient was treated with supplemental oxygen. Within 24 hours his symptoms resolved; his chest roentgenogram revealed partial resolution of the pneumomediastinum, and he was sent home.

DISCUSSION

Pneumomediastinum develops as a consequence of alveolar rupture, with air dissecting within fascial planes, along vascular sheaths, and into the mediastinum.[2] Air may further dissect into the soft tissues of the neck causing subcutaneous emphysema and throat

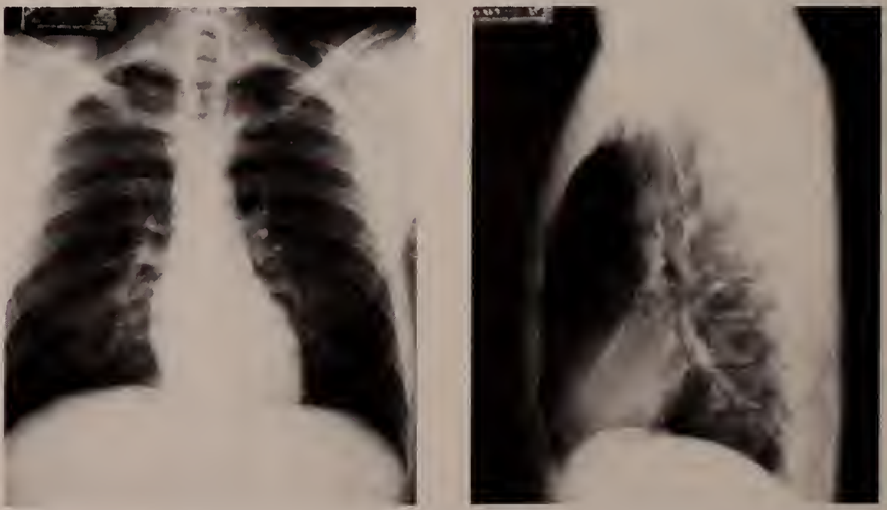

FIGURE 1. Chest roentgenograms showing pneumomediastinum in a patient two hours after inhalation of alkaloidal cocaine.

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pain. Macklin[3] postulated that only those marginal alveoli that abut the connective tissue of the vascular sheaths will rupture because of the tension between alveoli and adjacent structures. Such tension would increase in the overdistended alveoli of patients taking deep, vigorous inhalations—the likely mechanism of alveolar rupture in this patient.

The Valsalva maneuver, frequently employed by cocaine abusers to augment drug absorbtion, has also been postulated to cause alveolar rupture by creating a pressure imbalance between the alveolar and capillary structures. Munsell[2] suggests that during the Valsalva maneuver alveolar pressure increases due to the direct application of positive pleural pressure, while pulmonary capillary pressure decreases as a consequence of the decreased venous return to the right heart, with resultant decreased pulmonary capillary blood volume. The resultant difference in pressure could lead to alveolar rupture and pneumomediastinum.

A review of the literature revealed eight cases of cocaine-related pneumomediastinum, each with an inconsequential clinical course.[4-10] The benign course demonstrated by this case, and by previous cases, suggests that hospitalization and an extensive workup may be unnecessary for this condition. The probable cause of the pneumomediastinum is likely the respiratory maneuvers employed by these patients, rather than the effects of the drug itself. The accumulation of cases of pneumomediastinum following the inhalation of alkaloidal cocaine warrants this additional report of an entity only recently recognized, but certain to become a part of every internist's lexicon.

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Unusual autoimmune manifestations in furosemide-associated hypersensitivity angiitis

ROBERT Y. LIN, MD

Hypersensitivity reactions to sulfa-related drugs are both common and varied in their clinical manifestations.[1] One such drug is furosemide, which has been associated with allergic vasculitis, serum sickness, and bullous hemorrhagic eruptions, as well as other suspected immunologically mediated processes such as thrombocytopenia.[1-3]

The numerous reports of furosemide-associated hypersensitivity reactions have not, however, previously described drug-induced lupus. In the patient reported here, multisystem manifestations included mucocutaneous, renal, and hematologic sites. Some of these clinical findings were more consistent with an autoimmune disorder than with a typical allergic vasculitis which is the most common form of furosemide-induced hypersensitivity reaction.[1,2]

CASE REPORT

A 75-year-old man was admitted to Metropolitan Hospital on July 17, 1986, with a diffuse cutaneous eruption. One week prior to admission he had experienced the acute onset of a rash involving first the trunk and then the extremities. The patient stopped taking all his medications, which included furosemide, digoxin, phenytoin, and nifedipine, after which the rash diminished and was improving until two days prior to admission. At that time the patient was seen by a physician who represcribed furosemide. After the patient took furosemide again, fever, malaise, and an exacerbation of the rash developed. The patient was not taking any other medications during this period of time.

There was a history of cranial surgery for resection of a benign bone tumor 13 years prior to admission, and the patient had been treated with phenytoin for many years for a subsequent seizure disorder. The patient also had a history of hypertension and atherosclerotic heart disease for which he was being treated with digoxin, furosemide, and nifedipine.

The patient's temperature on admission was 39°C. There was diffuse involvement of the trunk, arms, and legs with a purpuric maculopapular rash (Fig 1). A well-healed scar over the right frontal scalp and a 1 cm ulcer on the right side of the tongue were noted. The examinations of the heart, lungs, abdomen, and neurologic system were unremarkable.

Laboratory examination revealed the following: hemoglobin, 10.8 g/dL; platelet count, 39,000/mm^3; white blood cell count, 16,900/mm^3 with 10% eosinophils. The initial creatinine and blood urea nitrogen levels were 1.4 mg/dL and 14 mg/dL, respective-

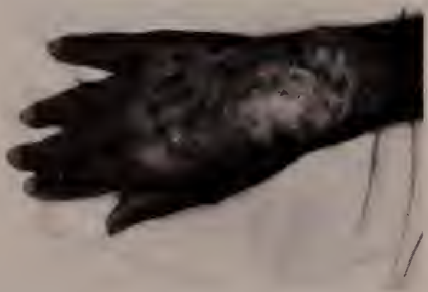

FIGURE 1. Purpuric lesions involving the upper extremity.

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ly, and rose progressively within the next seven days to 4.7 mg/dL and 53 mg/dL, respectively. The erythrocyte sedimentation rate was repeatedly within normal limits (15 mm/h). Urinalysis showed variable proteinuria, and numerous red and white blood cells were noted. No bacteria were present. A direct IgG Coombs' test was repeatedly positive (2+). The level of C4 was 14 mg/dL (normal, 20-50 mg/dL); the C3 level was normal. The Raji cell assay for immune complexes was elevated at 14.7 mg aggregated human globulin equivalents/mL (normal, <12.5). Antinuclear antibody (ANA) was present in a diffuse pattern at a titer of 1:160 (normal, <1:20). Anti-double-stranded DNA, Smith, and ribonucleoprotein antibodies were not detectable. HB_sAg was not present, and Epstein Barr viral capsid antigen IgG antibody titers were low and did not show a serial rise (1:40). Cold agglutinins were detectable up to a titer of 1:16,000. Tests for anti-single-stranded DNA and antihistone antibodies were not obtained. A biopsy of a purpuric lesion showed leukocytoclastic angiitis (Figs 2,3).

The patient was treated with high dose corticosteroids (prednisone, 80 mg/day), became afebrile, and showed gradual improvement of the cutaneous and oral lesions. Therapy with nifedipine, digoxin, and phenytoin was reinstituted. Five weeks after admission, the hemogram, creatinine/blood urea nitrogen, C4, and cold agglutinin titers were all within normal limits. The Coombs' test was negative; however, the test for ANA continued to be positive, with titers of between 1:160 and 1:320 as of five months after hospitalization. Since his discharge from the hospital, the patient continues to take phenytoin, digoxin, and nifedipine. He is currently asymptomatic and has no cutaneous or oral lesions.

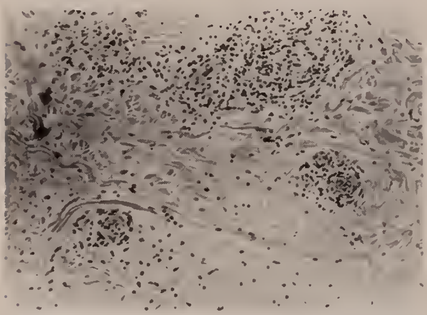

FIGURE 2. Low power photomicrograph showing multiple areas of perivascular infiltrates in the dermis.

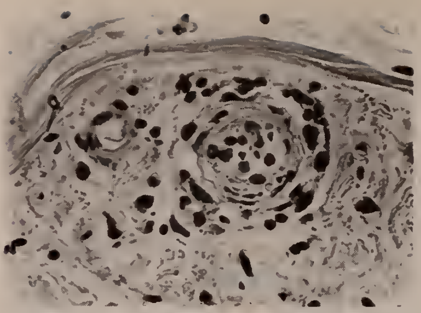

FIGURE 3. High power photomicrograph showing small vessel angiitis with prominent eosinophils (hematoxylin-eosin stain).

DISCUSSION

Furosemide-associated angiitis typically involves small blood vessels and falls within the general category of hypersensitivity vasculitis.[1] Discussions of vasculitis often describe overlaps between the various categories of vasculitis such as the polyangiitis overlap syndromes.[4] This report describes a patient who had both small vessel angiitis, evidence of acute glomerulonephritis, and some autoimmune features more characteristically seen with systemic lupus erythematosus (SLE), a disorder which can also manifest vasculitis. The clinical manifestations were temporally related to furosemide administration and did not recur in the absence of this drug.

The hematologic abnormalities in this patient consisted of cold and warm red blood cell antibodies as well as significant thrombocytopenia. Although the latter may be seen alone with furosemide hypersensitivity,[3] the former is definitely more consistent with SLE.[5,6] Also relatively specific for SLE are the positive ANA and the ulcerative stomatitis, both of which constitute criteria for the diagnosis of SLE.[7] Phenytoin, which the patient was taking, has occasionally been implicated in causing a positive ANA. However, many investigators[8] believe that the association between anticonvulsants and either positive ANA or drug-induced lupus is not significant. Studies of epileptics do not show an increase in positive ANA among those receiving anticonvulsant medication compared to those not receiving it.[9] The fact that the ANA in this patient was still positive after discontinuing furosemide therapy is still consistent with a drug-induced lupus syndrome because this test may continue to be positive for up to a year or more in cases of drug-induced lupus.[8] The incidence of a positive ANA increases with age, however, thus the specificity of this laboratory finding as related to a hypersensitivity reaction cannot be proven.

In conclusion, a patient with hypersensitivity vasculitis associated with furosemide also manifested significant autoimmune findings suggestive of a drug-induced lupus syndrome. Since furosemide is a commonly used medication, the possibility of this form of hypersensitivity syndrome should be considered by the prescribing physician.

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

The retirement of Dr Milton Helpern

TO THE EDITOR: I would like to clarify a statement made in the article by William Sturner, MD, in the March 1988 issue of the *Journal.*

Dr Milton Helpern was not pushed by the press and media to retire in 1974, at the age of 72. Mayor John V. Lindsay's administration changed the pension laws so that anyone who retired at the age of 65 would receive a better pension and could ask for five annual extensions. Doctor Helpern was 67 at the time the regulation was passed, and he chose this alternative.

By January 1974, when the Beame administration came in, Dr Helpern had taken advantage of the five extensions he was allowed, and could no longer stay on as chief medical examiner. Therefore, he retired.

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Shortage of preventive medicine physicians

TO THE EDITOR: The *Journal* should be commended for including two commentaries about preventive medicine in the January 1988 issue.[1,2] While I agree with the bulk of both articles, they overlook two fundamental reasons why preventive interventions have not been widely disseminated: the current lack of preventive medicine physicians, and the inadequate efforts to train a new cadre for the future.

While the numbers of practitioners in nearly all other medical specialties are in excess of or on par with current societal needs, preventive medicine, the physician specialty consisting of preventive medicine and public health, occupational medicine, and aerospace medicine, has had a shortage of personnel since at least the late 1970s. Shortages are predicted well into the 1990s.[3]

The Reagan administration's response to this shortage has been to cut federal funding for preventive medicine residency training even further. Federal support for these residencies has been eliminated by the administration for the past several years, although Congress has restored a small appropriation ($1.5 million) each time. Funding for the National Institute for Occupational Safety and Health (NIOSH) Educational Resource Center (ERC) program for training occupational physicians and nurses has also been drastically cut since 1980.

Other obstacles stand in the way of training preventive medicine physicians. Seven percent of preventive medicine residents receive no salary, according to a board member of the American College of Preventive Medicine.[4] This is unacceptable in general, but particularly so in an era when the average graduating medical student carries a debt load of $30,000.[5] Although 78% of medical schools have departments of community or preventive medicine,[6] few schools actively encourage students to enter preventive medicine, and few students perceive it as a career option. This must be rectified, and the teachers in these departments and the medical schools need to shoulder this responsibility.

The acquired immunodeficiency syndrome (AIDS) and other preventable chronic diseases such as cardiovascular disease and lung cancer will not wait for preventive medicine physicians to be trained. Unfortunately, the national need to transfer preventive knowledge into practice is not being supported by medical schools and the federal government. Funding and leadership are required of both groups in the effort to deal with this vexing problem.

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Generic drugs—A question of safety

TO THE EDITOR: The element of safety has been ignored by all sides to the debate in the increasingly partisan, and occasionally passionate, discussion of the issue of generic vs brand name drugs.[1-3] We refer to the need of the patient to be certain that a medication dispensed by the pharmacist is the one prescribed by the physician. The availability of the *Physicians' Desk Reference*[4] (PDR) to doctors in their offices and on hospital wards makes the identification of brand name medications an easy matter. Not so for generics. Although generic drugs are marked with code numbers, the physician has no means of identifying the product without actually calling the pharmacist or the manufacturer. Furthermore, the constancy of the color, shape, and insignia of the brand-name drug is a source of reassurance that a patient on chronic therapy is indeed receiving the prescribed medication all the time. This reassurance does not exist with generic prescriptions. The pharmacist may substitute one preparation for another because of price considerations, and neither the patient nor the physician can readily identify the dispensed drug and ascertain that no error has been made in the pharmacy.

A case in point is that of a patient at the Montefiore Medical Group who was diagnosed as having subclinical hypothyroidism and who had been taking two 0.025 mg tablets of brand name levothyroxine daily. On this regimen, her blood thyroxine level was 8.0 μg/dL. At her request, a renewal prescription was written for a generic equivalent. On follow-up examination, the patient appeared euthyroid but the blood thyroxine level fell to 2.1 μg/dL, a level that was confirmed twice. The patient, known to be reliable, stated that she had taken the two tablets daily as prescribed. At our request, she produced the medication. The manufacturer's name was not listed in the PDR. After spending a considerable amount of time and effort, we were able to locate and contact the manufacturer and determine that the drug dispensed was 0.025 mg triiodothyronine!

The generic-drug-related problem in this case differs from that reported by Fish et al.[5] In the latter, the clinical difficulty was created by incorrect formulation of generic levothyroxine. In our case, the confusion arose as a result of a dispensing error in which generic triiodothyronine was substituted for levothyroxine.

We believe that until the problem of identification of generic drugs is solved, generics cannot safely replace brand name drugs.

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Dispensing drugs for profit

TO THE EDITOR: I read with great interest the commentary, "Physicians and the dispensing of drugs for profit: A need for responsible legislation,"[1] by Leo Uzych. I dispense drugs for profit. The principal reason I got into it was to make money. This is also the principal reason I get up and go to work every morning. Maybe I am not alone in that. Yes, I want to help people, and I have helped many. No, I try not to put monetary concerns above the needs of my patients—more so than most physicians, I believe. But I do have to make a living, and that has become increasingly difficult.

In 1973, I did $60,000 worth of business, worked 50 hours per week, and was salaried at $30,000. In 1987, I did $220,000 worth of business, worked 70 hours per week, and was salaried at $60,000. In dollars adjusted for inflation, I am working harder and making less. Something terrible is happening to my business. It is called escalating overhead, and it is not under my control. Neither are most of my fees.

Mr Uzych is concerned that someone is ripping off the public.[1] I am, too. But are you looking in the right place, Mr Uzych? I sell a ten-day course of amoxicillin (250 mg) for $8 (drugstores for $16); amoxicillin/clavulanate (125 mg) for $16 (drugstores for $31); and doxycycline for $7 (drugstores for $21).

I have been studying medicine for 23 years. I read a lot, and I know a lot about drugs. I'm sorry, but I don't believe most pharmacists know more than I do about amoxicillin and the other common drugs which I dispense. I don't believe they do a better job of counseling, keep better records, make fewer errors, are more ethical, offer more availability, or any of the other assertions in Mr Uzych's commentary.[1]

The real concern about the resurgence of physician drug dispensing is coming from the pharmacy industry, whose members quite naturally feel threatened by it. I can understand this. I have no desire to injure my brothers in an allied profession, and I don't think those few drugs I sell hurt them significantly. But it helps me a little, and my patients love it. What right does the profession or the state have to limit me from a legitimate activity in which I am trained, licensed, competent, and fairly priced?

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Compassion

TO THE EDITOR: What is wrong with the medical profession? Great technological advances have been made. But the most important ingredient of a good doctor is being lost, what we used to call heart. What excuse is there for refusal to prescribe hypodermic morphine for a patient with a terminal illness who is suffering agonizing pain?

The patient I have in mind is a former patient of mine and a dear friend who was heartlessly told that she had an incurable cancer and was sent home to die. She was told that she had only a few weeks to live. She was given a handful of oral medications that were obviously inadequate to control her pain.

I am retired due to age and impaired vision, so I could not help her. I asked her if she wanted me to intervene and write to her doctor. But with the scarcity of doctors here, she was afraid that she could not get another doctor if I antagonized her physician. So I bit my tongue.

Her sister told me that the Society for the Prevention of Cruelty to Animals (S.P.C.A.) would not have allowed a dog to suffer so. She was put through hell on earth. Finally, the district nurse saw that she was dying and in horrible pain. The nurse insisted on hospitalizing her. She was immediately put on morphine and for the first time in months was made comfortable. She was then able to lie down and her suffering was over. She died the next day. The family's pleas for stronger medication had been ignored before that.

I know that morphine has side effects and has to be given cautiously. But, in 50 years of practice, I have used it repeatedly to alleviate the suffering of terminally ill cancer patients. This patient had been told that she had a very short time to live. The public was not allowed to view her after death because "she looked so terrible."

This letter is a plea for the return of that old-fashioned ingredient of all good therapy, compassion. Let's not send the patient home in agony with a couple of aspirin tablets and instructions to return in three weeks. The golden rule, though outmoded today, is still good, even in the practice of medicine. Forget lawsuits. Remember the patient.

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Norwalk gastroenteritis

Background

While outbreaks of acute nonbacterial gastroenteritis have long been known to those in public health it was not until 1972 that a definitive role for a specific viral agent was finally established. The prototype Norwalk virus was associated with an outbreak of presumed viral gastroenteritis affecting students and teachers in a school in Norwalk, Ohio, in 1968. Stool samples obtained from some of the sick children were stored for future laboratory studies after initial testing was negative for bacterial pathogens. In 1972, investigators reexamined the stored specimens by immune electron microscopy (IEM), and observed the putative agent which was appropriately named Norwalk virus. Follow-up studies involving oral challenge of human volunteers reproduced the disease and confirmed Norwalk as the causal agent.[1]

In recent years, a number of similar (Norwalk-like) agents have been described including Hawaii, Snow Mountain, Cockle, Marin County, Paramatta, MC and W.[2] These agents are morphologically similar but antigenically distinct. Serologic cross reactions have been documented among some of the agents. Eventually some of these antigens may be classified into other groups of viruses such as astrovirus, calicivirus and coronavirus.[3] Based on the similarity of clinical illness produced by these various agents, the term Norwalk-like virus has become popular in describing such outbreaks of viral gastroenteritis.

Epidemiology

A review of national data by the US Centers for Disease Control (CDC) in 1982 revealed that outbreaks of Norwalk-like gastroenteritis occurred in all age groups (except infants) and were reported during all four seasons of the year. Outbreaks were epidemiologically associated with a number of different vehicles including food, drinking water and infected people. Settings have included schools, summer camps, restaurants, hotels, hospitals, nursing homes and cruise ships.[4] Communitywide outbreaks have also been described. Epidemiologic investigation has shown both common source and propagated outbreaks. Cases occur over several days to several weeks with secondary person-to-person attack rates as high as 32%. Seroprevalence rates (immunity levels) are lowest in younger age groups and rise to approximately 60% of adults by 50 years of age.[3] Circulating antibodies against Norwalk do not always correlate with immunity from disease. Immunity appears transitory as symptomatic reinfection has been demonstrated in volunteers who had been previously exposed to the same antigen several years earlier.[2]

In 1986, 25 of 85 (29%) of the reported outbreaks of foodborne illness in New York State were described as Norwalk-like. Ten of the 25 were serologically confirmed as Norwalk virus. CDC data indicated that 31 of 74 (42%) outbreaks of nonbacterial gastroenteritis reported from multiple states between 1976 and 1980 were attributed to Norwalk virus.

High attack rates in groups following common source exposures, secondary transmission to contacts and communitywide propagated outbreaks suggest that Norwalk-like viruses are highly infectious. Transmission can occur by person-to-person contact, through food or water and possibly by fomites.[5]

Clinical Aspects

Sumary data obtained by CDC from 38 confirmed Norwalk outbreaks describe a sudden onset of acute gastrointestinal illness characterized by vomiting (25–100% of patients), diarrhea (21–100%), cramps (17–90%), headache (17–80%), chills (5–74%) and fever (13–71%).[4] Several investigations have reported vomiting more often in children and diarrhea occurring more often in adults.[6] Subclinical infection has been documented.

The incubation period has ranged from five to 72 hours but is usually 24–48 hours. The duration of symptoms is typically one to two days and rarely requires hospitalization.

The period of communicability is during the acute illness, but in one investigation, epidemiologic data suggested that a food handler was communicable the day before onset. In addition, electron microscopy studies have identified Norwalk-like virus up to 72 hours after onset of symptoms.[7]

Laboratory Diagnosis

Serologic and antigen detection methods for Norwalk-like virus are improving but have limitations in terms of specificity and sensitivity.[8] Norwalk virus and its related agents have yet to be grown in tissue culture and require complex immunoassays and electron microscopy. Most local laboratories are not equipped to perform such examinations.

Stool specimens obtained from people during outbreaks of Norwalk-like illness are negative for enteric bacteria, parasites, culturable viruses and rotavirus. Stools are described as nonbloody and lack leukocytes generally seen in bacterial enteritis. When examined by IEM, 27 nm particles are often observed. Examination of acute and convalescent sera generally shows a four-fold rise in titer in 50% or more of the cases. An IgM serology specific for Norwalk virus has been used to establish the diagnosis in a number of outbreaks.

As testing for Norwalk-like viruses is not available in most laboratories and cannot be justified in many outbreaks, a practical approach is to establish the diagnosis by exclusion. After ruling out enteric bacteria and rotavirus, an outbreak of presumed viral gastroenteritis could be classified as nonbacterial or Norwalk-like. The search for specific Norwalk-like agents should be reserved for outbreaks of particular epidemiologic interest. Arrangements for laboratory testing can be made through the New York State Department of Health.

Control Measures

There are no specific therapies or vaccines for Norwalk-like viruses. During suspect or confirmed outbreaks, control measures should focus on careful handwashing, exclusion from work when ill and use of plastic disposable gloves when handling food. In institutional outbreaks, cohorting of patients and staff, limiting visitors and use of plastic disposable gloves by health care staff during certain types of patient contact should be considered. Finally, the persistence of raw clam associated outbreaks of Norwalk gastroenteritis underscores the significant risk of consuming raw clams.[9] Consumers should be wary of consuming raw clams until more effective control measures can assure the safety of eating shellfish.

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Behavioral risk factor surveillance system

Nine of the ten leading causes of premature mortality in the United States are linked to one or more of six behaviors: cigarette smoking, misuse of alcohol, lack of exercise, not wearing seatbelts, overeating and failure to control high blood pressure.

Until recently, a systematic means for collecting population-based prevalence data on behavioral risks in New York State was lacking. Furthermore, there may be wide variations in the actual prevalence rates from one county or locality to another. Surveillance of behavioral trends over time is valuable in planning and evaluating intervention strategies.

In the early 1980s, the Department of Health received categorical funding from the Centers for Disease Control (CDC) to implement a behavioral risk factor telephone survey. The main purpose of this survey was to create a database for the incidence and prevalence of major behavioral risk factors associated with the leading causes of premature deaths. Conducted by the Institute for Social Research, University of Michigan, Ann Arbor, this survey was first administered in 1983 to approximately 1,000 randomly selected New York State residents. Questions focused on cigarette smoking history, alcohol use, drinking and driving, seatbelt use, level of physical activity, stress, hypertension, diabetes, height and weight.

Based on the success of the 1983 survey, the department joined 20 other states and the District of Columbia in the CDC's ongoing Behavioral Risk Factor Surveillance System (BRFSS). The survey was repeated in 1985, with the addition of questions on table salt use, attempts to quit smoking in the last year, the amount of alcohol consumed in the last month and demographics on annual household income, employment status and marital status. Since 1985, the survey has continued each month; the monthly samples are then aggregated to provide annual estimates of risk factor prevalence rates.

Recently, a statistical monograph summarizing the prevalence data from the 1983 and 1985 surveys was produced and distributed. A brief summary of the major findings and recommendations follows.

Seatbelt Use

The proportion of respondents who said they used seatbelts most of the time increased from 26.2% in 1983 to 82.4% in 1985, following the enactment of a state law making the use of seatbelts mandatory after January 1, 1985. Data from this and other surveys document the impact seatbelt laws have on their use.

Hypertension

Of those who reported having high blood pressure (about 25% of respondents), the proportion of uncontrolled cases dropped from 22% in 1983 to 5% in 1985. Recommended public health interventions to reduce the prevalence of uncontrolled hypertension include targeted screening of high risk populations, establishment of tracking systems to improve appointment retention and patient compliance, and public and professional education.

Obesity

Over 20% of respondents were judged to be overweight, based on self-reported height and weight. A slight decline in the proportion of obese respondents was noted from 1983 to 1985, although the proportion of respondents trying to lose weight increased from 27% in 1983 to 43% in 1985. Most reported using calorie restriction while only half used exercise as a weight reduction method. Because of the increased risk of obesity in hypertensive and diabetic patients, intervention programs that include nutrition education and exercise should be targeted to these groups.

Alcohol

The percentage of those who reported drinking at least once in the month prior to the survey decreased slightly from 66.9% in 1983 to 66.0% in 1985. However, the proportion of those who drink at problematic levels has grown.

Among those who drank at least once, males were twice as likely as females to report acute drinking (five or more drinks on at least one occasion in the past month), were four times as likely to be chronic drinkers (an average of two or more drinks a day) and were three to four times as likely to drive a car after drinking. Young drinkers, aged

18–24, reported the highest prevalence of acute drinking, with males at 59.2% in 1983 and 61% in 1985, and females at 29.3% in 1983 and 40.4% in 1985. Comprehensive strategies are needed to prevent alcohol abuse, including restricting the sale of alcoholic beverages, educating the public (particularly children and adolescents) and strict enforcement of drunk driving laws.

Cigarette Smoking

From 1983 to 1985, the percentage of smokers in the state increased from 30.4 to 31.4. During this period, smoking among males decreased while smoking among women increased, bringing the number of women smokers to nearly equal that of men. High or rapidly increasing rates of smoking-related diseases identify blacks, Hispanics, women and heavy smokers as special targets for smoking cessation programs. Intervention programs should be designed to help current smokers in their efforts to quit and to educate people, especially children and adolescents, not to smoke. Physicians, dentists and other health professionals are encouraged to fully utilize opportunities to counsel and influence their patients to stop smoking.

Physical Activity and Exercise

In the 1983 survey, 57% of respondents reported participation in some kind of physical exercise. By 1985, the percentage increased to 75%. Recommendations have been made to include physical activity and exercise as part of a comprehensive program of multiple risk factor intervention. Priority target populations include children, the elderly and people of lower socioeconomic status.

Diabetes

Over 5% of respondents have been diagnosed with diabetes. Intervention strategies to reduce the impact of diabetes include reducing excess weight or maintaining normal weight, controlling hypertension, exercising to improve circulation, educating patients on diabetes management, eliminating smoking, improving eye and foot care, and providing better prenatal care to avoid gestational diabetes.

Stress

Eighty percent of 1983 survey respondents reported experiencing stressful conditions in the past year. (There were no stress questions on the 1985 survey.) Inability to do daily activities because of worry or nervousness was reported to occur often (4.8%), sometimes (12.5%), rarely (25.3%) or never (57.4%). People often responded to stress in unhealthy ways by smoking, overeating or drinking alcohol. Public health interventions might consist of efforts to identify people who have difficulty coping with stressful life events and teaching them stress management skills or referring them to established programs.

Multiple Risk

Data were analyzed to determine how many people reported more than one of the following risk factors: nonuse of seatbelts, uncontrolled hypertension, obesity, cigarette smoking and acute drinking. Depending on which factors are present in combination, the resultant risk may be synergistically increased. The percent of the sample reporting none of these major risk factors nearly doubled from 1983 to 1985 (24% to 45%). There was no appreciable change in the proportion of each sample exhibiting just one risk factor (40%). People in the age range 45–64 years (the group most likely to report at least one risk factor) exhibited the most dramatic decrease in the reporting of one or more risk factors; those reporting more than two risk factors declined from 28% in 1983 to less than 1% in 1985.

The BRFSS is an important tool for health care planners and program staff at the state and local levels. It represents a source of data that can be used to estimate baseline rates for establishing program priorities and targeting services. For example, BRFSS data showing an increase in female smoking have prompted the Department of Health to develop initiatives to prevent young women from starting to smoke and to help smokers quit. The data were also used as supporting evidence in the Public Health Council's efforts to promulgate Part 25 of New York's Sanitary Code which would have restricted smoking in public places.

The BRFSS has also played an important role in the development of the department's cancer prevention five-year plan on smoking control. Further, the BRFSS will be used as a monitoring tool to evaluate progress toward the 1990 objectives of the nation in the areas of smoking, alcohol misuse, obesity and physical fitness.

One limitation of the BRFSS data is that the current sample size is not large enough to identify geographic variations of risk behavior within the state. There are plans to increase the size of the BRFSS sample to provide better estimates of risk factor prevalence rates in population subgroups and in different geographic areas to aid in the identification of high risk populations. Additional questions on preventive practices (physician checkups, cholesterol screening, breast cancer screening and influenza immunization) have been included in the survey.

For a copy of the statistical monograph or more information about the BRFSS, contact Christopher Maylahn at (518) 474-0512 or Helen Bzduch at (518) 474-1222, Bureau of Adult and Gerontological Health, Division of Family Health.

HIV antibody seroprevalence among military recruits

Since October 1985, the United States armed forces have been testing all prospective recruits for the human immunodeficiency virus (HIV) antibody. Those identified to be HIV antibody positive are advised of their status, counseled and denied entry into the military.

Through September 1987, approximately 73,000 New Yorkers, among more than 1.2 million prospective recruits nationwide, have been tested. Table I summarizes the experience in New York State and compares it with the total US experience.

The rate of positivity in New York State, exclusive of New York City, is comparable to the national rate. Within New York City, the percent testing positive is predictably higher. The positivity rate ratio between males and females has been the focus of considerable discussion. Statewide, the male-female ratio of recruits testing posi-

TABLE I. Military Recruits, HIV Antibody Seroprevalence by Sex, Race and Age, New York State, New York City and US, October 1985–September 1987

	New York City	Rest of State	Total NYS	Total USA
Total				
Recruits tested	17,910	55,651	73,561	1,254,068
# Positive	188	90	278	1,822
% Positive	1.05	0.16	0.38	0.15
Sex				
Males tested	15,109	47,702	62,811	1,081,054
% Positive	1.12	0.17	0.40	0.16
Females tested	2,801	7,949	10,750	173,014
% Positive	0.68	0.14	0.28	0.07
Race/Ethnicity				
Whites tested	4,078	43,082	47,160	921,030
% Positive	0.71	0.08	0.14	0.08
Blacks tested	8,851	9,457	18,308	234,452
% Positive	1.23	0.50	0.85	0.39
Hispanics tested	3,089	1,248	4,337	55,744
% Positive	0.81	0.08	0.60	0.22
Others tested[1]	1,892	1,864	3,756	42,842
% Positive	1.32	0.43	0.88	0.19
Age				
17–19—% Positive	0.22	0.04	0.08	0.04
20–24—% Positive	1.10	0.20	0.46	0.20
25–29—% Positive	2.80	0.42	1.20	0.41
30–44—% Positive	2.40	0.67	1.20	0.36
45+ —% Positive	1.60	0.46	0.71	0.14

[1] Others category includes Asian and Native Americans, plus those for whom race/ethnicity is unknown or unrecorded.

Source: US Centers for Disease Control.

tive is approximately 4:3, considerably different from the overall male-female ratio of adult/adolescent AIDS cases, approximately 8:1. The hypothesis has been offered that the military recruit seropositivity ratio suggests that heterosexual transmission has emerged as an important mode of HIV infection, especially in view of the presumption that military recruitment discourages voluntary enlistment by homosexual males and intravenous drug users.[1]

Risk group status is not systematically gathered on HIV antibody positive recruits. However, the New York City Department of Health has reported that among 23 HIV antibody positive military recruits who called the city's AIDS hotline following their notification by the military of their antibody status, 19 (83%) acknowledged risk behavior, primarily bisexual activity or intravenous drug use, consistent with known AIDS risk factors.[2] Also, the number of HIV antibody positive female recruits statewide has totaled only 30, a small number on which to base interpretations in the absence of specific case information.

At the request of the State Health Department, the federal government has recently provided a computer tape listing of the zip code distribution of all state recruits, without identifiers, who have been tested for HIV antibody. Department staff will analyze that tape and compare the zip coded rates of military recruit positivity to such indices as the zip code distribution of AIDS cases, the distribution of drug-related hospital discharges and the distribution of HIV antibody positive women giving birth. The results of the analysis will be reported in a future issue of *Epidemiology Notes*.

The relative stability in the rate of positivity over time (October 1985–September 1987) among military recruits in New York State is suggestive of the absence of an "explosive" spread of HIV into the population that elects to enlist in the military. It is unknown, however, to what extent this stability is affected by the self-deferral of potential recruits who are at increased risk for HIV infection who wish to avoid HIV antibody testing.

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Farming injuries

In the last 50 years, farm injuries have increased, while most other injury categories have decreased. In 1985, there were 900 farm work-related deaths and 90,000 disabling injuries nationwide. Once every three minutes, 540 times a day, a fatal or disabling injury occurs on a farm. The death rate in 1985 per 100,000 farm residents was 16.8 for farm work-related incidents. Fatal and nonfatal farm injuries cost nearly $5 billion annually.[1] In 1986, agriculture became the most hazardous occupation in the US.

Children

In the United States, approximately 300 children die and more than 24,000 sustain serious farm injury each year. Tractors, farm machinery and grain entrapment (storage) cause most of the injuries and over 80% of the fatalities in children. Over 2.1 million children are directly exposed to agricultural hazards by living and working on farms. Many children as well as other farm laborers are exposed to other significant dangers that include respiratory disease from air pollution, grain dust, fungi and animals; entrapment and suffocation; drowning; electrocution; and kicks, bites and butting by farm animals.[2]

For New York State, mortality data gathered by the Agricultural Engineering Department at Cornell University showed that in 1985 and 1986 combined, there was an average of 34 deaths per year, of which 18 per year (53%) were tractor related and seven per year (21%) were related to use of other farm machinery.[3]

For 1985 and 1986 combined, there were seven deaths in the 14 and under age group; nine for 15 to 24 year olds; 17 for 25 to 44 year olds; 20 for 45 to 64 year olds; and 14 for those 65 years and older. The age of one fatality was unknown.

A 1983 survey conducted by Cornell University monitored 504 farms and reported accidents and injuries that occurred among 2,027 farm family members and employees. One result of the survey showed that children aged five to 14 had a disproportionate injury rate of 35.56 per 100 full-time equivalent workers compared to 15 to 24 year olds (13.45), 25 to 44 year olds (10.33), 45 to 64 year olds (6.55) and 65+ (10.17). This accident rate was adjusted for the time a worker was exposed to the potential for injury versus age group.

Agriculture is the only industry that can legally use paid child labor starting with 12 year olds. It is illegal for 12 and 13 year olds to use machinery. At age 14 and 15, they can operate tractors but have to complete a training course and be certified by 4-H or Future Farmers of America. Farm families generally do not send their own children for tractor operation training but provide on-the-job training themselves.[3]

Farm safety has, to date, not attracted attention or funding at the federal level to the extent of other occupational safety programs. The federal government spends $472.4 million annually on all occupational safety programs. Approximately $189 million are directed to the 800,000 worker mining industry, $236.25 per miner, while in contrast, only $1 million, or 31¢ per farmer, is directed to the 3.2 million worker agriculture industry. Greater attention to describing the nature and causes of farming injuries and to developing preventive strategies is warranted.

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The Boat House in Prospect Park, Brooklyn, NY (Jeffrey Birnbaum, MD).

OXFORD TEXTBOOK OF MEDICINE

Second Edition. Edited by D.J. Weatherall, J.G.G. Ledingham, and D.A. Warrell. 2 volumes, illustrated. Oxford, England, Oxford University Press, 1987. $135.00 (clothbound)

INTERNAL MEDICINE

Second Edition. Edited by Jay H. Stein, MD. 2,300 pp, illustrated. Boston, Little, Brown & Co, 1987. $85.00 (clothbound)

There are several widely used textbooks of medicine in the United States and so one might rightly ask if there is a need for more. Most would be inclined to answer no, that is if they had not seen the two books under review here. *The Oxford Textbook of Medicine* is the lineal heir of Frederick Price's standard work, *Textbook of the Practice of Medicine*, which was widely used in Great Britain and the Commonwealth for almost six decades. The first edition of the new text was published in 1982 and was well received. Although intended to serve many of the same purposes as its American counterparts, it differs from them in important ways. While it is a reference text reflecting current British practice, it also includes the views of an international group of authors. This makes it a superb reference for many of the problems of clinical practice in the third world. Users worldwide will find much more in these two volumes that they can relate to than in comparable American texts.

While providing the usual introductory chapters in the sciences basic to general medicine, it also has some unique features. There is a stimulating opening section called "On Textbooks of Medicine" by A.M. Cook of Oxford followed by one called "On Modern Scientific Medicine." The latter contains chapters on medical ethics and on scientific medicine. A third section follows that includes four chapters, "Medicine in an Unjust World," "Health and Sickness in the Community," "Primary Health Care," and "Primary Health Care in a Developing Country." The first dozen sections, each divided into a variable number of chapters, are contained in the first volume, and the remaining 16 in the second. Each entry is well referenced.

There is much emphasis on the art of medicine in this text which gives it a dimension apart from most of its fellows. It is extremely comprehensive, authoritative, up to date, and easy to use and read. It is highly recommended to all who have an interest in general internal medicine.

Internal Medicine, edited by Jay H. Stein of the University of Texas Health Science Center, San Antonio, is also in its second edition. The first edition of this text was published in 1983. Like the first edition, the second is organized according to organs and organ systems and emphasizes the underlying pathophysiology of disease, especially as it relates to diagnosis and treatment. This approach is somewhat unique and places this text in a class unto itself.

The book is divided into ten broad sections which in turn are subdivided into 361 chapters written by a very large number of distinguished contributors from around the United States. There are also a few contributors from overseas. Although this text covers much the same ground as others in the field, its basic approach to covering that ground is different. Practicing internists will find this method of analyzing illness both stimulating and useful.

PASCAL JAMES IMPERATO, MD
Editor

PEDIATRIC TRAUMA CARE

Edited by Martin R. Eichelberger, MD, and Geraldine L. Pratsch, RN. 219 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1988. $33.00 (hardcover)

This is a timely book about a subject which regrettably involves many American families. The authors and their group write from the perspective of a growing experience at the Trauma Service of the Children's Hospital, National Medical Center, in Washington, DC. The book primarily puts in written form what has been known by many who treat trauma. First, good initial care may not only save a life, but also shorten the hospital stay and prevent substantial morbidity. Second, the full extent of the injury must be initially determined so that it can be properly treated without causing inadvertent additional injury. Finally, early rehabilitation is crucial in the management of trauma.

The book is of high quality, easily readable, and has good charts and excellent pictures. The writing style, even though there are many authors, is excellent. The facts as listed appear accurate and the charts, flow sheets, and forms are easily understood. The book is written on a "modular" basis and is organized with experts in the differing fields offering short chapters on their areas of expertise. The approaches appear, in each of the modules, to be among the standard methods of managing the particular problems encountered. The function of the book is to inform about trauma, and to codify what have been in the authors' hands successful approaches to the management of this difficult problem.

However, it has frequently been said that a camel is a horse designed by a committee. This book, like many on the subject of trauma, addresses the issues on a purely objective basis founded on logic. Injury, in fact, is illogical, unexpected, unpredictable, and, most important of all, an unknown. Because it is unplanned, trauma places the greatest burden on the health care system. This book is organized to cover the issues. It does not deal with the judgmental elements of trauma care. For example, the most important part of trauma management is to "triage down" from the most experienced to the least experienced. The reason for this is that judgment plays a critical role in setting priorities (ie, life, limb, function, and cosmetics), and how one makes these assessments is of crucial importance. Some injuries can be initially ignored and others, when treated, assume a much lower priority as the patient's care evolves.

This book deals with answers. It would have been interesting to deal with questions. How do you assign priorities? What steps can be skipped? Who makes the final decision when conflicting specialists differ? Does the "surgical coordinator" assume responsibility for the patient during his or her stay in the hospital? How can you conduct a trauma service at the level advocated and not require senior attending surgeons to be responsible for this critical initial evaluation and care?

The essence of trauma care is predicated upon the belief that a forcible confrontation between the individual and the environment will result in injury; that depending on the forces involved, the extent of injury can be predicted; and that the body responds to injury in a predictable manner. Therefore, our job as physicians involved in trauma care is to understand the effects of these injuring forces, help the body with the healing process by giving appropriate replacement substances, removing the devitalized tissues, appropriately draining the affected areas, and thereby helping the body to heal. More importantly, those who treat trauma know and depend upon the human organism's remarkable capacity to heal. This is where a well trained trauma surgeon can make a difference. Because trauma is the most unpredictable event in medical practice, it requires that those who manage its consequences be the most disciplined: to know what to do, to do it, to do it right, and to do it right the first time.

Trauma crosses many disciplines. This book approaches trauma from the perspective of modules. It would have useful if some space were devoted to the common principles which cross all modules—in essence giving the readers a little more help with how judgments are made.

Finally, while the authors stress that rehabilitation is important, the rehabilitation chapters are, as in all books, at the end. I would expect from their knowledge of the subject that they would raise the point that rehabilitation begins with the injury and that the only satisfactory criterion for successful trauma care must be that of an individual returned to function within family and society.

It is unfortunate that at a time when so much can be accomplished in trauma care, there is still so much more that needs to be done. For anyone involved with trauma care or considering entering the field, this book is an excellent place to begin.

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THE PARENTS' EMERGENCY MEDICAL GUIDE

By Robert Kaplan, MD. 274 pp, illustrated. New York, McGraw-Hill Book Co, 1987. $9.95 (paperback)

This book has a somewhat misleading title. Designed with parents and other caregivers in mind, it is ambitious and covers more material than the title implies. The book actually is a general medical guide for nonprofessionals, written in a clear, understandable, noncondescending style. The author, a pediatrician at Children's Hospital in Buffalo, attempts not only to educate but also to make caregivers more responsible in evaluating the need for children's medical care, rather than making a Pavlovian dash for the telephone at the first sign of a medical problem. He accomplishes this through a series of "maps," decision trees which guide the parents through scores of emergent, urgent, and nonurgent conditions. The three outcomes are: get help fast; get help; or don't worry it will go away. Understanding and following these maps involves a degree of intelligence and confidence in one's decision-making ability, so the tool may intimidate parents at first glance. However, if they can become comfortable with the maps, the book will guide them through many difficult situations, from breathing problems to diarrhea and nosebleeds.

About a third of the book is devoted to true emergencies and begins with a series of basic comments with guidelines to follow when confronted with an emergent condition. These are followed by an excellent narrative which gives the parent insight concerning the chaotic-appearing, busy emergency department and provides practical advice on how to handle a child's hospitalization. Individual chapters concerning 15 different emergency conditions, all with decision maps, are variably effective. Especially noteworthy are the chapters on breathing problems, seizures, drowning, and eye injuries. A major disappointment is the section on poisoning, which was disproportionately brief and superficial.

Approximately half of the work is devoted to nonemergent medical conditions, and features extensive chapters on bites and stings, child abuse, and rashes. Additional material included in this section explains the basics of child care, including the care of newborns, their growth and development, the use of medicines, and the method of feeding babies. The third brief section emphasizes physical fitness and accident prevention. Although written in a style that will appeal to many parents because of its simplicity, the organization of the individual sections is chaotic and extremely difficult to follow. The author insists on listing the chapter headings in alphabetical order when there appears no good reason to do so, producing a confusing progression of ideas and conditions. Furthermore, the interpositioning of informational chapters such as those on growth and development and feeding with those on illnesses such as earaches and sore throats creates an image of a work whose scope was not well planned and whose organization is faulty. These defects also transform a concise handbook of action for medical conditions into a work whose effectiveness suffers. Despite this overreaching, however, the author has attained his objective of providing a quick reference to help parents make a decision in times of medical need.

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USEFUL PROCEDURES IN MEDICAL PRACTICE

Edited by Paul W. Roberts, MD. 610 pp, illustrated. Philadelphia, Lea & Febiger, 1986. $34.50 (softcover)

This is a book one wishes one had had as a resident. It describes every procedure residents need to do in the middle of the night, and more. With the wealth of medical information that inundates us all, it is remarkable how few books actually tell us how to do something. And the practice of medicine is nothing if not pragmatic. Dr Roberts, an associate professor of family and community medicine at the University of Toronto, is to be complimented for compiling such a valuable and unpretentious resource. He has ably organized the contributions of two dozen specialists from the medical faculty of his university into a clear, concise, and highly practical manual. This work is not, nor is it intended to be, a resource for surgeons or subspecialists. Its aim is to describe common office and hospital procedures that are often required of physicians providing primary care. As such it can be used to upgrade or expand one's repertoire. Harried housestaff and medical students who wish for more than to "see one, do one, teach one" and who want to learn psychomotor skills will find it an excellent handbook.

A broad range of emergency and elective procedures is described. These vary greatly in complexity. Some of them are quite basic and one may question the need for their inclusion. Among these are simple venipuncture, for example, or how to take electrocardiograms. This illustrates the completeness of the book and the fact that the authors take nothing for granted. To give examples at the other end of the spectrum, standard techniques in bone marrow biopsy and aspiration, splinting, chest tube drainage, dermatologic surgery, and even exploratory burr holes are described. There is an excellent chapter on local and regional anesthesia. Some useful skills outside of the usual scope of such manuals are also included, such as those dealing with common dental emergencies and techniques of physiotherapy. The work is amply illustrated, and good use is made of figures to clearly define anatomy. Indications, equipment, method, and precautions are listed for each procedure.

While most physicians' practices do not encompass the wide range of procedures described in this book, it is a handy, practical reference for family physicians and other generalists. Those who practice in rural or remote areas without the luxury of easy referral may find it most useful, but there is something here for everyone. If I ever did a residency again, I would make a point of carrying it with me.

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DeGOWIN AND DeGOWIN'S BEDSIDE DIAGNOSTIC EXAMINATION

Fifth Edition. By Richard L. DeGowin, MD. 1,053 pp, illustrated. New York, Macmillan Publishing Co, 1987. $29.95 (hardcover)

The fifth edition of DeGowin and DeGowin's *Bedside Diagnostic Examination* reaffirms its acceptance as a standard textbook for medical students and physicians. The book is dedicated to office and bedside diagnostic examination. The success of this text in an age of high technologic imperatives underlines the important value of patient rapport and touch. The authors condense in an organized format of 1,053 pages the known diagnostic maneuvers for a wide range of signs. This edition represents an increase of only 14 pages since the fourth edition in 1981.

Richard L. DeGowin, Professor of Internal Medicine, University of Iowa College of Medicine, is the author, as he has been since the first edition in 1965. His father, Elmer L. DeGowin, MD, MACP, was the founding and senior author of the text until his death in 1981. The fifth edition witnesses for the first time two new contributors: Peter R. Jochimsen, MD, Professor of Surgery, and Ernest O. Theilen, MD, Professor of Internal Medicine, both of the University of Iowa College of Medicine.

New additions to this edition include a discussion of the computer in diagnostic reasoning; presentation of new syndromes such as Lyme disease, toxic shock, Turcot's and AIDS; use of the Système International units for laboratory values, and enhanced laboratory studies like iron, ferritin, blood films, and urinalysis. Certain presentations in the fourth edition have been reduced or deleted (ie, diagnostic imaging, ultrasound).

The text measures 4½" by 7½" and weighs 1 lb, 8 oz, thus allowing it to serve as an easy pocket or black bag reference. The cost of $29.95 also makes it accessible to student, house officer, and attending staff alike. Comparable texts include B. Bates' *A Guide to Physical Examination*, MacByrdes' *Signs and Symptoms*, and *Clinical Diagnosis* by Judge et al. All of these references are larger, heavier, and more costly, although each in its own right is more comprehensive by way of illustrations and factual material.

Nevertheless, the fifth edition of *Bedside Diagnostic Examination* has earned the right to stand in a time-honored place next to its four predecessors. Of its many merits, perhaps the most important is its clear message that medicine begins and ends at the patient's bedside.

RICHARD B. BIRRER, MD
Consulting Editor

HANDBOOK OF TOXICOLOGY

Edited by Thomas J. Haley, PhD, and William O. Berndt, PhD. 607 pp, illustrated. New York, Hemisphere Publishing Corp, 1987. $110.00 (hardcover)

When I was asked to review Haley and Berndt's *Handbook of Toxicology*, I imagined I would receive a short paperback covering a minimal amount of material. Instead, the "handbook" was a hardback text consisting of 697 pages divided into 16 major chapters and numerous subheadings by 21 different contributors. Topics range from basic pharmacokinetics of xenobiotics to mechanisms of metal-induced cell injury. The introductory chapter on the history of poisoning by Walter Decker was fascinating and is recommended reading for those interested in medical history.

In spite of its length, the text does remain a handbook in that in-depth reviews and discussions of toxicologic topics are not common. Rather short informative paragraphs concerning the toxicology of a multitude of agents dominate the text in a consistent style of writing. However, the presentations of normal physiology and anatomy in the organ system chapters are generally quite complete and are frequently lacking in other toxicology textbooks.

This book appears to have been written for the PhD research and/or academic toxicologist since most of the data presented is in reference to animal studies (eg, rat LD_{50}, rabbit eye irritant test, etc). The practicing physician will not find this text useful in the everyday practice of medicine.

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A POCKET MANUAL OF DIFFERENTIAL DIAGNOSIS

By Stephen N. Adler, MD, Mildred Lam, MD, and Alfred F. Connors, Jr, MD. 268 pp. Boston, Little, Brown & Co, 1982. $12.00 (paperback)

The task of reviewing *A Pocket Manual of Differential Diagnosis* is similar to that of evaluating the index of an internal medicine text. The listings are complete and the references fair. I carried this manual for three months and had the opportunity to use it twice. The manual, by virtue of its intent, is lacking in thought and in the provision of information regarding the interrelationships and clustering of symptoms/clinical/laboratory findings.

This book offers no advantages over Wallach's *Handbook of Differential Dx*. It is a fair reference text for medical students who are beginning their clinical rotations on the wards in medicine. I do not find this book to be a useful addition to the body of medical literature.

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THE AGE OF MIRACLES: MEDICINE AND SURGERY IN THE NINETEENTH CENTURY

By Guy Williams. 234 pp, illustrated. Chicago, Academy Chicago Publishers, 1987. $16.95 (hardcover)

Having always been interested in medical history, I began reading this little book full of bias and doubt. How, I thought, can this unknown author write an interesting monograph about a period so well described in the classical work by Arturo Castiglioni and the more recent book by Bordley and Harvey? When I finished the last page, I searched for more information about Guy Williams. *The Age of Miracles* is extremely well written and each chapter will hold your attention as well as any fine novel.

There are two chapters on John Hunter. Williams takes us in detail from his early life through the development of his interest in animal and human anatomy to his amazing accomplishments in teaching, research, and surgery. One anecdote about the "Irish Giant" is worthy of summarization here. In order to avoid the post mortem fate he expected, the "tall man" stipulated in his will that he be buried in a very deep spot in the ocean. Dr Hunter bribed the undertaker who stole the coffin with the corpse and it was promptly "dissected and skeletonized."

The chapters on such varied subjects as body snatchers, orthopaedics, anaesthetics, antiseptics, blood, watering places, the birth of tropical medicine, x-rays, and radium are excellent. Those of us who served in the South Pacific and learned the clinical aspects of tropical medicine from worn-out texts with pages partially destroyed by excessive heat and humidity will enjoy Guy Williams' account of Manson's life and the discovery of the causes of elephantiasis, yellow fever, and malaria. It will stimulate and reawaken the memory of experiences dulled by age and the absence from teaching.

All physicians and medical students should grasp any opportunity to read this informative, instructive, and fascinating account. The only criticism I have of this marvelous book is the size of the type. It's so small that I found it necessary, against my will, to reduce reading time.

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Observations on medical glove use

Due to mounting concern over the risk of transmission of acquired immunodeficiency syndrome (AIDS), health care workers have demonstrated an increasing reliance on medical gloves. Following the discovery by a health care worker of two consecutive defective gloves, Fairmount Hospital (San Leandro, Calif) conducted a quality assurance survey of the gloves they use (*N Engl J Med* 1988; 318:1333).

The study produced the following results: 10% of nonsterile vinyl gloves and 35% of nonsterile latex gloves exhibited defects when inflated with air and held under water. None of the sterile latex gloves proved defective.

In their letter, the authors stated that "The standards of the American Society for Testing and Materials (ASTM) for testing latex gloves, which are voluntary, call for a failure rate no higher than 1.5 percent for latex surgical gloves (Class II) and 2.5 percent for surgical-examination gloves (Class I). There is apparently no standard for vinyl gloves." The authors recommend review and revision of the ASTM standard to include vinyl gloves, testing of gloves by filling them with air and placing them under water, grading and labeling of gloves based on quality, and the development of guidelines for glove use. Sterile latex gloves should be the gloves of choice when handling blood products.

Saliva shown to inhibit HIV-1 infectivity

The National Institute of Dental Research (NIDR) recently released a special report (*JADA* 1988; 116:635-637) demonstrating that saliva can inhibit the ability of human immunodeficiency virus 1 (HIV-1)—the virus that causes AIDS—to infect human lymphocytes. Previous studies had shown that HIV-1 was rarely recovered from salivary secretions and that dental health care workers have a low occupational risk of acquiring HIV-1, suggesting a low risk of AIDS transmission via saliva.

In the NIDR report, authors Fox et al incubated HIV-1 with whole saliva and with secretions from the parotid and submandibular/sublingual glands. The saliva samples were obtained from healthy male volunteers not at high risk for AIDS. The saliva/HIV-1 mixtures were incubated for one hour at 37°C, as were a negative control consisting of only growth medium and a positive control of growth medium and HIV-1. Following incubation, the samples were passed through filters, diluted, and cultured with normal human peripheral blood lymphocytes. The presence of HIV-1 in these cultures was monitored by measuring reverse transcriptase activity.

Preliminary tests demonstrated that incubation with saliva under the experimental conditions is not toxic to human peripheral blood lymphocytes. Subsequently, the authors found that "whole saliva completely inhibited the ability of HIV-1 to infect human lymphocytes." They were able to detect no reverse transcriptase activity. Other tests revealed that submandibular saliva could also completely inhibit the infectivity of HIV-1, whereas parotid saliva produced inhibition in some samples and no inhibition in others. Finally, the authors found that filtering the samples of whole saliva before incubation with HIV-1 completely abolished the saliva's inhibitory activity.

From these findings, the authors concluded that "the inhibitory activity found in whole saliva is secreted by major salivary glands and does not exist in whole saliva simply because of leakage of serum components through oral lesions or through the gingival crevice." They propose that some macromolecular component is responsible for the inhibitory action. Loss of saliva's inhibitory effect after filtration of whole saliva through a 0.45-μm filter supports this hypothesis. Experiments are ongoing to identify this antiviral component. "It is worth noting," the authors state, "that saliva does not contain broad spectrum antiviral activity, that this HIV-1 inhibition is specific."

Hepatitis associated with recurrent Lyme disease

Lyme disease, a tick-borne, multisystemic disorder caused by the spirochete *Borrelia burgdorferi*, is not uncommon in certain regions of the northeastern United States, including areas of New York and Connecticut. Acute symptoms of the disease include malaise, fever, headache, arthralgia, myalgia, stiff neck, lymphadenopathy, paresthesia, and a distinctive rash. Neurologic, cardiac, and joint abnormalities can occur weeks or months after the initial illness. Goellner et al recently described a case of hepatitis due to recurrent Lyme disease (*Ann Intern Med* 1988; 108:707-708).

In June 1980, a 73-year-old woman experienced fever, chills, vomiting, myalgia, pedal paresthesia, arthralgia, bilateral ankle arthritis, and a generalized erythematous rash. Laboratory studies, including blood cultures, as well as other tests such as an electrocardiogram, chest roentgenogram, and barium enema, were negative. The patient's condition improved without therapy. Fever, palpitations, weight loss, and hip pain occurred two months later. Complement levels, urinalysis, serum protein electrophoresis, and bone marrow aspirate revealed no abnormalities. A tuberculin skin test and tests for hepatitis A and B were negative. Once again, her symptoms disappeared despite no specific therapy.

Her symptoms recurred five years later, in May 1986, when she sought treatment for fever, chills, rash, and anorexia. She also had two petechiae on the soft palate and maculopapular erythematous lesions on the abdomen and limbs. "A specimen obtained by needle biopsy of the liver showed moderate ballooning of hepatocytes, marked hepatocyte mitotic activity, prominent microvesicular fat, Kupffer cell hyperplasia, and sinusoidal infiltration by mononuclear cells . . . and polymorphonuclear neutrophils." Prednisone treatment yielded no improvement. An indirect fluorescent antibody test for *B burgdorferi* demonstrated an IgG antibody titer of 1:128 and a negative result for IgM antibody. The patient's symptoms responded to doxycycline treatment, 100 mg orally twice a day for three weeks. Later review of liver and skin biopsy specimens led to the identification of spirochetes.

Based on the clinical findings, the IgG and IgM titers,

NEWS BRIEFS are compiled and written by Vicki Glaser.

the visualization of spirochetes, and the response to doxycycline, the authors characterized the patient's later illness as Lyme disease, and they concluded that the illness described in 1980 was probably also due to Lyme disease. Either relapse or reinfection could have caused the later illness, since the patient lived in an area where Lyme disease is endemic. The marked ballooning of hepatocytes was similar to that seen in acute forms of viral hepatitis. "In our patient," the authors concurred, "hepatitis appears to have been the result of direct tissue invasion by the spirochete."

Medical groups develop plan to resolve malpractice crisis

The AMA/Specialty Society Medical Liability Project (ASA/SSMLP), a joint effort of the American Medical Association and 32 national medical specialty organizations, has proposed a fault-based administrative system for medical liability cases. Either state medical boards or a new state agency, whose members would be appointed by the governor, would oversee the legal process, replacing the existing court/jury system. This Board would include some physician members, but they would not represent a majority. The group believes that "more patients injured by medical negligence will be compensated under this plan, but that fewer dollars will be spent on meritless claims and unnecessary transaction costs."

In a written statement, the AMA/SSMLP outlined the three basic functions of the proposed system: claims resolution; credentialling and discipline; and codification of the legal elements of medical liability. Resolution of claims would first require presentation of a medical malpractice case to an expert administrative agency. At this stage, experienced claims reviewers would examine medical records and interview the persons involved. The group hopes that most claims would be dismissed or settled at this point in the process. If not, a hearing examiner would conduct a full hearing on the merits of the claim. The board would then review the hearing examiner's decision and would hold the power to award fees and determine the legal costs to the losing party. The AMA/SSMLP contends that this system will hasten the decision-making process and ensure the consistency of judgments and awards.

An interesting feature of the proposed system is that any claimant can request representation by a lawyer from the state agency's office of general counsel at no cost.

Under this system, an information clearinghouse would be established to record all settlements and awards as well as "periodic physician performance credentialling" reports, and reports by insurers of cancellations and failures to renew policies because of physician incompetence. The investigative branch of the state agency would use this information to identify patterns of substandard conduct.

With respect to the legal elements of medical liability, the proposed system would abolish "the rules governing standard of care based on custom and locality . . . in favor of a standard that focuses on whether the challenged actions fall within a range of reasonableness, to be determined by reference to the standards of a prudent and competent practitioner in the same or similar circumstances."

The AMA/SSMLP "invites discussion and debate of the proposal." This system must be tested in one or more states prior to recommendation as a widespread solution to the problem of medical professional liability.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Michael V. Aiello, MD, Brooklyn. Died May 19, 1988; age 54. Dr Aiello was a 1965 graduate of Medizinische Fakultaet der Universitaet Erlangen, Erlangen, Bayern. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. His memberships included the New York Ophthalmology Society, the American Academy of Ophthalmology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Rudolph J. Bauersachs, MD, Brooklyn. Died May 23, 1988; age 63. Dr Bauersachs was a 1949 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Thomas Carmine Beneventano, MD, Mamaroneck. Died April 28, 1988; age 56. Dr Beneventano was a 1957 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Radiology and the American College of Gastroenterology, and a Diplomate of the American Board of Radiology. He was a member of the New York Roentgen Society, the New York Academy of Medicine, the Radiological Society of North America, Inc, and the American Roentgen Ray Society. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Edward P. Bergin, MD, Pelham Manor. Died May 6, 1988; age 87. Dr Bergin was a 1931 graduate of Georgetown University School of Medicine, Washington, DC. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Herman Joseph Burman, MD, Hallandale, Florida. Died March 30, 1988; age 90. Dr Burman was a 1923 graduate of Tufts University School of Medicine, Boston. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the New York Rhino-Otolaryngological Society, the American Academy of Otolaryngology, the American Academy of Facial Plastic and Reconstructive Surgery, the Bronx County Medical Society, and the Medical Society of the State of New York.

John Charles Cardona, MD, Nyack. Died May 18, 1988; age 75. Dr Cardona was a 1939 graduate of the New York Medical College. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the New York Society of Laryngology and Otology, the American Academy of Otolaryngology, the Medical Society of the County of Rockland, and the Medical Society of the State of New York.

Stanley B. Chapman, MD, Bath. Died April 29, 1988; age 67. Dr Chapman was a 1945 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Steuben County Medical Society and the Medical Society of the State of New York.

Maurice H. Cowen, MD, New York. Died May 25, 1988; age 50. Dr Cowen was a 1964 graduate of the New York Medical College. He was a Diplomate of the American Board of Orthopedic Surgery. His memberships included the American Academy of Orthopaedic Surgeons, the New York County Medical Society, and the Medical Society of the State of New York.

John DiNoto, MD, Garden City. Died May 16, 1988; age 88. Dr DiNoto was a 1943 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine and a member of the New York County Medical Society and the Medical Society of the State of New York.

Richard B. Duane, MD, New York. Died May 12, 1988; age 70. Dr Duane was a 1943 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine and a member of the New York County Medical Society and the Medical Society of the State of New York.

James Francis Durkin, MD, Olean. Died May 4, 1988; age 77. Dr Durkin was a 1937 graduate of Georgetown University School of Medicine, Washington. He was a member of the Medical Society of the County of Cattaraugus and the Medical Society of the State of New York.

Joseph P. Gambacorta, MD, Buffalo. Died April 25, 1988; age 65. Dr Gambacorta was a 1948 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Diplomate of the American Board of Urology and a member of the New York State Urological Society, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

William Samuel Grosof, MD, New York. Died May 20, 1988; age 88. Dr Grosof was a 1925 graduate of the Medical College of Virginia Commonwealth University School of Medicine, Richmond. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Amir H. Hemmat, MD, Syracuse. Died April 25, 1988; age 61. Dr Hemmat was a 1953 graduate of the School of Medicine, National University of Iran, Tehran. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Abraham Karger, MD, Hollywood, Florida. Died April 15, 1988; age 83.

Dr Karger was a 1931 graduate of the University of Maryland School of Medicine, Baltimore. He was a member of the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Russell W. Lavengood, MD, New York. Died May 11, 1988; age 64. Dr Lavengood was a 1951 graduate of the University of Louisville School of Medicine, Louisville, Kentucky. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology. His memberships included the New York Academy of Medicine, the New York State Urological Society, the American Urological Association, the Pan American Medical Association, the American Geriatrics Society, the American Fertility Society, the New York County Medical Society, and the Medical Society of the State of New York.

James Liebmann Leland, MD, Bronx. Died May 6, 1988; age 81. Dr Leland was a 1933 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Leland was a Diplomate of the American Board of Internal Medicine and a member of the New York County Medical Society and the Medical Society of the State of New York.

Joseph Lintz, MD, New York. Died May 8, 1988; age 94. Dr Lintz was a 1916 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York Academy of Medicine, the New York Pathological Society, the New York County Medical Society, and the Medical Society of the State of New York.

Leon Merkin, MD, New York. Died March 6, 1988; age 88. Dr Merkin was a 1927 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin. He was a member of the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Alfred Ring, MD, Jamaica. Died February 16, 1988; age 85. Dr Ring was a 1927 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Physicians and the American College of Chest Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Samuel P. Schwartzfarb, MD, New York. Died January 14, 1988; age 82. Dr Schwartzfarb was a 1931 graduate of the New York University School of Medicine, New York. He was a member of the American Academy of Family Physicians, the Bronx County Medical Society, and the Medical Society of the State of New York.

James Edward Sheehy, MD, Syracuse. Died April 16, 1988; age 81. Dr Sheehy was a 1937 graduate of Duke University School of Medicine, Durham. He was a Diplomate of the American Board of Ophthalmology. His memberships included the American Academy of Ophthalmology, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Beverly Chew Smith, MD, New York. Died March 2, 1988; age 92. Dr Smith was a 1919 graduate of the University of Virginia School of Medicine, Charlottesville. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the Academy of Medicine, the New York State Surgical Society, the Clinical Society of the New York Diabetes Association, the New York County Medical Society, and the Medical Society of the State of New York.

Edward Tennen, MD, New York. Died May 16, 1988; age 72. Dr Tennen was a 1941 graduate of University J.K. Wydzial Lekarski, Lwow. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Armand R. Vanore, MD, Tonkawa, Oklahoma. Died December 6, 1987; age 90. Dr Vanore was a 1921 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Society of Abdominal Surgeons, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Saul Yafa, MD, Glens Falls. Died April 26, 1988; age 79. Dr Yafa was a 1933 graduate of Boston University School of Medicine, Boston. He was a member of the New York Academy of Medicine, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Warren, and the Medical Society of the State of New York.

Uniform requirements for manuscripts submitted to biomedical journals

INTERNATIONAL COMMITTEE OF MEDICAL JOURNAL EDITORS*

IN JANUARY 1978 a group of editors from some major biomedical journals published in English met in Vancouver, British Columbia, and decided on uniform technical requirements for manuscripts to be submitted to their journals. These requirements, including formats for bibliographic references developed for the Vancouver group by the National Library of Medicine, were published in three of the journals early in 1979. The Vancouver group evolved into the International Committee of Medical Journal Editors (ICMJE). At the October 1981 meeting the requirements were revised slightly and published in a second edition in 1982. Since then the group has issued several separate statements, and these have been incorporated into the main part of this, the third, edition.

Over 300 journals have agreed to receive manuscripts prepared in accordance with the initial, previously published, requirements. It is important to emphasize what these requirements imply and what they do not.

Firstly, the requirements are instructions to authors on how to prepare manuscripts, not to editors on publication style. (But many journals have drawn on these requirements for elements of their publication styles.)

Secondly, if authors prepare their manuscripts in the style specified in these requirements, editors of the participating journals will not return manuscripts for changes in these details of style. Even so, manuscripts may be altered by journals to conform with details of their own publication styles.

Thirdly, authors sending manuscripts to a participating journal should not try to prepare them in accordance with the publication style of that journal but should follow the "Uniform Requirements for Manuscripts Submitted to Biomedical Journals."

Nevertheless, authors *must also* follow the instructions to authors in the journal as to what topics are suitable for that journal and the types of papers that may be submitted (for example, original articles, reviews, case reports). In addition, the journal's instructions are likely to contain other requirements unique to that journal, such as number of copies of manuscripts, acceptable languages, length of articles, and approved abbreviations.

Participating journals are expected to state in their instructions to authors that their requirements are in accordance with "Uniform Requirements for Manuscripts Submitted to Biomedical Journals" and to cite a published version.

This document will be revised at intervals. Inquiries and comments from Central and North America about these requirements should be sent to Edward J. Huth, MD, *Annals of Internal Medicine*, 4200 Pine Street, Philadelphia, PA 19104, USA; those from other regions should be sent to Stephen P. Lock, MD, *British Medical Journal*, British Medical Association, Tavistock Square, London WC1H 9JR, United Kingdom. Note that these two journals provide secretariat services for the International Committee of Medical Journal Editors; they do not handle manuscripts intended for other journals. Papers intended for other journals should be sent directly to the offices of those journals.

SUMMARY OF REQUIREMENTS

Type the manuscript double spaced, including title page, abstract, text, acknowledgments, references, tables, and legends.

Each manuscript component should begin on a new page, in the following sequence.

- Title page
- Abstract and key words
- Text
- Acknowledgments
- References
- Tables: each table, complete with title and footnotes, on a separate page
- Legends for illustrations

Illustrations must be good-quality, unmounted glossy prints usually 127 by 173 mm (5 by 7 in) but no larger than 203 by 254 mm (8 by 10 in).

Submit the required number of copies of manuscript and figures (see journal's instructions) in a heavy-paper envelope. The submitted manuscript should be accompanied by a covering letter, as described under "Submission of Manuscripts," and permissions to reproduce previously published materials or to use illustrations that may identify human subjects.

Follow the journal's instructions for transfer of copyright. Authors should keep copies of everything submitted.

PRIOR AND DUPLICATE PUBLICATION

Most journals do not wish to consider for publication a paper on work that already has been reported in a published paper or is described in a paper submitted or accepted for publication elsewhere. This policy does not usually preclude consideration of a paper that has been rejected by another journal or of a complete report that follows publication of a preliminary report, usually in the form of an abstract. When submitting a paper, an author should always make a full statement to the editor about all submissions and previous reports that might be regarded as prior or duplicate publication of the same or very similar work. Copies of such material should be included with the submitted paper to help the editor decide how to deal with the matter.

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* Edward J. Huth, MD; *Annals of Internal Medicine*; Kathleen King, MRC PATH; *The Medical Journal of Australia*; Stephen P. Lock, MD; *British Medical Journal*; George D. Lundberg, MD; *Journal of the American Medical Association*; Ian Munro, MB; *The Lancet*; Magne Nylenna, MD; *Tidsskrift for Den Norske Laegeforening*; Roy Rada, MD; *Index Medicus*; Arnold S. Relman, MD; *New England Journal of Medicine*; Povl Riis, MD; *Journal of the Danish Medical Association* and *Danish Medical Bulletin*; Richard G. Robinson, ChM; *New Zealand Medical Journal*; Bruce P. Squires, MD; *Canadian Medical Association Journal*; Dr Ilkka Vartiovaara; *Finnish Medical Journal*; Malcolm S. M. Watts, MD; *The Western Journal of Medicine.*

Multiple publication—that is, the publication more than once of the same study results, irrespective of whether the wording is the same—is rarely justified. Secondary publication in another language is one possible justification, provided the following conditions are met.

(a) The editors of both journals concerned are fully informed; the editor concerned with secondary publication should have a photocopy, reprint, or manuscript of the primary version.
(b) The priority of the primary publication is respected by a publication interval of at least two weeks.
(c) The paper for secondary publication is written for a different group of readers and is not simply a translated version of the primary paper; an abbreviated version will often be sufficient.
(d) The secondary version reflects faithfully the data and interpretations of the primary version.
(e) A footnote on the title page of the secondary version informs readers, peers, and documenting agencies that the paper was edited, and is being published, for a national audience in parallel with a primary version based on the same data and interpretations. A suitable footnote might read as follows: "This article is based on a study first reported in the [title of journal, with full reference]."

Multiple publication other than as defined above is not acceptable to editors. If authors violate this rule, they may expect appropriate editorial action to be taken.

Preliminary release, usually to public media, of scientific information described in a paper that has been accepted but not yet published is a violation of the policies of many journals. In a few cases, and only by arrangement with the editor, preliminary release of data may be acceptable, for example, to warn the public of health hazards.

PREPARATION OF MANUSCRIPT

Type the manuscript on white bond paper, 216 by 279 mm (8½ by 11 in) or ISO A4 (212 by 297 mm), with margins of at least 25 mm (1 in). Type only on one side of the paper. Use double spacing throughout, including title page, abstract, text, acknowledgments, references, tables, and legends for illustrations. Begin each of the following sections on separate pages: title page, abstract and key words, text, acknowledgments, references, individual tables, and legends. Number pages consecutively, beginning with the title page. Type the page number in the upper or lower right-hand corner of each page.

Title Page. The title page should carry 1) the title of the article, which should be concise but informative; 2) first name, middle initial, and last name of each author, with highest academic degree(s) and institutional affiliation; 3) name of department(s) and institution(s) to which the work should be attributed; 4) disclaimers, if any; 5) name and address of author responsible for correspondence about the manuscript; 6) name and address of author to whom requests for reprints should be addressed, or statement that reprints will not be available from the author; 7) the source(s) of support in the form of grants, equipment, drugs, or all of these; and 8) a short running head or footline of no more than 40 characters (count letters and spaces) placed at the foot of the title page and identified.

Authorship. All persons designated as authors should qualify for authorship. Each author should have participated sufficiently in the work to take public responsibility for the content.

Authorship credit should be based only on substantial contributions to (a) conception and design, or analysis and interpretation of data; (b) drafting the article or revising it critically for important intellectual content; and on (c) final approval of the version to be published. Conditions (a), (b), and (c) must all be met. Participation solely in the acquisition of funding or the collection of data does not justify authorship. General supervision of the research group is also not sufficient for authorship. Any part of an article critical to its main conclusions must be the responsibility of at least one author.

A paper with corporate (collective) authorship must specify the key persons responsible for the article; others contributing to the work should be recognized separately (see Acknowledgments and Other Information).

Editors may require authors to justify the assignment of authorship.

Abstract and Key Words. The second page should carry an abstract of no more than 150 words. The abstract should state the purposes of the study or investigation; basic procedures (selection of study subjects or experimental animals, observational and analytic methods); main findings (give specific data and their statistical significance, if possible); and the principal conclusions. Emphasize new and important aspects of the study or observations.

Below the abstract, provide, and identify as such, 3 to 10 key words or short phrases that will assist indexers in cross-indexing your article and that may be published with the abstract. Use terms from the Medical Subject Headings (MeSH) list of *Index Medicus*; if suitable MeSH terms are not yet available for recently introduced terms, present terms may be used.

Text. The text of observational and experimental articles is usually—but not necessarily—divided into sections with the headings Introduction, Methods, Results, and Discussion. Long articles may need subheadings within some sections to clarify their content, especially the Results and Discussion sections. Other types of articles such as case reports, reviews, and editorials are likely to need other formats. Authors should consult individual journals for further guidance.

Introduction: State the purpose of the article. Summarize the rationale for the study or observation. Give only strictly pertinent references, and do not review the subject extensively. Do not include data or conclusions from the work being reported.

Methods: Describe your selection of the observational or experimental subjects (patients or experimental animals, including controls) clearly. Identify the methods, apparatus (manufacturer's name and address within parenthesis marks [round brackets]), and procedures in sufficient detail to allow other workers to reproduce the results. Give references to established methods, including statistical methods (see below); provide references and brief descriptions for methods that have been published but are not well known; describe new or substantially modified methods, give reasons for using them, and evaluate their limitations. Identify precisely all drugs and chemicals used, including generic name(s), dose(s), and route(s) of administration.

Ethics: When reporting experiments on human subjects, indicate whether the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional or regional) or with the Helsinki Declaration of 1975, as revised in 1983. Do not use patients' names, initials, or hospital numbers, expecially in any illustrative material. When reporting experiments on animals indicate whether the institution's or the National Research Council's guide for, or any national law on, the care and use of laboratory animals was followed.

Statistics: Describe statistical methods with enough detail to enable a knowledgeable reader with access to the original data to verify the reported results. When possible, quantify findings and present them with appropriate indicators of measurement error or uncertainty (such as confidence intervals). Avoid sole reliance on statistical hypothesis testing, such as the use of *P* values, which fails to convey important quantitative information. Discuss eligibility of experimental subjects. Give details about randomization. Describe the methods for, and success of, any blinding of observations. Report treatment complications. Give numbers of observations. Report losses to observation (such as dropouts from a clinical trial). References for study design and statistical methods should be to standard works (with pages stated) when possible rather than to papers where designs or methods were originally reported. Specify any general-use computer programs used.

Put general descriptions of methods in the Methods section. When data are summarized in the Results section, specify the statistical methods used to analyze them. Restrict tables and figures to those needed to explain the argument of the paper and to assess its support. Use graphs as an alternative to tables with many entries; do not duplicate data in graphs and tables. Avoid non-technical uses of technical terms in statistics, such as "random" (which implies a randomizing device), "normal," "significant," "correlation," and "sample." Define statistical terms, abbreviations, and most symbols.

Results: Present your results in logical sequence in the text, tables, and illustrations. Do not repeat in the text all the data in the tables, illustrations, or both; emphasize or summarize only important observations.

Discussion: Emphasize the new and important aspects of the study and the conclusions that follow from them. Do not repeat in detail data or other material given in the Introduction or the Results section. Include in the Discussion section the implications of the findings and their limitations, including implications for future research. Relate the observations to other relevant studies. Link the conclusions with the goals of the study but avoid unqualified statements and conclusions not completely supported by your data. Avoid claiming priority and alluding to work that has not been completed. State new hypotheses when warranted, but clearly label them as such. Recommendations, when appropriate, may be included.

Acknowledgments. At an appropriate place in the article (title-page footnote or appendix to the text; see the journal's requirement) one or more statements should specify: (a) contributions that need acknowledging but do not justify authorship, such as general support by a departmental chairman; (b) acknowledgments of technical help; (c) acknowledgments of financial and material support, specifying the nature of the support; (d) financial relationships that may pose a conflict of interest.

Persons who have contributed intellectually to the paper but whose contributions do not justify authorship may be named and their function or contribution described, for example, "scientific adviser," "critical review of study proposal," "data collection," "participation in clinical trial." Such persons must have given their permission to be named. Authors are responsible for obtaining written permission from persons acknowledged by name because readers may infer their endorsement of the data and conclusions.

Technical help should be acknowledged in a paragraph separate from those acknowledging other contributions.

References. Number references consecutively in the order in which they are first mentioned in the text. Identify references in text, tables, and legends by arabic numerals within parenthesis marks. References cited only in tables or in legends to figures should be numbered in accordance with a sequence established by the first identification in the text of the particular table or illustration.

Use the style of the examples below, which are based on the formats used by the US National Library of Medicine in *Index Medicus*. The titles of journals should be abbreviated according to the style used in *Index Medicus*. Consult *List of Journals Indexed in Index Medicus*, published annually as a separate publication by the Library and as a list in the January issue of *Index Medicus*; also see the list of journal titles and abbreviated titles at the end of this document.

Try to avoid using abstracts as references; "unpublished observations" and "personal communications" may not be used as references, although references to written, not oral, communications may be inserted (within parenthesis marks) in the text. Include among the references papers accepted but not yet published; designate the journal and add "in press" (within parenthesis marks). Information from manuscripts submitted but not yet accepted should be cited in the text as "unpublished observations" (within parenthesis marks).

The references must be verified by the author(s) against the original documents.

Examples of correct forms of references are given below.

Journals

1. Standard Journal Article (List all authors when six or less; when seven or more, list only first three and add et al.)
   You CH, Lee KY, Chey RY, Menguy R. Electrogastrographic study of patients with unexplained nausea, bloating and vomiting. Gastroenterology 1980;79:311–4.
2. Corporate Author
   The Royal Marsden Hospital Bone-Marrow Transplantation Team. Failure of syngeneic bone-marrow graft without preconditioning in post-hepatitis marrow aplasia. Lancet 1977;2:242–4.
3. No Author Given
   Anonymous. Coffee drinking and cancer of the pancreas [Editorial]. Br Med J 1981;283:628.
4. Journal Supplement
   Mastri AR. Neuropathy of diabetic neurogenic bladder. Ann Intern Med 1980;92(2 Pt 2):316–8. Frumin AM, Nussbaum J, Esposito M. Functional asplenia: demonstration of splenic activity by bone marrow scan [Abstract]. Blood 1979; 54(Suppl 1):26a.
5. Journal Paginated by Issue
   Seaman WB. The case of the pancreatic pseudocyst. Hosp Pract 1981;16(Sep):24–5.

Books and Other Monographs

6. Personal Author(s)
   Eisen HN. Immunology: an introduction to molecular and cellular principles of the immune response. 5th ed. New York: Harper and Row, 1974;406.
7. Editor, Compiler, Chairman as Author
   Dausset J, Colombani J, eds. Histocompatibility testing 1972. Copenhagen: Munksgaard, 1973:12–8.
8. Chapter in a Book
   Weinstein L, Swartz MN. Pathogenic properties of invading microorganisms. In: Sodeman WA Jr, Sodeman WA, eds. Pathologic physiology: mechanisms of disease. Philadelphia: WB Saunders, 1974:457–72.
9. Published Proceedings Paper
   DuPont B. Bone marrow transplantation in severe combined immunodeficiency with an unrelated MLC compatible donor. In: White HJ, Smith R, eds. Proceedings of the third annual meeting of the International Society for Experimental Hematology. Houston: International Society for Experimental Hematology, 1974:44–6.
10. Monograph in a Series
    Hunninghake GW, Gadek JE, Szapiel SV, et al. The human alveolar macrophage. In: Harris CC, ed. Cultured human cells and tissues in biomedical research. New York: Academic Press, 1980:54–6. (Stoner GD, ed. Methods and perspectives in cell biology; vol 1).
11. Agency Publication
    Ranofsky AL. Surgical operations in short-stay hospi-

tals: United States-1975. Hyattsville, Maryland: National Center for Health Statistics, 1978; DHEW publication no. (PHS) 78-1785, (Vital and health statistics; series 13; no 34).

12. Dissertation or Thesis
Cairns RB. Infrared spectroscopic studies of solid oxygen [Dissertation]. Berkeley, California: University of California, 1965. 156 p.

Other Articles

13. Newspaper Article
Shaffer RA. Advances in chemistry are starting to unlock mysteries of the brain: discoveries could help cure alcoholism and insomnia, explain mental illness. How the messengers work. Wall Street Journal 1977 Aug 12:1(col 1), 10(col 1).
14. Magazine Article
Roueche B. Annals of medicine: the Santa Claus culture. The New Yorker 1971 Sep 4:66-81.

Tables. Type each table double spaced on a separate sheet. Do not submit tables as photographs. Number tables consecutively in the order of their first citation in the text and supply a brief title for each. Give each column a short or abbreviated heading. Place explanatory matter in footnotes, not in the heading. Explain in footnotes all nonstandard abbreviations that are used in each table. For footnotes, use the following symbols, in this sequence: *, †, ‡, §, ‖, ¶, **, ††, ...

Identify statistical measures of variations such as standard deviation and standard error of the mean.

Do not use internal horizontal and vertical rules.

Be sure that each table is cited in the text.

If you use data from another published or unpublished source, obtain permission and acknowledge fully.

The use of too many tables in relation to the length of the text may produce difficulties in the layout of pages. Examine issues of the journal to which you plan to submit your paper to estimate how many tables can be used per 1000 words of text.

The editor, on accepting a paper, may recommend that additional tables containing important back-up data too extensive to publish be deposited with an archival service, such as the National Auxiliary Publication Service (NAPS) in the United States, or made available by the authors. In that event, an appropriate statement will be added to the text. Submit such tables for consideration with the paper.

Illustrations. Submit the required number of complete sets of figures. Figures should be professionally drawn and photographed; freehand or typewritten lettering is unacceptable. Instead of original drawings, roentgenograms, and other material, send sharp, glossy black-and-white photographic prints, usually 127 by 173 mm (5 by 7 in) but no larger than 203 by 254 mm (8 by 10 in). Letters, numbers, and symbols should be clear and even throughout, and of sufficient size that when reduced for publication each item will still be legible. Titles and detailed explanations belong in the legends for illustrations, not on the illustrations themselves.

Each figure should have a label pasted on its back indicating the number of the figure, author name, and the top of the figure. Do not write on the back of the figures, or scratch or mar them using paper clips. Do not bend figures or mount them on cardboard.

Photomicrographs must have internal scale markers. Symbols, arrows, or letters used in the photomicrographs should contrast with the background.

If photographs of persons are used, either the subjects must not be identifiable or their pictures must be accompanied by written permission to use the photograph.

Figures should be numbered consecutively according to the order in which they have been first cited in the text. If a figure has been published, acknowledge the original source and submit written permission from the copyright holder to reproduce the material. Permission is required, irrespective of authorship or publisher, except for documents in the public domain.

For illustrations in color, ascertain whether the journal requires color negatives, positive transparencies, or color prints. Accompanying drawings marked to indicate the region to be reproduced may be useful to the editor. Some journals publish illustrations in color only if the author pays for the extra cost.

Legends for Illustrations. Type legends for illustrations double spaced, starting on a separate page, with arabic numerals corresponding to the illustrations. When symbols, arrows, numbers, or letters are used to identify parts of the illustrations, identify and explain each one clearly in the legend. Explain internal scale and identify method of staining in photomicrographs.

UNITS OF MEASUREMENT

Measurements of length, height, weight, and volume should be reported in metric units (metre, kilogram, litre) or their decimal multiples.

Temperatures should be given in degrees Celsius. Blood pressures should be given in millimetres of mercury.

All hematologic and clinical chemistry measurements should be reported in the metric system in terms of the International System of Units (SI). Editors may request that alternative or non-SI units be added by the author before publication.

ABBREVIATIONS AND SYMBOLS

Use only standard abbreviations. Avoid abbreviations in the title and abstract. The full term for which an abbreviation stands should precede its first use in the text unless it is a standard unit of measurement.

SUBMISSION OF MANUSCRIPTS

Mail the required number of manuscript copies in a heavy-paper envelope, enclosing the manuscript copies and figures in cardboard, if necessary, to prevent bending of photographs during mail handling. Place photographs and transparencies in a separate heavy-paper envelope.

Manuscripts must be accompanied by a covering letter. This must include (a) information on prior or duplicate publication or submission elsewhere of any part of the work; (b) a statement of financial or other relationships that might lead to a conflict of interest; (c) a statement that the manuscript has been read and approved by all authors; and (d) the name, address, and telephone number of the corresponding author, who is responsible for communicating with the other authors about revisions and final approval of the proofs. The letter should give any additional information that may be helpful to the editor, such as the type of article in the particular journal the manuscript represents and whether the author(s) will be willing to meet the cost of reproducing color illustrations.

The manuscript must be accompanied by copies of any permissions to reproduce published material, to use illustrations or report sensitive personal information of identifiable persons, or to name persons for their contributions.

Participating Journals. The journals listed in Table 1 are those that notified the ICMJE of their willingness to consider for publication manuscripts prepared in accordance with the guidance given in the second (1982) edition of the Uniform Requirements for the Submission of Manuscripts to Biomedical Journals. Their listing here does not imply that they endorse this present version (3rd) of "Uniform Requirements . . . ". The *Index Medicus* abbreviations for journal titles are given within square brackets.

TABLE 1. Journals Participating in the Uniform Requirements Agreement

Acta Medica Colombiana [Acta Med Colomb]
Acta Orthopaedica Scandinavica [Acta Orthop Scand]
Acta Paediatrica Japonica [Acta Paediatr Jpn (Overseas)]
Acta Paediatrica Scandinavica [Acta Paediatr Scand]
Acta Pharmacologica Sinica [Acta Pharmacol Sin]
Activox [Activox]
AIDS: An International Bimonthly Journal [AIDS]
American Family Physician [Am Fam Physician]
The American Journal of Cardiology [Am J Cardiol]
The American Journal of Clinical Nutrition [Am J Clin Nutr]
American Journal of Diseases of Children [Am J Dis Child]
The American Journal of Emergency Medicine [Am J Emerg Med]
American Journal of Epidemiology [Am J Epidemiol]
American Journal of Hospital Pharmacy [Am J Hosp Pharm]
The American Journal of Human Genetics [Am J Hum Genet]
The American Journal of Medicine [Am J Med]
American Journal of Obstetrics and Gynecology [Am J Obstet Gynecol]
American Journal of Optometry and Physiological Optics [Am J Optom Physiol Opt]
The American Journal of Pathology [Am J Pathol]
The American Journal of Psychiatry [Am J Psychiatry]
The American Journal of Public Health [Am J Public Health]
AJR: American Journal of Roentgenology [AJR]
The American Journal of Surgery [Am J Surg]
American Review of Respiratory Disease [Am Rev Respir Dis]
The American Surgeon [Am Surg]
Anaesthesia [Anaesthesia]
Anaesthesia and Intensive Care [Anaesth Intensive Care]
Anesthesia and Analgesia [Anesth Analg]
Annals of Clinical Biochemistry [Ann Clin Biochem]
Annals of Clinical and Laboratory Science [Ann Clin Lab Sci]
Annals of Internal Medicine [Ann Intern Med]
The Annals of Otology, Rhinology and Larynogology [Ann Otol Rhinol Laryngol]
Annals of the Rheumatic Diseases [Ann Rheum Dis]
The Annals of the Royal College of Physicians and Surgeons of Canada [Ann R Coll Physicians Surg Can]
Annals of the Royal College of Surgeons of England [Ann R Coll Surg Engl]
Annals of Surgery [Ann Surg]
The Annals of Thoracic Surgery [Ann Thorac Surg]
Annals of Tropical Paediatrics [Ann Trop Paediatr]
Archives of Dermatology [Arch Dermatol]
Archives of Disease in Childhood [Arch Dis Child]
Archives of General Psychiatry [Arch Gen Psychiatry]
Archives of Internal Medicine [Arch Intern Med]
Archives of Neurology [Arch Neurol]
Archives of Ophthalmology [Arch Ophthalmol]
Archives of Otolarynogology—Head and Neck Surgery [Arch Otolaryngol]
Archives of Pathology and Laboratory Medicine [Arch Pathol Lab Med]
Archives of Surgery [Arch Surg]
Archivos de Investigacion Medica [Arch Invest Med (Mex)]
Arizona Medicine [Ariz Med]
Arteriosclerosis: A Journal of Vascular Biology and Thrombosis [Arteriosclerosis]
Australasian Journal of Dermatology [Australas J Dermatol]
Australian and New Zealand Journal of Medicine [Aust NZ J Med]
Australian and New Zealand Journal of Ophthalmology [Aust NZ J Ophthalmol]
The Australian and New Zealand Journal of Surgery [Aust NZ J Surg]
Australian Family Physician [Aust Fam Physician]
Australian Journal of Hospital Pharmacy [Aust J Hosp Pharm]
Australian Orthoptic Journal [Aust Orthopt J]
Australian Paediatric Journal [Aust Paediatr J]
Bangladesh Journal of Child Health [Bangladesh J Child Health]
Bibliothek for Laeger [Bibl Laeger]
Biomedical Bulletin [Biomed Bull]
Boletin de la Asociacion Medica de Puerto Rico [Bol Asoc Med PR]
Boletin Medico del Hospital Infantil de Mexico [Bol Med Hosp Infant Mex]
Bordeaux Medical [Bord Med]
Brain and Development [Brain Dev]
British Dental Journal [Br Dent J]
British Heart Journal [Br Heart J]
British Homoeopathic Journal [Br Homoeopath J]
British Journal of Anaesthesia [Br J Anaesth]
British Journal of Industrial Medicine [Br J Ind Med]
British Journal of Occupational Therapy [Br J Occup Ther]
British Journal of Ophthalmology [Br J Ophthalmol]
British Journal of Pain [Br J Pain]
British Journal of Rheumatology [Br J Rheumatol]
British Journal of Surgery [Br J Surg]
British Medical Bulletin [Br Med Bull]
British Medical Journal [Br Med J]
Bulletin of the Medical Library Association [Bull Med Libr Assoc]
British Osteopathic Journal [Br Osteopath J]
Bulletin of the World Health Organization [Bull WHO]
Canadian Family Physician [Can Fam Physician]
Canadian Journal of Anaesthesia [Can J Anaesthes]
Canadian Journal of Comparative Medicine [Can J Comp Med]
Canadian Journal of Public Health [Can J Public Health]
Canadian Journal of Surgery [Can J Surg]
Canadian Medical Association Journal [Can Med Assoc J]
Canadian Veterinary Journal [Can Vet J]
Cardiovascular Research [Cardiovasc Res]
Central African Journal of Medicine [Cent Afr J Med]
Cephalalgia [Cephalalgia]
Chest [Chest]
Chinese Journal of Anesthesiology [Chin J Anesthesiol]
Chinese Journal of Cardiovascular Disease [Chin J Cardiovasc Dis]
Chinese Journal of Dermatology [Chin J Dermatol]
Chinese Journal of Digestion [Chin J Dig]
Chinese Journal of Endocrinology and Metabolism [Chin J Endocrinol Metab]
Chinese Journal of Epidemiology [Chin J Epidemiol]
Chinese Journal of Experimental Surgery [Chin J Exp Surg]
Chinese Journal of Geriatology [Chin J Geriatol]
Chinese Journal of Hematology [Chin J Hematol]
Chinese Journal of Hospital Administration [Chin J Hosp Adm]
Chinese Journal of Industrial Hygiene and Occupational Disease [Chin J Ind Hyg Occup Dis]
Chinese Journal of Infectious Diseases [Chin J Infect Dis]
Chinese Journal of Internal Medicine [Chin J Intern Med]
Chinese Journal of Medical History [Chin J Med Hist]
Chinese Journal of Medical Laboratory Technology [Chin J Med Lab Technol]
Chinese Journal of Microbiology and Immunology [Chin J Microbiol Immunol]
Chinese Journal of Nephrology [Chin J Nephrol]
Chinese Journal of Neurology and Psychiatry [Chin J Neurol Psychiatr]
Chinese Journal of Neurosurgery [Chin J Neurosurg]
Chinese Journal of Nuclear Medicine [Chin J Nucl Med]
Chinese Journal of Obstetrics and Gynecology [Chin J Obstet Gynecol]
Chinese Journal of Oncology [Chin J Oncol]
Chinese Journal of Ophthalmology [Chin J Ophthalmol]
Chinese Journal of Organ Transplantation [Chin J Organ Transplant]
Chinese Journal of Orthopedics [Chin J Orthop]
Chinese Journal of Otolaryngology [Chin J Otolaryngol]
Chinese Journal of Pathology [Chin J Pathol]
Chinese Journal of Pediatric Surgery [Chin J Pediatr Surg]
Chinese Journal of Pediatrics [Chin J Pediatr]

Table 1 (continued)

Chinese Journal of Physical Medicine [Chin J Phys Med]
Chinese Journal of Physical Therapy [Chin J Phys Ther]
Chinese Journal of Plastic Surgery and Burns [Chin J Plast Surg Burn]
Chinese Journal of Preventive Medicine [Chin J Prev Med]
Chinese Journal of Radiological Medicine and Protection [Chin J Radiol Med]
Chinese Journal of Radiology [Chin J Radiol]
Chinese Journal of Stomatology [Chin J Stomatol]
Chinese Journal of Surgery [Chin J Surg]
Chinese Journal of Tuberculosis and Respiratory Diseases [Chin J Tuberc Respir Dis]
Chinese Journal of Urology [Chin J Urol]
Chinese Medical Journal [Chin J Med]
Chronic Diseases in Canada [Chronic Dis Can]
Circulation [Circulation]
Clinica Chimica Acta [Clin Chim Acta]
Clinical Chemistry [Clin Chem]
Clinical and Experimental Optometry [Clin Exp Optom]
Clinical Diabetes [Clin Diabet]
Clinical and Investigative Medicine [Clin Invest Med]
Clinical Pediatrics [Clin Pediatr (Phila)]
Clinical Pharmacology and Therapeutics [Clin Pharmacol Ther]
Clinical Pharmacy [Clin Pharm]
Clinical Preventive Dentistry [Clin Prev Dent]
Community Dentistry and Oral Epidemiology [Community Dent Oral Epidemiol]
Community Medicine [Community Med]
Cuadernos del Hospital de Clinicas [Cua Hosp Clin]
Danish Dental Journal [Dan Dent J]
Danish Medical Bulletin [Dan Med Bull]
Diabetes [Diabetes]
Diabetes Care [Diabetes Care]
Diabetes Journal [Diabetes J]
Diabetologia [Diabetologia]
Diagnostic Cytopathology [Diagn Cytopathol]
Drug Intelligence and Clinical Pharmacy [Drug Intell Clin Pharm]
Environmental Medicine [Environ Med]
European Heart Journal [Eur Heart J]
European Journal of Cancer and Clinical Oncology [Eur J Cancer Clin Oncol]
European Journal of Clinical Investigation [Eur J Clin Invest]
European Journal of Respiratory Diseases [Eur J Respir Dis]
European Journal of Rheumatology and Inflammation [Eur J Rheumatol Inflamm]
Family Medicine [Fam Med]
Family Practice Research Journal [Fam Pract Res J]
The Finnish Medical Journal [Finn Med J]
Gastroenterology [Gastroenterology]
Gastrointestinal Endoscopy [Gastrointest Endosc]
Genitourinary Medicine [Genitourin Med]
Geriatrics [Geriatrics]
Gut [Gut]
Hawaii Medical Journal [Hawaii Med J]
Health Trends [Health Trends]
Hellenike Cheirourgike [Hell Cheir]
Helleniki Iatrike [Hell Iatr]
Hong Kong Medical Technology Association Journal [Hong Kong Med Technol Assoc J]
Hospital Pharmacy [Hosp Pharm]
Iatrike [Iatrike]
Indian Journal of Dermatology, Venereology and Leprology [Indian J Dermatol Venereol Lepr]
Indian Journal of Gastroenterology [Indian J Gastroenterol]
Indian Journal of Urology [Indian J Urol]
International Disability Studies [Int Disabil Stud]
International Journal of Epidemiology [Int J Epidemiol]
International Journal of Pediatric Nephrology [Int J Pediatr Nephrol]
International Surgery [Int Surg]
Israel Journal of Psychiatry and Related Sciences [Isr J Psychiatry Relat Sci]
JAMA (Chicago) [JAMA]
The Journal of Allergy and Clinical Immunology [J Allergy Clin Immunol]
Journal of the American College of Cardiology [J Am Coll Cardiol]
Journal of the American Medical Association (see *JAMA*)
The Journal of Applied Nutrition [J Appl Nutr]
Journal of Biological Standardization [J Biol Stand]
Journal of the British Association for Immediate Care [J Br Assoc Immed Care]
Journal of the Canadian Association of Radiologists [J Can Assoc Radiol]
Journal of the Canadian Chiropractic Association [J Can Chiropr Assoc]
Journal of Cardiovascular Surgery [J Cardiovasc Surg]
Journal of Chronic Diseases [J Chronic Dis]
Journal of Clinical Gastroenterology [J Clin Gastroenterol]
Journal of Clinical Pathology [J Clin Pathol]
Journal of the Danish Medical Association (see *Ugeskrift for Laeger*)
The Journal of Diabetic Complications [J Diabetic Compl]
Journal of Diarrhoeal Disease Research [J Diarrhoeal Dis Res]
Journal of Epidemiology and Community Health [J Epidemiol Community Health]
Journal of the Faculty of Medicine Baghdad [J Fac Med Baghdad]
Journal of the Institute of Medicine [J Inst Med]
Journal of the Irish Colleges of Physicians and Surgeons [J Ir Coll Physicians Surg]
The Journal of Laboratory and Clinical Medicine [J Lab Clin Med]
The Journal of Maternal and Child Health [J Maternal Child Health]
Journal of Manipulative and Physiological Therapeutics [J Manipulative Physiol Ther]
Journal of Medical Ethics [J Med Ethics]
Journal of Medical Genetics [J Med Genet]
Journal of the National Cancer Institute [JNCI]
Journal of Neurology, Neurosurgery and Psychiatry [J Neurol Neurosurg Psychiatry]
Journal of Neuropathology and Experimental Neurology [J Neuropathol Exp Neurol]
The Journal of Nuclear Medicine [J Nucl Med]
Journal of Nuclear Medicine Technology [J Nucl Med Technol]
The Journal of Palliative Care [J Palliat Care]
Journal of Pathology [J Pathol]
Journal of Pharmacy Technology [J Pharm Technol]
Journal of Psychosomatic Research [J Psychosom Res]
Journal of the Royal Army Medical Corps [J R Army Med Corps]
Journal of the Royal College of Physicians of London [J R Coll Physicians Lond]
Journal of the Royal College of Surgeons of Edinburgh [J R Coll Surg Edinb]
Journal of the Royal Naval Medical Service [J R Nav Med Serv]
Journal of the Vivekananda Institute of Medical Sciences [J Vivekananda Inst Med Sci]
Lakartidningen [Lakartidningen]
The Lancet [Lancet]
Leprosy Review [Lepr Rev]
Malaysian Journal of Pathology [Malays J Pathol]
Manedsskrift for Praktisk Laegegerning [Manedsskr Prakt Laegegern]
Medicina Intensiva [Med Intensiv]
Medical Care [Med Care]
Medical and Pediatric Oncology [Med Pediatr Oncol]
The Medical Journal of Australia [Med J Aust]
Medical Laboratory Sciences [Med Lab Sci]
Medicina Clinica [Med Clin (Barc)]
Medicine (*Oxford*) [Medicine (Oxford)]
Military Medicine [Milit Med]

Table 1 (continued)

The Mount Sinai Journal of Medicine [Mt Sinai J Med (NY)]
National Medical Journal of China [Chung Hua I Hsueh Tsa Chih]
Nederlands Tijdschrift voor Geneeskunde [Ned Tijdschr Geneeskd]
Neurology [Neurology]
New Doctor [N Doctor]
The New England Journal of Medicine [N Engl J Med]
New York State Journal of Medicine [NY State J Med]
New Zealand Family Physician [NZ Fam Physician]
New Zealand Journal of Medical Laboratory Technology [NZ J Med Lab Technol]
New Zealand Medical Journal [NZ Med J]
Newfoundland Medical Association Journal [Newfoundland Med Assoc J]
Nigerian Medical Journal [Niger Med J]
No To Hattatsu [No To Hattatsu]
Nordisk Medicin [Nord Med]
North Carolina Medical Journal [NC Med J]
Nosokomaka Chronica [Nosokom Chron]
Nursing [Nursing]
Obstetrics and Gynecology [Obstet Gynecol]
Ophthalmology [Ophthalmology]
Otolaryngology and Head and Neck Surgery [Otolaryngol Head Neck Surg]
Papua New Guinea Medical Journal [Papua New Guinea Med J]
Pharmacy Management Combined with the American Journal of Pharmacy [Pharm Manage Comb Am J Pharm]
Pathology [Pathology]
Pediatric Emergency Care [Pediatr Emerg Care]
Peritoneal Dialysis Bulletin [Perit Dial Bull]
Pharmaceutisch Weekblad [Pharm Weekbl]
Pharmaceutisch Weekblad. Scientific Edition [Pharm Weekbl (Sci)]
Pharmacological Research Communications [Pharmacol Res Commun]
Pharmacotherapy [Pharmacotherapy]
The Physician and Sports Medicine [Physician Sports Med]
Postgraduate Doctor—Africa [Postgrad Doctor Afr]
Postgraduate Doctor—Asia [Postgrad Doctor Asia]
Postgraduate Medical Journal [Postgrad Med J]
Postgraduate Medicine [Postgrad Med]
Psychiatria Fennica [Psychiatr Fenn]
Public Health [Public Health]
Puerto Rico Health Sciences Journal [PR Health Sci J]
Quarterly Journal of Medicine [Q J Med]
Radiology [Radiology]
Revista Chilena de Pediatria [Rev Chil Pediatr]
Revista Clinica Espanola [Rev Clin Esp]
Revista de Gastroenterologia de Mexico [Rev Gastroenterol Mex]
Revista Espanola de Reumatologia [Rev Esp Reumatol]
Revista Medica de Chile [Rev Med Chil]
Revista Medica del Instituto Mexicano del Seguro Social [Rev Med Inst Mex Seguro Soc]
Revista Mexicana de Anestesiologia [Rev Mex Anestesiol]
Revista Mexicana de Radiologia [Rev Mex Radiol]
Salud Publica de Mexico [Salud Publica Mex]
Saudi Medical Journal [Saudi Med J]
Scandinavian Journal of Dental Research [Scand J Dent Res]
Scandinavian Journal of Haematology [Scand J Haematol]
Schumpert Medical Quarterly [Schumpert Med Q]
Schweizerische Medizinische Wochenschrift [Schweiz Med Wochenschr]
Sexually Transmitted Diseases [Sex Transm Dis]
Shinkei Byorigaku [Shinkei Byorigaku]
South African Medical Journal [S Afr Med J]
Southern Medical Journal [South Med J]
The Springfield Clinic Medical Bulletin [Springfield Clin Med Bull]
Sri Lankan Family Physician [Sri Lankan Fam Physician]
Thorax [Thorax]
Tidsskrift for den Norske Laegeforening [Tidsskr Nor Laegeforen]
Transactions—American Society for Artificial Internal Organs [Trans Am Soc Artif Intern Organs]
Transfusion [Transfusion]
Tropical Gastroenterology [Trop Gastroenterol]
Ugeskrift for Laeger [Ugeskr Laeger]
Ulster Medical Journal [Ulster Med J]
Undersea Biomedical Research [Undersea Biomed Res]
Veterinary Radiology [Vet Radiol]
The West Virginia Medical Journal [W Va Med J]
The Western Journal of Medicine [West J Med]
WHO Chronicle [WHO Chron]
World Health Statistics Quarterly [World Health Stat Q]
World Medical Journal [World Med J]
Yale Journal of Biology and Medicine [Yale J Biol Med]

FOR MY PATIENTS

NUMBER 2/1988

DECLARE W.A.R.R. ON SKIN CANCERS

Dear Patient,

Since the beginning of time, the sun has been called nature's Great Healer. But now medical scientists know that if our skin is exposed to too much of the sun's rays, great harm can develop. With more than half a million cases occurring each year, skin cancer is the most common of all cancers in the United States. The most frequent type is called ***basal cell carcinoma*** which is the most benign. The next frequent type is ***squamous cell carcinoma*** which is also benign and slow-growing. The least common and most serious type is ***melanoma*** which can be fatal if not treated early.

All of the above skin cancers are related to sun exposure, and all have a lag time (incubation period) of 10-20 years. The harmful effects of the sun are especially true for the basal cell and squamous cell cancers.

In recent years, the incidence of melanoma has been increasing at a faster rate in this country than any other cancer except lung cancer in women. Like cancers in general, all skin cancer rates increase with age. But they are now being seen in larger numbers of young people.

LINKING THE SUN WITH THE CANCERS

. . . More skin cancers develop on areas of the skin which are usually exposed to the sun. . . . More skin cancers occur in outdoor workers. . . . More skin cancers are found in warmer latitudes. . . . Light-skinned persons are more at risk of developing skin cancers than dark-skinned persons. . . . Skin cancers have been produced in experimental studies by exposing animals repeatedly to ultraviolet radiation similar to that of the sun.

WHO ARE AT RISK FOR MELANOMA?

Melanoma affects white persons ten times more often than black persons. The following factors in whites present the highest risks:

- Light eye color
- Light hair color
- Light complexion
- Tan poorly; sunburn easily
- Freckle easily
- Many moles on body

Tumor-causing viruses, chemical carcinogens, and genetic factors may also play a role in causing melanomas.

WHICH MOLES LOOK SUSPICIOUS?

You should examine your entire skin with a full-body mirror every two months and use the **ABCD** guidelines for early detection of suspected melanomas:

A. Asymmetry (one-half of the mole doesn't look like the other)

B. Border irregularity (the outline of the mole is not smoothly round or oval in shape)

C. Color variability (play of colors from browns to blacks and sometimes red, white, or blue)

D. Diameter of the mole is larger than a quarter of an inch.

You should examine your entire naked "birthday suit" every two months and ask yourself: "Have any of my moles or skin marks changed in size, shape, or color since the last time?" If you think they look different, contact my office for an early appointment.

The true diagnosis must be made by removing the suspicious mole or mark by minor office surgery and examining its cells under a microscope by a pathologist.

WHAT ABOUT PREVENTION OF SKIN CANCER

1. Declare *W.A.R.R.*

Wear protective clothing over skin exposed to the sun.

Apply number 15 (or higher) sunblocker on all exposed areas in summer sun.

Refrain from unnecessary sun exposure.

Remove certain moles that look suspicious (let me examine them as soon as possible).

2. Use *Sense in the Sun*

Avoid summer sun exposure between 10 a.m. and 3 p.m.

Between 10 a.m. and 3 p.m., use sunblocker (no. 15 or higher) on all exposed skin areas or stay completely in the shade.

Avoid all indoor tanning methods using any form of ultraviolet lights. They are as dangerous to your skin as the natural sun.

Follow the advice, and you will likely prevent skin cancer. Most skin cancers are preventable. It's up to you to take control steps and lower your lifetime risk.

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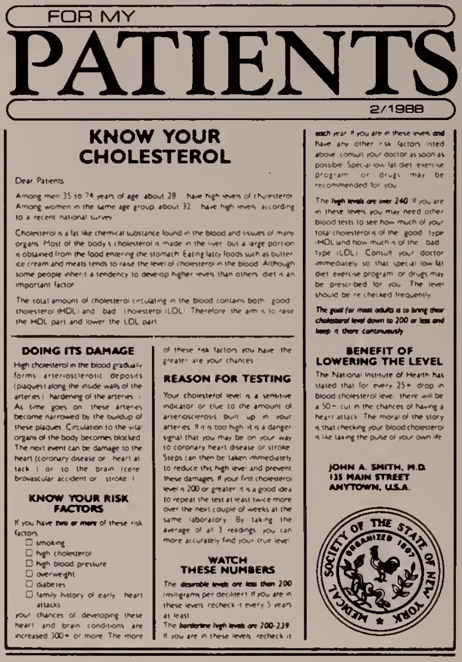
Office Handouts for Your Patients

This issue of the *New York State Journal of Medicine* contains a special additional item, "For My Patients." This feature will be run in future issues as well.

Use this new feature to keep your patients informed about timely issues concerning diseases, treatments and health maintenance. Simply make photocopies of the page and distribute them to your patients with your newsletter or other correspondence, or place them in your reception area for your patients to read and take home.

MSSNY and the *New York State Journal of Medicine* have waived the copyright for these handouts, so you may duplicate them without permission.

As an added feature, MSSNY will personalize this form and make copies for you, at a nominal charge. See the example below for how these forms would look with your name and address professionally printed on them. Use the order form below, and we'll ship the personalized forms to you as soon as possible.



FOR MY

PATIENTS

2/1988

KNOW YOUR CHOLESTEROL

Dear Patients

Among men 35 to 74 years of age about 28 have high levels of cholesterol Among women in the same age group about 32 have high levels according to a recent national survey

Cholesterol is a fat like chemical substance found in the blood and tissues of many organs Most of the body s cholesterol is made in the liver but a large portion is obtained from the food entering the stomach Eating fatty foods such as butter ice cream and meats tends to raise the level of cholesterol in the blood Although some people inherit a tendency to develop higher levels than others diet is an important factor

The total amount of cholesterol circulating in the blood contains both good cholesterol (HDL) and bad cholesterol (LDL) Therefore the aim is to raise the HDL part and lower the LDL part

DOING ITS DAMAGE

High cholesterol in the blood gradually forms arteriosclerotic deposits (plaques) along the inside walls of the arteries (hardening of the arteries) As time goes on these arteries become narrowed by the buildup of these plaques Circulation to the vital organs of the body becomes blocked The next event can be damage to the heart (coronary disease or heart attack) or to the brain (cerebrovascular accident or stroke)

KNOW YOUR RISK FACTORS

If you have **two or more** of these risk factors

- ☐ smoking
- ☐ high cholesterol
- ☐ high blood pressure
- ☐ overweight
- ☐ diabetes
- ☐ family history of early heart attacks

your chances of developing these heart and brain conditions are increased 300+ or more The more of these risk factors you have the greater are your chances

REASON FOR TESTING

Your cholesterol level is a sensitive indicator or clue to the amount of arteriosclerosis built up in your arteries If it is too high it is a danger signal that you may be on your way to coronary heart disease or stroke Steps can then be taken immediately to reduce this high level and prevent these damages If your first cholesterol level is 200 or greater it is a good idea to repeat the test at least twice more over the next couple of weeks at the same laboratory By taking the average of all 3 readings you can more accurately find your true level

WATCH THESE NUMBERS

The **desirable levels are less than 200** (milligrams per deciliter) If you are in these levels recheck it every 5 years at least

The **borderline high levels are 200-239** If you are in these levels recheck it each year If you are in these levels and have any other risk factors listed above consult your doctor as soon as possible Special low fat diet exercise program or drugs may be recommended for you

The **high levels are over 240** If you are in these levels you may need other blood tests to see how much of your total cholesterol is of the good type (HDL) and how much is of the bad type (LDL) Consult your doctor immediately so that special low fat diet exercise program or drugs may be prescribed for you The level should be re checked frequently

The goal for most adults is to bring their cholesterol level down to 200 or less and keep it there continuously

BENEFIT OF LOWERING THE LEVEL

The National Institute of Health has stated that for every 25• drop in blood cholesterol level there will be a 50• cut in the chances of having a heart attack The moral of the story is that checking your blood cholesterol is like taking the pulse of your own life

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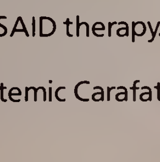
Aspirin and other nonsteroidal anti-inflammatory drugs are known to weaken mucosal defenses by inhibiting prostaglandin production. As a result, NSAID users may be more prone to duodenal ulcers.[1] For these patients, CARAFATE® (sucralfate/Marion) is first-line therapy that won't knuckle  under. Carafate rebuilds mucosal defenses, including endogenous prostaglandins, through a unique, nonsystemic mode of action. Carafate works by enhancing the body's natural healing ability while it protects damaged mucosa from further injury. Carafate offers a local healing approach to a local problem, allowing patients to continue their NSAID therapy. So the next time you see an arthritis patient with a duodenal ulcer, prescribe nonsystemic Carafate: therapy for the ulcer-prone patient.

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Please see brief summary of prescribing information and reference on adjacent page

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BRIEF SUMMARY

CONTRAINDICATIONS
There are no known contraindications to the use of sucralfate.

PRECAUTIONS
Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that simultaneous administration of CARAFATE (sucralfate) with tetracycline, phenytoin, digoxin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The clinical significance of these animal studies is yet to be defined. However, because of the potential of CARAFATE to alter the absorption of some drugs from the gastrointestinal tract, the separate administration of CARAFATE from that of other agents should be considered when alterations in bioavailability are felt to be critical for concomitantly administered drugs.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). There was no evidence of drug-related tumorigenicity. A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies were not conducted.

Pregnancy: Teratogenic effects. Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4.7%).

Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

OVERDOSAGE
There is no experience in humans with overdosage. Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose. Risks associated with overdosage should, therefore, be minimal.

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED
CARAFATE (sucralfate) 1-gm tablets are supplied in bottles of 100 (NDC 0088-1712-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1712-49). Light pink scored oblong tablets are embossed with CARAFATE on one side and 1712 bracketed by M's on the other.

Issued 1/87

Reference:
1. Eliakim R, Ophir M, Rachmilewitz D. *J Clin Gastroenterol* 1987; 9(4):395-399.

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(*continued from p 4A*)

grams in Medical Education, Room 569-U, University of California School of Medicine, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Sept 24–25. **Second Annual Aging Symposium: 1988 Drug Therapy and Nutrition in the Elderly.** 15 Cat I Credits. Cole Hall, University of California, San Francisco. Contact: Continuing Education S/P, University of California, Box 0446, San Francisco, CA 94143. Tel: (415) 476-4194.

Sept 26–30. **14th Annual Meeting of the American Association of Pathologists' Assistants (AAPA).** Sheraton-Fisherman's Wharf, San Francisco. Contact: Anne Walsh-Feeks, Department of Pathology, New York University Medical Center, 560 First Ave, New York, NY 10016. Tel: (212) 340-5747.

LOUISIANA

Sept 24–25. **1988 Regional Review Courses in Hand Surgery.** 12 Cat I Credits. New Orleans. Contact: American Society for Surgery of the Hand, 3025 South Parker Rd, Suite 65, Aurora, CO 80014. Tel: (303) 755-4588.

MARYLAND

Sept 7–9. **Prenatal Abuse of Licit and Illicit Drugs.** Hyatt Regency, Bethesda. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

NEBRASKA

Sept 19–24. **Emergency Medicine Review.** Omaha. Contact: Center for Continuing Education, University of Nebraska Medical Center, 42nd and Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

SOUTH CAROLINA

Sept 25–28. **Postgraduate Course in Uroradiology.** 21 Cat I Credits. Inter-Continental Hotel, Hilton Head. *Sept 29–Oct 1.* **Fall Update in Geriatrics.** 26 Cat I Credits. Omni Hotel, Charleston Place, Charleston. Contact: R. Dawne Ryals, Ryals and Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

TEXAS

Sept 29–30. **Issues and Concerns Facing IRBs and Clinical Investigators,** Doubletree Hotel, Austin. Contact: Office of Continuing Medical Education, Scott and White, 2401 South 31st St, Temple, TX 76508. Tel: (817) 774-2350.

WASHINGTON

Sept 17–24. **Behavioral Medicine for the Family Practitioner.** San Juan Islands, Washington. Contact: Kathy Rairigh, Epic Expeditions, PO Box 209, Sun Valley, ID 83353. Tel: (208) 788-4995.

WEST VIRGINIA

Sept 9–11. **New Antibiotics for Life Threatening Infections.** *Sept 16–18.* **Interventional Radiology.** Timberline Four Seasons Resort, Davis. Contact: Frederick A. Reichle, MD, Presbyterian-University of Pennsylvania Medical Center, 39th and Market Sts, Philadelphia, PA 19104. Tel: (215) 662-1715.

AROUND THE WORLD

ENGLAND

Sept 11–13. **Fourth Annual Fall Ultrasound Symposia.** Gloucester Hotel, London. Contact: Medical Seminars International, Inc, 21915 Roscoe Blvd, Suite 222, Canoga Park, CA 91304. Tel: (818) 719-7380.

FRANCE

Sept 15–17. **Fourth Annual Fall Ultrasound Symposia.** Grand Hotel, Paris. Contact: Medical Seminars International, Inc, 21915 Roscoe Blvd, Suite 222, Canoga Park, CA 91304. Tel: (818) 719-7380.

MONACO

Sept 14–19. **Cosmetic Surgery of the Face and Breast.** 18 Cat I Credits. Convention Centre, Monte Carlo. Contact: Francine Leinhardt, Conference Coordinator, 210 East 64th St, New York, NY 10021. Tel: (212) 838-9200, ext 2776.

SWEDEN

Sept 19–23. **Fifth International Conference on Human Tumor Markers.** Stockholm. Contact: G.D. Birkmayer, Doz Dr, Schwarzspanierstrasse 15 A-1090 Vienna, Austria. □

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Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

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1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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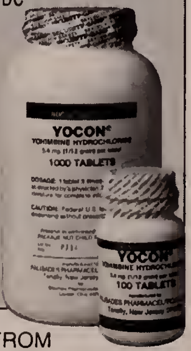

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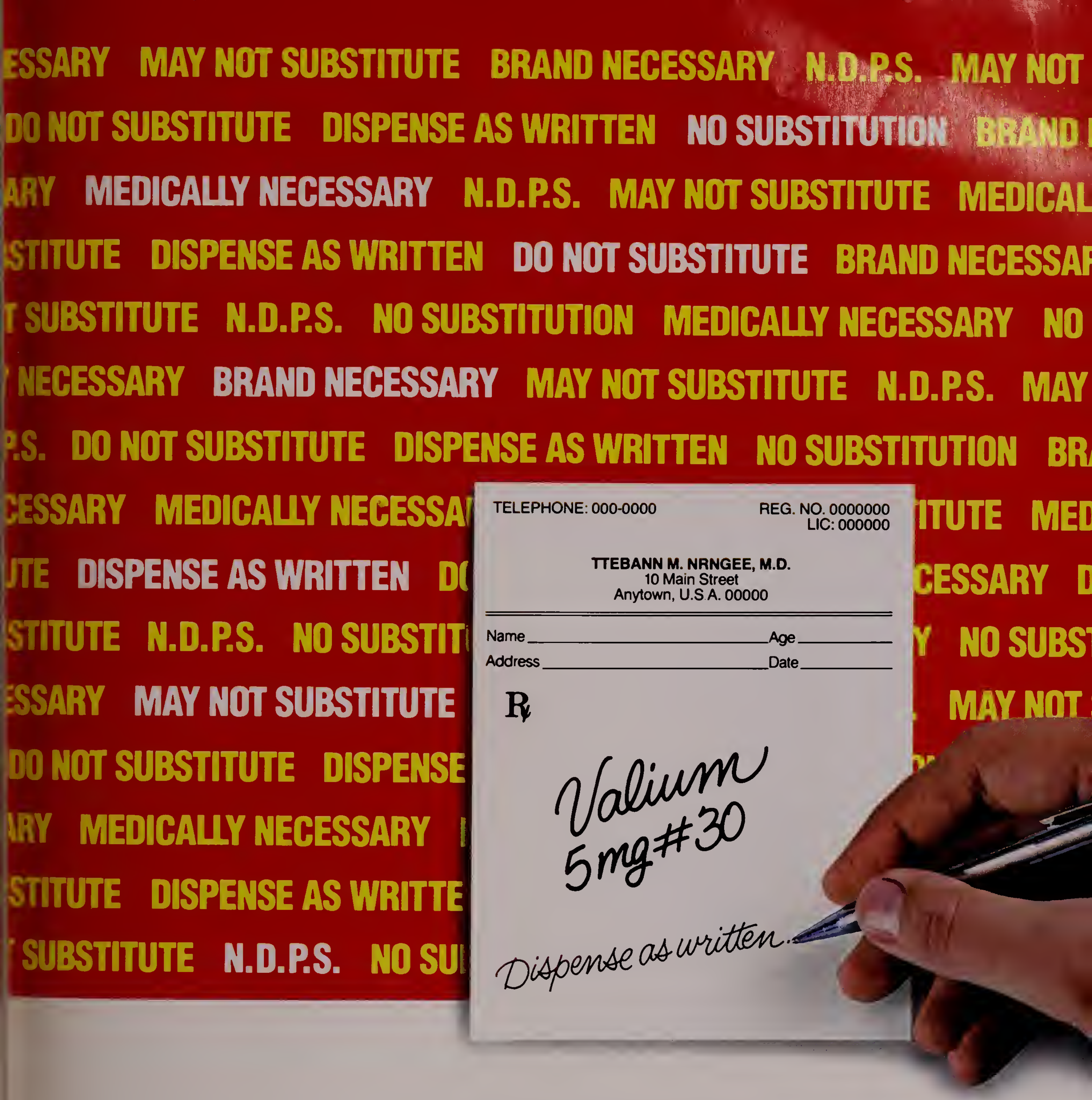
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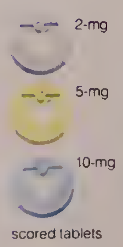

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Oct 22. **Fifth Annual Conference: Recent Advances in Anesthesia.** Desmond Americana Inn. *Oct 27.* **Biologic Response Modifiers and Cancer.** Albany Medical College. Contact: Diane M. Martin, Conference Coordinator, Albany Medical College, Office of Continuing Medical Education, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

BRONX

Oct 26. **The Medical Malpractice Crisis.** Lehman College. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 130 Gold Pl, Malverne, NY 11565. Tel: (516) 887-4691.

COOPERSTOWN

Oct 12. **Focus on Family Medicine.** 3 Cat I Credits. Bassett Hall Conference Center. Contact: Charlotte Hoag, Medical Education, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326. Tel: (607) 547-3926.

MANHATTAN

Oct 6–8. **MRI—Clinical State of the Art.** 18 Cat I Credits. NYU Medical Center. *Oct 17–19.* **The Second Conference on DNA Topoisomerases in Cancer Chemotherapy.** 12½ Cat I Credits. Alumni Hall, New York University Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Oct 7. **Effective Clinical Laboratory Utilization—Second Annual Course.**

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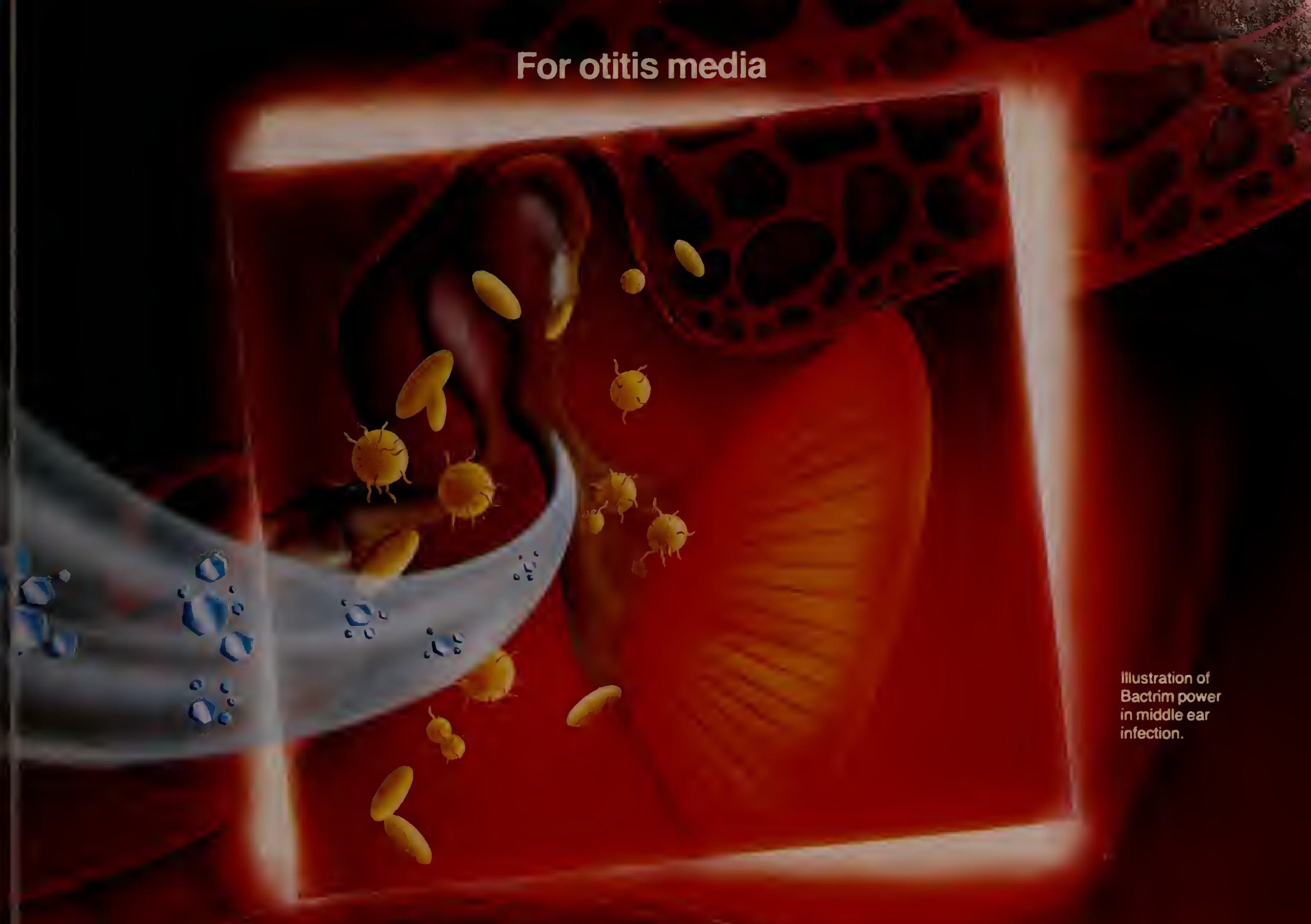

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BACTRIM SHOULD NOT BE USED IN THE TREATMENT OF STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A ß-hemolytic streptococcal tonsillopharyngitis have a greater incidence of bacteriologic failure when treated with Bactrim than with penicillin.
PRECAUTIONS: *General:* Give with caution to patients with impaired renal or hepatic function, possible folate deficiency (*e.g.*, elderly, chronic alcoholics, patients on anticonvulsants, with malabsorption syndrome, or in malnutrition states) and severe allergies or bronchial asthma. In glucose-6-phosphate dehydrogenase deficient individuals, hemolysis may occur, frequently dose-related.
Use in the Elderly: May be increased risk of severe adverse reactions in elderly, particularly with complicating conditions, *e.g.*, impaired kidney and/or liver function, concomitant use of other drugs. Severe skin reactions, generalized bone marrow suppression (see WARNINGS and ADVERSE REACTIONS) or a specific decrease in platelets (with or without purpura) are most frequently reported severe adverse reactions in elderly. In those concurrently receiving certain diuretics, primarily thiazides, increased incidence of thrombocytopenia with purpura reported. Make appropriate dosage adjustments for patients with impaired kidney function (see DOSAGE AND ADMINISTRATION).
Use in the Treatment of Pneumocystis Carinii Pneumonia in Patients with Acquired Immunodeficiency Syndrome (AIDS): AIDS patients may not tolerate or respond to Bactrim in same manner as non-AIDS patients. Incidence of side effects, particularly rash, fever, leukopenia, elevated aminotransferase (transaminase) values, with Bactrim in AIDS patients treated for *Pneumocystis carinii* pneumonia reported to be greatly increased compared with incidence normally associated with Bactrim in non-AIDS patients.
Information for Patients: Instruct patients to maintain adequate fluid intake to prevent crystalluria and stone formation.
Laboratory Tests. Perform complete blood counts frequently; if a significant reduction in the count of any formed blood element is noted, discontinue Bactrim. Perform urinalyses with careful microscopic examination and renal function tests during therapy, particularly for patients with impaired renal function.
Drug Interactions: In elderly patients concurrently receiving certain diuretics, primarily thiazides, an increased incidence of thrombocytopenia with purpura has been reported. Bactrim may prolong the prothrombin time in patients who are receiving the anticoagulant warfarin. Keep this in mind when Bactrim is given to patients already on anticoagulant therapy and reassess coagulation time. Bactrim may inhibit the hepatic metabolism of phenytoin. Given at a common clinical dosage, it increased the phenytoin half-life by 39% and decreased the phenytoin metabolic clearance rate by 27%. When giving these drugs concurrently, be alert for possible excessive phenytoin effect. Sulfonamides can displace methotrexate from plasma protein binding sites, thus increasing free methotrexate concentrations.
Drug/Laboratory Test Interactions: Bactrim, specifically the trimethoprim component, can interfere with a serum methotrexate assay as determined by the competitive binding protein technique (CBPA) when a bacterial dihydrofolate reductase is used as the binding protein. No interference occurs if methotrexate is measured by a radioimmunoassay (RIA). The presence of trimethoprim and sulfamethoxazole may also interfere with the Jaffé alkaline picrate reaction assay for creatinine, resulting in overestimations of about 10% in the range of normal values.
Carcinogenesis, Mutagenesis, Impairment of Fertility: Carcinogenesis. Long-term studies in animals to evaluate carcinogenic potential not conducted with Bactrim. *Mutagenesis:* Bacterial mutagenic studies not performed with sulfamethoxazole and trimethoprim in combination. Trimethoprim demonstrated to be nonmutagenic in the Ames assay. No chromosomal damage observed in human leukocytes *in vitro* with sulfamethoxazole and trimethoprim alone or in combination; concentrations used exceeded blood levels of these compounds following therapy with Bactrim. Observations of leukocytes obtained from patients treated with Bactrim revealed no chromosomal abnormalities. *Impairment of Fertility.* No adverse effects on fertility or general reproductive performance observed in rats given oral dosages as high as 70 mg/kg/day trimethoprim plus 350 mg/kg/day sulfamethoxazole.
Pregnancy: Teratogenic Effects: Pregnancy Category C. Trimethoprim and sulfamethoxazole may interfere with folic acid metabolism; use during pregnancy only if potential benefit justifies potential risk to fetus. Nonteratogenic Effects: See CONTRAINDICATIONS section.
Nursing Mothers: See CONTRAINDICATIONS section.
Pediatric Use. Not recommended for infants under two months (see INDICATIONS and CONTRAINDICATIONS sections)
ADVERSE REACTIONS: Most common are gastrointestinal disturbances (nausea, vomiting, anorexia) and allergic skin reactions (such as rash and urticaria). **FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS (SEE WARNINGS SECTION)**
Hematologic: Agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, neutropenia, hemolytic anemia, megaloblastic anemia, hypoprothrombinemia, methemoglobinemia, eosinophilia. *Allergic Reactions:* Stevens-Johnson syndrome, toxic epidermal necrolysis, anaphylaxis, allergic myocarditis, erythema multiforme, exfoliative dermatitis, angioedema, drug fever, chills, Henoch-Schoenlein purpura, serum sickness-like syndrome, generalized allergic reactions, generalized skin eruptions, photosensitivity, conjunctival and scleral injection, pruritus, urticaria and rash. Periarteritis nodosa and systemic lupus erythematosus have been reported. *Gastrointestinal:* Hepatitis (including cholestatic jaundice and hepatic necrosis), elevation of serum transaminase and bilirubin, pseudomembranous enterocolitis, pancreatitis, stomatitis, glossitis, nausea, emesis, abdominal pain, diarrhea, anorexia. *Genitourinary:* Renal failure, interstitial nephritis, BUN and serum creatinine elevation, toxic nephrosis with oliguria and anuria, crystalluria. *Neurologic:* Aseptic meningitis, convulsions, peripheral neuritis, ataxia, vertigo, tinnitus, headache. *Psychiatric:* Hallucinations, depression, apathy, nervousness. *Endocrine.* Sulfonamides bear certain chemical similarities to some goitrogens, diuretics (acetazolamide and the thiazides) and oral hypoglycemic agents; cross-sensitivity may exist. Diuresis and hypoglycemia have occurred rarely in patients receiving sulfonamides. *Respiratory:* Pulmonary infiltrates. *Musculoskeletal:* Arthralgia, myalgia. *Miscellaneous.* Weakness, fatigue, insomnia.
DOSAGE AND ADMINISTRATION: Not recommended for use in infants less than two months of age.
URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN. *Usual adult dosage* for urinary tract infections is one DS tablet, two tablets or four teaspoonfuls (20 ml) *b.i.d.* for 10 to 14 days. Use identical daily dosage for 5 days for shigellosis. *Recommended dosage for children* with urinary tract infections or acute otitis media is B mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses every 12 hours for 10 days. Use identical daily dosage for 5 days for shigellosis. *Renal Impaired:* Creatinine clearance above 30 ml/min, give usual dosage; 15-30 ml/min, give one-half the usual regimen; below 15 ml/min, use not recommended.
ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS: Usual adult dosage is one DS tablet, two tablets or four teasp. (20 ml) *b.i.d.* for 14 days.
PNEUMOCYSTIS CARINII PNEUMONIA. Recommended dosage is 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.
HOW SUPPLIED: *DS (double strength) Tablets* (160 mg trimethoprim and B00 mg sulfamethoxazole)—bottles of 100, 250 and 500, Tel-E-Dose® packages of 100; Prescription Paks of 20. *Tablets* (B0 mg trimethoprim and 400 mg sulfamethoxazole)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension* (40 mg trimethoprim and 200 mg sulfamethoxazole per teasp.)—bottles of 100 ml and 16 oz (1 pint). *Suspension* (40 mg trimethoprim and 200 mg sulfamethoxazole per teasp.)—bottles of 16 oz (1 pint).
STORE TABLETS AT 15°-30°C (59°-B6°F) IN A DRY PLACE PROTECTED FROM LIGHT. STORE SUSPENSIONS AT 15°-30°C (59°-86°F) PROTECTED FROM LIGHT.

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THEO-DUR®

THEOPHYLLINE (Anhydrous)
Sustained Action Tablets

INDICATIONS: THEO-DUR is indicated for relief and/or prevention of symptoms of asthma and for reversible bronchospasm associated with chronic bronchitis and emphysema
CONTRAINDICATIONS: THEO-DUR is contraindicated in individuals who have shown hypersensitivity to theophylline or any of the tablet components.
WARNINGS: Status asthmaticus should be considered a medical emergency and is defined as that degree of bronchospasm which is not rapidly responsive to usual doses of conventional bronchodilators Optimal therapy for such patients frequently requires both *additional medication*, parenterally administered, and *close monitoring*, preferably in an intensive care setting

Although increasing the dose of theophylline may bring about relief, such treatment may be associated with toxicity The likelihood of such toxicity developing increases significantly when the serum theophylline concentration exceeds 20 mcg/ml Therefore, determination of serum theophylline levels is recommended to assure maximal benefit without excessive risk.

Serum levels above 20 mcg/ml are rarely found after appropriate administration of recommended doses. However, in individuals in whom theophylline plasma clearance is reduced *for any reason*, even conventional doses may result in increased serum levels and potential toxicity Reduced theophylline clearance has been documented in the following readily identifiable groups. 1) patients with impaired renal or liver function, 2) patients over 55 years of age, particularly males and those with chronic lung disease, 3) those with cardiac failure from any cause; 4) neonates, and 5) those patients taking certain drugs (macrolide antibiotics and cimetidine). Decreased clearance of theophylline may be associated with either influenza immunization or active infection with influenza

Reduction of dosage and laboratory monitoring is especially appropriate in the above individuals. Less serious signs of theophylline toxicity (i e nausea and restlessness) may occur frequently when initiating therapy, but are usually transient, when such signs are persistent during maintenance therapy, they are often associated with serum concentrations above 20 mcg/ml Unfortunately, however, serious side effects such as ventricular arrhythmias, convulsions or even death may appear as the first sign of toxicity without any previous warning Stated differently: *serious toxicity is not reliably preceded by less severe side effects*

Many patients who require theophylline may exhibit tachycardia due to their underlying disease process so that the cause/effect relationship to elevated serum theophylline concentrations may not be appreciated

Theophylline products may cause dysrhythmia and/or worsen pre-existing arrhythmias and any significant change in rate and/or rhythm warrants monitoring and further investigation.

The occurrence of arrhythmias and sudden death (with histological evidence of necrosis of the myocardium) has been recorded in laboratory animals (minipigs, rodents and dogs) when theophylline and beta agonists were administered concomitantly, although not when either was administered alone The significance of these findings when applied to human usage is currently unknown
PRECAUTIONS: THEO-DUR TABLETS SHOULD NOT BE CHEWED OR CRUSHED
General: Theophylline half-life is shorter in smokers than in non-smokers Therefore, smokers may require larger or more frequent doses Morphine and curare should be used with caution in patients with airway obstruction as they may suppress respiration and stimulate histamine release Alternative drugs should be used when possible. Theophylline should not be administered concurrently with other xanthine medications. Use with caution in patients with severe cardiac disease, severe hypoxemia, hypertension, hyperthyroidism, acute myocardial injury, cor pulmonale, congestive heart failure, liver disease, in the elderly (especially males) and in neonates. In particular, great caution should be used in giving theophylline to patients with congestive heart failure. Frequently, such patients have markedly prolonged theophylline serum levels with theophylline persisting in serum for long periods following discontinuation of the drug Individuals who are rapid metabolizers of theophylline, such as the young, smokers, and some non-smoking adults, may not be suitable candidates for once-daily dosing These individuals will generally need to be dosed at 12 hour or sometimes 8 hour intervals. Such patients may exhibit symptoms of bronchospasm near the end of a dosing interval, or may have wider peak-to-trough differences than desired

Use theophylline cautiously in patients with history of peptic ulcer. Theophylline may occasionally act as a local irritant to the G.I. tract although gastrointestinal symptoms are more commonly centrally mediated and associated with serum drug concentrations over 20 mcg/ml
Information for Patients: The physician should reinforce the importance of taking only the prescribed dose and time interval between doses THEO-DUR tablets should not be chewed or crushed. When dosing THEO-DUR on a once daily (q24h) basis, tablets should be taken whole and not split. As with any controlled-release theophylline product, the patient should alert the physician if symptoms occur repeatedly, especially near the end of the dosing interval.
DRUG INTERACTIONS: **Drug-Drug**: Toxic synergism with ephedrine has been documented and may occur with some other sympathomimetic bronchodilators In addition, the following drug interactions have been demonstrated.

Drug	**Effect**
Theophylline with lithium carbonate	Increased excretion of lithium carbonate
Theophylline with propranolol	Antagonism of propranolol effect
Theophylline with cimetidine	Increased theophylline blood levels
Theophylline with troleandomycin, erythromycin	Increased theophylline blood levels

Drug-Food: *THEO-DUR 100 mg Sustained Action Tablets* have not been adequately studied to determine whether their bioavailability is altered when given with food Available data suggest that drug administration at the time of food ingestion may influence the absorption characteristics of theophylline controlled-release products resulting in serum values different from those found after administration in the fasting state.

A drug-food effect, if any, would likely have its greatest clinical significance when high theophylline serum levels are being maintained and/or when large single doses (greater than 13 mg/kg or 900 mg) of a controlled-release theophylline product are given

THEO-DUR (200, 300, and 450 mg) Sustained Action Tablets The rate and extent of absorption of theophylline from THEO-DUR 200 mg, 300 mg, and 450 mg tablets when administered fasting or immediately after a moderately high fat content breakfast is similar
Drug-Laboratory Test Interactions: When plasma levels of theophylline are measured by spectrophotometric methods, coffee, tea, cola beverages, chocolate, and acetaminophen contribute falsely high values.
Carcinogenesis, Mutagenesis, and Impairment of Fertility: Long-term animal studies have not been performed to evaluate the carcinogenic potential, mutagenic potential, or the effect on fertility of xanthine compounds
Pregnancy: Category C—Animal reproduction studies have not been conducted with theophylline It is not known whether theophylline can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Xanthines should be given to a pregnant woman only if clearly needed
Nursing Mothers: It has been reported that theophylline distributes readily into breast milk and may cause adverse effects in the infant Caution must be used if prescribing xanthine to a mother who is nursing, taking into account the risk-benefit of this therapy
Pediatric Use: Safety and effectiveness of THEO-DUR administered:
1 Every 24 hours in children under 12 years of age, have not been established
2 Every 12 hours in children under 6 years of age, have not been established
ADVERSE REACTIONS: The most consistent adverse reactions are usually due to overdose and are
1 *Gastrointestinal* nausea, vomiting, epigastric pain, hematemesis, diarrhea
2 *Central nervous system* headaches, irritability, restlessness, insomnia, reflex hyperexcitability, muscle twitching, clonic and tonic generalized convulsions
3 *Cardiovascular* palpitation, tachycardia, extrasystoles, flushing, hypotension, circulatory failure, ventricular arrhythmias
4 *Respiratory* tachypnea
5 *Renal* albuminuria, increased excretion of renal tubular and red blood cells, potentiation of diuresis.
6 *Other* rash hyperglycemia and inappropriate ADH syndrome
OVERDOSAGE. **Management**. If potential oral overdose is established and seizure has not occurred
A Induce vomiting
B Administer a cathartic (this is particularly important if sustained-release preparations have been taken).
C Administer activated charcoal
If patient is having a seizure
A Establish an airway
B Administer oxygen
C Treat the seizure with intravenous diazepam, 0 1 to 0 3 mg/kg up to 10 mg
D Monitor vital signs [illegible] blood pressure and provide adequate hydration
Post Seizure Coma.
A Maintain airway and [illegible]
B If a result of oral medication, follow above recommendations to prevent absorption of the drug, but intubation and lavage will have to be performed instead of inducing emesis, and the cathartic and charcoal will need to be introduced via a large bore gastric lavage tube
C Continue to provide full supportive care and adequate hydration while waiting for drug to be metabolized. In general, the drug is metabolized sufficiently rapid so as not to warrant consideration of dialysis, however, if serum levels exceed 50 mcg/ml charcoal hemoperfusion may be indicated
CAUTION: Federal law prohibits dispensing without prescription For full prescribing information, see package insert
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AXID®
nizatidine capsules

Brief Summary. Consult the package insert for prescribing information.

Indications and Usage: Axid is indicated for up to eight weeks for the treatment of active duodenal ulcer. In most patients, the ulcer will heal within four weeks.

Axid is indicated for maintenance therapy for duodenal ulcer patients, at a reduced dosage of 150 mg h.s. after healing of an active duodenal ulcer. The consequences of continuous therapy with Axid for longer than one year are not known.

Contraindication: Axid is contraindicated in patients with known hypersensitivity to the drug and should be used with caution in patients with hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Because nizatidine is excreted primarily by the kidney, dosage should be reduced in patients with moderate to severe renal insufficiency.

3. Pharmacokinetic studies in patients with hepatorenal syndrome have not been done. Part of the dose of nizatidine is metabolized in the liver. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests—False-positive tests for urobilinogen with Multistix® may occur during therapy with nizatidine.

Drug Interactions—No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high dose males compared to placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the lower dose groups. The rate of hepatic carcinoma in the high dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement compared to concurrent controls, and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive, and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery is not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, and the mouse lymphoma assay.

In a two-generation, perinatal and postnatal, fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose, and in Dutch Belted rabbits at doses up to 55 times the human dose, revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Nizatidine is secreted and concentrated in the milk of lactating rats. Pups reared by treated lactating rats had depressed growth rates. Although no studies have been conducted in lactating women, nizatidine is assumed to be secreted in human milk, and caution should be exercised when nizatidine is administered to nursing mothers.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Ulcer healing rates in elderly patients are similar to those in younger age groups. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations. Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo. Among the more common adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common in the nizatidine group. A variety of less common events was also reported; it was not possible to determine whether these were caused by nizatidine.

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Hepatic—Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients possibly or probably related to nizatidine. In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L), and in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid. Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo. Rare reports of gynecomastia occurred.

Hematologic—Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist. On previous occasions, this patient had experienced thrombocytopenia while taking other drugs.

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine than in placebo patients. Rash and exfoliative dermatitis were also reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported.

Overdosage: There is little clinical experience with overdosage of Axid in humans. If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea. Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal. Intravenous LD_{50} values in the rat and mouse were 301 mg/kg and 232 mg/kg respectively.

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COMMENTARIES

Blood: Whose choice and whose conscience?

Physicians are committed to applying their knowledge, skills, and experience in fighting disease and death. Yet, what if a patient refuses a recommended treatment? This will likely occur if the patient is a Jehovah's Witness and the treatment is whole blood, packed red blood cells, plasma, or platelets.

When it comes to the use of blood, a physician may feel that a patient's choice of nonblood treatment will tie the hands of dedicated medical personnel. Still, one must not forget that patients other than Jehovah's Witnesses often choose not to follow their doctor's recommendations. According to Appelbaum and Roth,[1] 19% of patients at teaching hospitals refused at least one treatment or procedure, even though 15% of such refusals "were potentially life endangering."

The general view that "the doctor knows best" causes most patients to defer to their doctor's skill and knowledge. But how subtly dangerous it would be for a physician to proceed as if this phrase were a scientific fact and to treat patients accordingly. True, our medical training, licensing, and experience give us noteworthy *privileges* in the medical arena. Our patients, though, have *rights*. And, as we are likely aware, the law (even the Constitution) gives greater weight to rights.

On the walls of most hospitals, one sees displayed the "Patient's Bill of Rights." One of these rights is informed consent, which might more accurately be called informed choice. After the patient is informed of the potential results of various treatments (or of nontreatment), it is his choice what he will submit to. At Albert Einstein Hospital in the Bronx, New York, a draft policy on blood transfusion and Jehovah's Witnesses stated: "Any adult patient who is not incapacitated has the right to refuse treatment no matter how detrimental such a refusal may be to his health."[2]

While physicians may voice concerns about ethics or liability, courts have stressed the supremacy of patient choice.[3] The New York Court of Appeals stated that "the patient's right to determine the course of his own treatment [is] paramount . . . [A] doctor cannot be held to have violated his legal or professional responsibilities when he honors the right of a competent adult patient to decline medical treatment."[4] That court has also observed that "the ethical integrity of the medical profession, while important, cannot outweigh the fundamental individual rights here asserted. It is the needs and desires of the individual, not the requirements of the institution, that are paramount."[5]

When a Witness refuses blood, physicians may feel pangs of conscience at the prospect of doing what seems to be less than the maximum. What the Witness is asking conscientious doctors to do, though, is to provide the best alternative care possible under the circumstances. We often must alter our therapy to accommodate circumstances, such as hypertension, severe allergy to antibiotics, or the unavailability of certain costly equipment. With the Witness patient, physicians are being asked to manage the medical or surgical problem in harmony with the patient's choice and conscience, his moral/religious decision to abstain from blood.

Numerous reports of major surgery on Witness patients show that many physicians can, in good conscience and with success, accommodate the request not to employ blood. For example, in 1981, Cooley reviewed 1,026 cardiovascular operations, 22% on minors. He determined "that the risk of surgery in patients of the Jehovah's Witness group has not been substantially higher than for others."[6] Kambouris[7] reported on major operations on Witnesses, some of whom had been "denied urgently needed surgical treatment because of their refusal to accept blood." He said: "All patients received pretreatment assurances that their religious beliefs would be respected, regardless of the circumstances in the operating room. There were no untoward effects of this policy."

When a patient is a Jehovah's Witness, beyond the matter of choice, conscience comes into the picture. One cannot think only of the physician's conscience. What of the patient's? Jehovah's Witnesses view life as God's gift represented by blood. They believe the Bible's command that Christians must "abstain from blood" (Acts 15:28, 29).[8] Hence, if a physician paternalistically violated such patients' deep and long-held religious convictions, the result could be tragic. Pope John Paul II has observed that forcing someone to violate his conscience "is the most painful blow inflicted to human dignity. In a certain sense, it is worse than inflicting physical death, or killing."[9]

While Jehovah's Witnesses refuse blood for religious reasons, more and more non-Witness patients are choosing to avoid blood because of risks such as AIDS, non-A non-B hepatitis, and immunologic reactions. We may present to them our views as to whether such risks seem minor compared to the benefits. But, as the American Medical Association points out, the patient is "the final arbiter as to whether he will take his chances with the treatment or operation recommended by the doctor or risk living without it. Such is the natural right of the individual, which the law recognizes."[10]

Related to this, Macklin[11] brought up the risk/benefit issue regarding a Witness "who risked bleeding to death without a transfusion." A medical student said: "His thought processes were intact. What do you do when religious beliefs are against the only source of treatment?" Macklin reasoned: "We may believe very strongly this man is making a mistake. But Jehovah's Witnesses believe that to be transfused . . . [may] result in eternal damnation. We are trained to do risk-benefit analyses in medicine but if you weigh eternal damnation against remaining life on earth, the analysis assumes a different angle."[11]

Vercillo and DuPrey[12] in this issue of the *Journal* refer to *In re Osborne* to highlight the interest in ensuring the security of dependents, but how was that case resolved? It concerned a severely injured father of two minor children. The court determined that if he died, relatives would materially and spiritually care for his children. So, as in other recent cases,[13] the court found no compelling state interest to justify overriding the patient's choice of treatment; judicial intervention to authorize treatment deeply objectionable to him was unwarranted.[14] With alternative treatment the patient recovered and continued to care for his family.

Is it not true that the vast majority of cases physicians have confronted, or likely will, can be managed without blood? What we studied and know best has to do with medical problems, yet patients are human beings whose individual values and goals cannot be ignored. They know best about their own priorities, their own morals and conscience, which give life meaning for them.

Respecting the religious consciences of Witness patients may challenge our skills. But as we meet this challenge, we underscore valuable liberties that all of us cherish. As John Stuart Mill aptly wrote: "No society in which these liberties are not, on the whole, respected, is free, whatever may be its form of government . . . Each is the proper guardian of his own health, whether bodily, or mental and spiritual. Mankind are greater gainers by suffering each other to live as seems good to themselves, than by compelling each to live as seems good to the rest."[15]

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Jehovah's Witnesses and the right to refuse blood

The article in this issue by Vercillo and Duprey[1] informs physicians about Jehovah's Witnesses and their religious beliefs. These authors state that Jehovah's Witnesses believe it is better to die than to accept blood transfusions, and though the Witnesses actively pursue the best of medical care, including "bloodless" major surgery, they refuse blood. This paper adds to a growing literature to help physicians understand the religious, moral, and legal foundations of the Witnesses' beliefs and rights to refuse blood transfusions.[2-18] This understanding is important, for whether or not a physician supports the Witnesses' beliefs, all physicians must understand their patient's religious credos concerning medicine. Without such understanding, physicians cannot respect or help protect their patients' religious and individual rights.

An informed and competent adult has the right to refuse medical treatment including blood transfusions. This right arises primarily from respect for self-determination and is enhanced by both religious and individual freedom. Religious liberty, the freedom to worship, and the right to practice religious beliefs are guaranteed in the Constitution of the United States. The right of the individual to pursue life, liberty, and happiness is stated in the Declaration of Independence. These freedoms are not absolute, for they allow an individual his religious and individual freedom unless such practices are specifically prohibited by law or result in harm to others. Vercillo and Duprey[1] point to instances in which Witnesses may not refuse blood, ie, where the Witness is a minor, is pregnant, is responsible for the sole care of children, or is not competent. The right of Jehovah's Witnesses to refuse blood brings controversy into medicine,[2] for the belief that death is preferable to accepting blood conflicts with the desire of many physicians to save life at all costs.

Certain guidelines may help avoid conflicts between Jehovah's Witnesses and their physicians. The patient has an obligation to inform his doctor early about his unwillingness to accept blood. Though most Witnesses inform their physicians of their beliefs, and many witnesses carry emergency information cards documenting their refusal to accept blood, previous studies have shown that some Witnesses do not inform their physicians of their beliefs.[12] This fact has prompted church leaders to suggest that Jehovah's Witnesses schedule an appointment with their physician to discuss their beliefs. This would allow ample time for patients to discuss important issues free from the stresses of acute and emergent illness. This early discussion would allow a physician to explore the reasons for and consequences of refusal of treatment. Such discussion is ethically necessary for an informed refusal of potentially lifesaving treatment. In addition, this discussion allows a physician to assess the capacity of the patient to make a decision to refuse treatment. Refusing lifesaving treatment does not indicate mental incapacity, but might reasonably prompt an assessment of the capacity to consent. In life threatening situations, this mental assessment is best done by a specialized professional (ie, a psychiatrist). Finally, early discussions allow the physician to disclose any reluctance to abide by the patient's wishes. If the patient's wishes are not compatible with the physician's ethical and professional values, he should arrange to turn over the care of the patient to another physician.

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Gynecologic cancer in Brooklyn, New York, 1978–1982

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ABSTRACT. To assess the need for cancer prevention efforts in Brooklyn, NY, the incidences of cancers of the cervix, corpus uteri, and ovary were computed for the ten health districts of Brooklyn using cancer cases for 1978–1982 and population data from the 1980 census. The incidence of invasive cancer of the cervix was higher than the national average in low income, predominantly minority districts. The incidence of carcinoma in situ of the cervix was below average, primarily because of low rates in districts with many immigrants. The incidence of corpus cancer in white women was below average in both Hispanic and non-Hispanic areas. The incidence of ovarian cancer was above average in the white populations of the Gravesend and Flatbush districts. The data suggest that preventive efforts, while substantial, should be intensified among the low income, immigrant, and older female populations of Brooklyn.

(NY State J Med 1988; 88:466–473)

In 1973, the New York State (NYS) Department of Health embarked on a new initiative requiring that all cancer cases in New York City (NYC) be registered with the NYS Cancer Registry, which had been registering cancer cases for the rest of the state since 1940.[1] By 1976, cancer registration in NYC was virtually complete and, with population data from the 1980 US Census, it became possible to calculate cancer incidence in NYC for the first time.[2]

The vast size of New York City or even of the borough of Brooklyn (with its 2.4 million population) makes citywide or boroughwide data of limited value to practitioners or institutions wishing to understand the nature and magnitude of the disease in the local communities they serve. The population of NYC is so large and diverse that the usual categories by which cancer data have been evaluated (age, sex, and race) are not always revealing. An age/race/sex group in one geographic area may be of a totally different socioeconomic status or national origin than the same age/race/sex group in another area. A multidimensional matrix of age, sex, race, socioeconomic status, national origin, language, and geographic area is often needed. Fortunately, cancer cases in each of the four large boroughs of NYC are registered to the local health district, which is the geographic area the NYC Department of Health uses to collect vital statistics. The ten health districts of Brooklyn are shown in Figure 1.

Obstetrician-gynecologists function both as primary care physicians and as specialists. For the past 30 years, early detection of cancer has been a primary function of the preventive care provided by gynecologists through

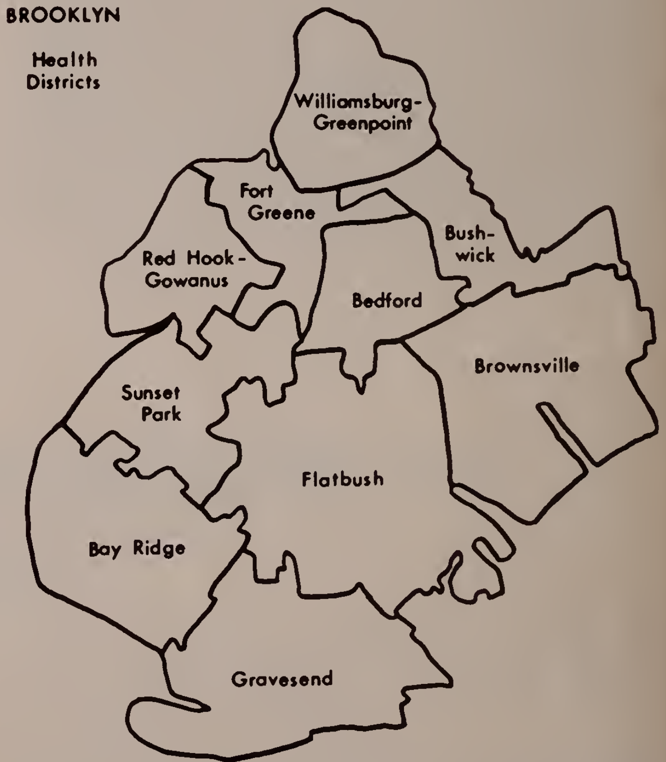

FIGURE 1. The ten health districts in Brooklyn. The names of the health districts do not necessarily correspond to the neighborhoods of the same name. Example: a part of the neighborhood of Bedford-Stuyvesant is actually in the Fort Greene health district, while the health district of Gravesend includes both the neighborhood of Gravesend in the west and the neighborhood of Coney Island in the south.

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routine check-ups and cervical cytology. This study, one of a series on cancer incidence and mortality in Brooklyn,[3,4] was designed to assess the characteristics of gynecologic cancer in the borough and to determine the need for further preventive efforts.

PATIENTS AND METHODS

Population Data. Information on the size of each age/race/sex group for the ten health districts of Brooklyn based on the 1980 census was obtained from the NYC Department of Health's Office of Epidemiologic Surveillance. Race was categorized as white, black, Hispanic (Spanish origin), and other. In most computations in this paper, Hispanic persons were included as white. Additional data on the socioeconomic and ethnic characteristics of the health districts derived from the 1980 census were obtained from the Columbia University Center for the Social Sciences. The age structures of the black, Hispanic, and non-Hispanic white populations are shown in Figure 2. Other demographic and socioeconomic characteristics of the female populations counted in the 1980 census of each health district are shown in Table I.

One limitation of the US Census data in large urban areas is the considerable degree of undercounting. Racial minorities and undocumented immigrants were less likely to have been counted than other populations.[5] Estimates of the uncounted population in NYC vary.[6-8] In Brooklyn, with large Afro-American and Hispanic populations, undercounting was significant. McCarthy[6] has estimated that black and Hispanic populations were 10% undercounted, with the major uncounted groups being immigrants from Latin America and the Caribbean. In this report, we have computed adjusted populations for the black (and Hispanic) women of the borough assuming that there was one uncounted immigrant for every two counted immigrants who entered the country between 1970 and 1980, and that the age distribution of the uncounted women was similar to that of the counted immigrants in 1970-1980. Brooklyn-wide cancer incidence rates are therefore reported as a range, with the high value using the counted population in the denominator and the low value using the adjusted population. Because of insufficient information about local immigrant populations, adjusted populations were not computed for the ten health districts.

Cancer Cases. Information on cancer among Brooklyn residents initially diagnosed in 1978-1982 was obtained from the New York State Cancer Registry. Cancer sites are coded using ICD-9 classifications as follows: cervix (invasive cancer only), ICD-9 180; cervix (carcinoma in situ), ICD-9 2331; corpus uteri, ICD-9 182 and 179; ovary, ICD-9 183.0. Each cancer case was identified by site, age, sex, race, and health district. Racial groups included white, black, Hispanic (Spanish origin), and others.

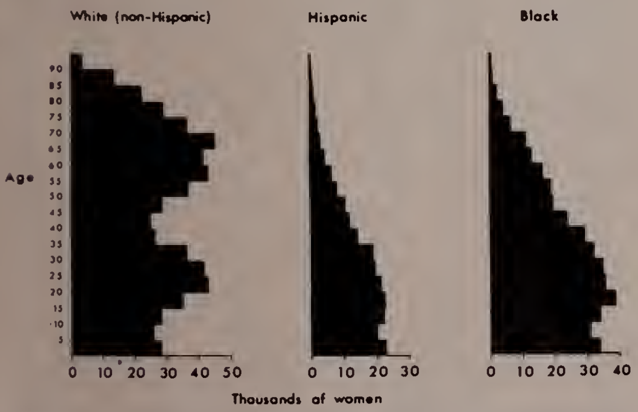

FIGURE 2. Age structure of the female population of Brooklyn by ethnic/race group (NYC Department of Health based on 1980 US Census).

TABLE I. Characteristics of the Health District Populations

Health District	N	Elderly % > 65	Education* % ≥ 12 yrs	Income* % > poverty	Hispanic (% of total)† > 20 yrs	> 40 yrs
White Females						
Bay Ridge	130,074	22	55	89	5	3
Bedford	11,334	15	59	71	56	45
Brownsville	66,051	13	55	84	31	22
Bushwick	59,245	10	33	67	65	48
Flatbush	160,606	21	65	88	12	7
Fort Greene	26,633	14	71	79	43	38
Gravesend	140,427	24	58	88	5	3
Red Hook/ Gowanus	45,334	14	61	72	33	30
Sunset Park	80,544	16	49	79	30	20
Williamsburg/ Greenpoint	66,984	12	38	68	42	32
Total	787,232	18	55	82	15	9

Health District	N	Elderly % > 65	Education* % ≥ 12 yrs	Income* % > poverty	% Foreign born*
Black Females					
Bay Ridge	527	7	76	69	N/A
Bedford	102,462	8	54	67	23
Brownsville	79,148	5	55	65	19
Bushwick	29,773	6	47	59	12
Flatbush	90,591	4	70	79	49
Fort Greene	51,351	7	53	62	14
Gravesend	10,903	5	54	65	N/A
Red Hook/ Gowanus	11,521	7	55	66	14
Sunset Park	2,149	4	61	70	N/A
Williamsburg/ Greenpoint	5,887	11	50	69	13
Total	384,312	6	57	68	26

All items refer to the female population only unless asterisked.

* Education: percentage of persons over age 24 with 12 or more years' education; income: percentage of families above the poverty level; foreign born: percentage of persons foreign born.

† Proportion of white females over 20 years and over 40 years who were Hispanic.

Statistical Methods—Age Standardized Incidence Rates. These rates were determined by the direct method. Age specific incidence rates were calculated for each health district/race group and standardized to the 1970 US standard population to allow comparison with the national data for 1978-1981 from the Surveillance, Epidemiology and End Results (SEER) program of the National Cancer Institute.[9] Where there were ten cases or less in a health district, the direct method was considered unreliable, so the incidence rate was calculated from the product of the Indirect Standardized Incidence Ratio and the standard SEER rate.

Indirect Standardized Incidence Ratios (ISIR). Age-specific incidence rates for black and white women from the national SEER population for 1978-1981[9] were applied to each health district/race group to determine the number of cases of cancer that would be expected in that population if national rates applied. The 95% confidence limits of the ratio of observed to expected cases (ISIR) were calculated from a Poisson distribution.[10]

RESULTS

Invasive Cancer of the Cervix. The incidence of invasive cancer of the cervix (ICC) in Brooklyn for both black women (27.4/100,000) and white women (10.3/100,000) was significantly higher than the corresponding SEER incidence. Brooklyn-wide, the incidence of ICC in Hispanic whites was 17.2/100,000, which was significantly high, in contrast to the incidence of 8.5/100,000 in non-Hispanic whites, which was similar to the SEER rate. Among black women, the incidence was above average in eight of the ten health districts, although it was significantly high in only three—Bedford, Flatbush, and Bushwick (Table II). Among white women the incidence varied four-fold in different health districts. In Bushwick, Fort Greene, Red Hook, and Williamsburg, where Hispanic females comprised one third or more of the white population over 20 years of age, the incidence of ICC was significantly higher than SEER rates for white women. In contrast, in Gravesend, where the Hispanic female population was small, the incidence of ICC was significantly lower (6.5/100,000) than the SEER rate. In Brownsville, Flatbush, and Bay Ridge, the incidences of ICC in non-Hispanic white women were 5.4, 7.3, and 9.0/100,000, respectively.

Although white women had more (56.3%) localized ICC than black women (50.7%), the difference in stage was not statistically significant, and there were no significant differences in stage between non-Hispanic and Hispanic white areas.

Carcinoma in Situ of the Cervix. The incidence of carcinoma in situ of the cervix (CIS) in Brooklyn was significantly lower than the SEER rate in both white and black populations. However, among black women the rate was significantly low in only one health district, Flatbush (Table III). Two health districts (Bedford and Bushwick) with significantly high rates of ICC did not have low rates of CIS. In the white population, the incidence of CIS was significantly lower than SEER in eight of the ten health districts, including three (Fort Greene, Red Hook, and Williamsburg) where the incidence of ICC was high, and one (Gravesend) where the incidence of ICC was low.

Among black women, the ratio of CIS to ICC ranged only from 1.2 to 1.9 (Table III). Among white women, the ratio of CIS to ICC was low in predominantly Hispanic districts, but was greater than two in non-Hispanic districts.

TABLE III. Carcinoma in Situ of the Cervix in Brooklyn 1978–1982: Incidence* by Health District and Race

	White Women			Black Women		
Health District	N	Incidence	CIS:ICC	N	Incidence	CIS:ICC
Bay Ridge	130	19.9†	2.2	2	63.2	1.4
Bedford	13	24.5	—	262	43.0	1.6
Brownsville	79	22.0†	2.5	177	39.7	1.6
Bushwick	81	24.7	1.2	85	54.9	1.6
Flatbush	139	16.9†	2.1	208	38.2†	1.3
Fort Greene	32	19.5†	0.8	118	41.5	1.9
Gravesend	107	15.8†	2.4	33	52.0	1.4
Red Hook/ Gowanus	54	20.4†	1.5	30	43.5	1.5
Sunset Park	99	23.5†	2.6	10	71.9	1.5
Williamsburg/ Greenpoint	62	17.9†	1.1	14	45.1	1.2
Total	796	19.3†	1.9	939	40.8†	1.5
SEER Rate (1977–1981)		29.3			46.1	

* Cases per 100,000 per year. Calculated by direct standardization and adjusted to 1970 US standard population. When there were ten cases or less in a health district, the incidence was computed as the product of the ISIR and the SEER rate.
N, number of cases in five years.
† Significantly lower than SEER ($p < 0.05$).

Cancer of the Corpus Uteri. Brooklyn-wide, the incidence of cancer of the corpus uteri was significantly below the national SEER incidence for white women (25.1/100,000), but was significantly above the SEER incidence for black women (13.4/100,000). Among white women the incidence was below the SEER rate in nine of the ten health districts, including those such as Bay Ridge with very few Hispanic women, and those such as Red Hook and Williamsburg with large numbers of Hispanic women (Table IV). Brooklyn-wide, the incidence of corpus cancer in Hispanic women was 9.8/100,000, and because of the youthful age structure of the Hispanic population there were only 60 cases in five years. The incidence in non-Hispanic white women was 21.0/100,000.

Significantly more white women (69.9%) than black women (59.1%) had corpus cancer diagnosed in the localized stage. There

TABLE II. Invasive Cancer of the Cervix in Brooklyn 1978–1982: Incidence* by Health District and Race

	White Women			Black Women		
Health District	N	Incidence	% Local Stage	N	Incidence	% Local Stage
Bay Ridge	72	9.1	57	2	89.9	100
Bedford	8	13.2	50	140	27.4†	52
Brownsville	32	8.7	72	74	24.4	53
Bushwick	52	20.7†	55	42	34.7†	55
Flatbush	82	8.1	55	97	30.1†	50
Fort Greene	34	25.7†	61	50	22.3	40
Gravesend	68	6.5‡	58	14	36.3	43
Red Hook/ Gowanus	36	13.6†	66	16	29.8	44
Sunset Park	39	9.0	59	4	49.3	100
Williamsburg/ Greenpoint	57	16.2†	42	11	36.2	60
Total	482	10.3†	56.3	450	27.4†	50.7
95% Confidence interval			51.9–60.7			46.1–55.3
SEER Rate (1977–1981)		8.8			20.2	

* Cases per 100,000 per year. Calculated by direct standardization and adjusted to 1970 US standard population. When there were ten cases or less in a health district, the incidence was computed as the product of the ISIR and the SEER rate.
N = number of cases in five years.
† Significantly higher than SEER ($p < 0.05$).
‡ Significantly lower than SEER ($p < 0.05$).

TABLE IV. Cancer of the Corpus Uteri in Brooklyn 1978–1982: Incidence* by Health District and Race

	White Women			Black Women		
Health District	N	Incidence	% Local Stage	N	Incidence	% Local Stage
Bay Ridge	200	18.8†	68	1	—	
Bedford	17	28.2	65	79	16.3	62
Brownsville	81	20.1†	73	44	17.5	71
Bushwick	61	23.1	79	21	20.9	43
Flatbush	274	21.3†	69	31	11.0	52
Fort Greene	19	13.6†	55	39	18.2	65
Gravesend	282	22.9†	70	7	20.4	57
Red Hook/ Gowanus	42	16.5†	80	7	15.0	29
Sunset Park	83	17.3†	73	0	—	—
Williamsburg/ Greenpoint	62	17.4†	65	1	10.5	—
Total	1121	20.1†	69.9	233	15.7‡	59.1
95% Confidence interval			67.2–72.6			52.8–65.4
SEER Rate (1977–1981)		25.1			13.4	

* Cases per 100,000 per year. Calculated by direct standardization and adjusted to 1970 US standard population. When there were ten cases or less in a health district, the incidence was computed as the product of the ISIR and the SEER rate.
N, number of cases in five years.
† Significantly lower than SEER ($p < 0.05$).
‡ Significantly higher than SEER ($p < 0.05$).

was no significant difference between Hispanic and non-Hispanic whites in stage at diagnosis.

The age-specific incidence of corpus cancer in Brooklyn women compared to SEER women is shown in Figure 3. In white Brooklyn women the incidence was lower and the peak occurred at older ages than among white SEER women. The peak incidence was at ages 65–69 years in both Hispanic and non-Hispanic white women. Compared to black women in SEER, black Brooklyn women had a higher incidence after age 60 and a peak incidence at a younger age.

Cancer of the Ovary. Brooklyn-wide, the incidence of cancer of the ovary in white women was significantly higher than in SEER, whereas the incidence in black women was similar to the SEER rate. Among white women (Table V), the areas of above-average incidence were primarily the non-Hispanic areas, with the incidence significantly high in Gravesend and Flatbush. In contrast, those areas with a high proportion of Hispanic women had below-average rates, with one area (Williamsburg) significantly low. There was no difference between white women and black women in the stage at diagnosis.

DISCUSSION

In common with most health data recorded in the United States, cancer cases are categorized by race, but not by income, education, or other measures of socioeconomic status. In Brooklyn, as nationally, black and Hispanic people usually have lower incomes and less education than non-Hispanic whites (Table I). For cancer of the cervix there is a strong inverse relationship between incidence and socioeconomic status, whereas for cancers of the corpus and ovary, there is a direct association.[11] In this report, therefore, ethnic/racial categories must often be read as surrogates for socioeconomic status.

A summary of the Brooklyn-wide incidence of gynecologic cancers together with comparison data from the SEER program for the national population is shown in Table VI. However, since the national SEER data includes affluent urban and suburban areas, we have also shown incidence data[12] for Wayne County, Michigan, which includes the city of Detroit, the most comparable northern, low-income, urban area for which data are available. SEER data from Puerto Rico are presented for comparison with the Brooklyn Hispanic population.

As in the national experience, the incidence of cervical cancer, both ICC and CIS, in relatively low-income Brook-

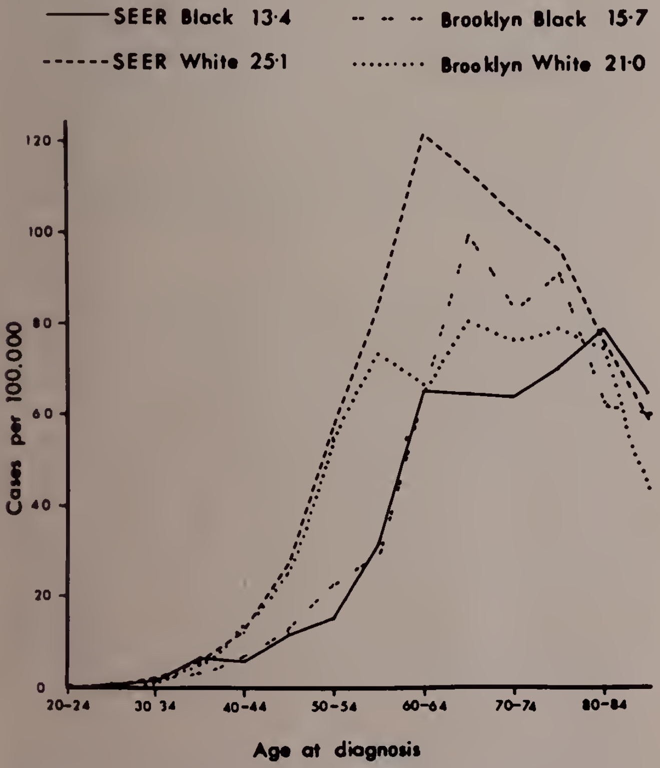

FIGURE 3. Age-specific incidence rates of corpus cancer by race (SEER rates 1977–1981; Brooklyn rates 1978–1982).

TABLE V. Cancer of the Ovary in Brooklyn 1978–1982: Incidence* by Health District and Race

	White Women			Black Women		
Health District	N	Incidence	% Local Stage	N	Incidence	% Local Stage
Bay Ridge	146	15.4	27	0	0	—
Bedford	4	6.5	50	45	9.5	26
Brownsville	55	14.4	35	17	6.6	29
Bushwick	29	11.8	28	14	12.0	31
Flatbush	193	17.4†	26	43	14.3†	30
Fort Greene	22	15.0	23	22	9.5	10
Gravesend	193	17.0†	16	5	13.5	0
Red Hook/ Gowanus	34	13.1	29	4	8.2	0
Sunset Park	67	13.8	24	2	24.1	50
Williamsburg/ Greenpoint	37	10.1‡	24	1	2.9	0
Total	784	15.3†	24.1	154	10.2	24.3
SEER Rate (1977–1981)		13.6			9.5	

[*] Cases per 100,000 per year. Calculated by direct standardization and adjusted to 1970 US standard population. When there were ten cases or less in a health district, the incidence was computed as the product of the ISIR and the SEER rate.
N, number of cases in five years.
[†] Significantly higher than SEER ($p < 0.05$).
[‡] Significantly lower than SEER ($p < 0.05$).

TABLE VI. Incidence of Gynecologic Cancer in Brooklyn by Race/Ethnicity

	Cancer Site			
Population	Cervix Invasive	Cervix In Situ	Corpus	Ovary
Brooklyn 1978–1982				
Black	23.4*–27.4	34.3†–40.8†	14.2–15.7*	9.1–10.2
White—total	10.3*	19.3†	20.1†	15.3*
Hispanic	15.8*–17.2*	21.6†–24.0†	9.3†–9.8†	7.4–7.9†
Non-Hispanic	8.5	16.3†	21.0†	15.4*
SEER 1978–1981				
Black	20.2	46.1	13.4	9.5
White	8.8	29.3	25.1	13.6
Puerto Rico 1978–1981	18.1		9.2	6.8
Wayne County 1979–1981 (including Detroit)				
Black	24.7	—	14.4	10.1
White	10.5	—	26.7	14.0

[*] Significantly higher than SEER ($p < 0.05$).
[†] Significantly lower than SEER ($p < 0.05$).
Note: The high value in the range is the incidence using the population figures counted in the 1980 census. The low value in the range uses the population adjusted for undercounting of immigrants. It was assumed that there was one uncounted black or Hispanic immigrant for every two counted immigrants who entered the USA between 1970 and 1980.

lyn black and Hispanic women is considerably higher than in non-Hispanic white women. The incidence of cancers of the corpus and ovary is lower in Brooklyn minority women than in non-Hispanic whites.

Compared to the SEER data, there is a low incidence of CIS in both black and white communities in Brooklyn. The low CIS rate in black women is further decreased if the population is adjusted for uncounted immigrants. The excess incidence of ICC is reduced and that of corpus cancer disappears if the Brooklyn black population is adjusted for the census undercount. There is a low incidence of corpus cancer in both Hispanic and non-Hispanic white women. The Brooklyn-wide high incidence of ovarian cancer is attributable to a high rate in non-Hispanic white women, since Hispanic women have significantly low rates.

Invasive Cancer of the Cervix. Both the Brooklyn-wide and individual health district data show consistently high incidences of ICC in black women. In another study,[4] we have demonstrated that the incidence of ICC in West Indian immigrants (18–21/100,000) in Brooklyn is lower than that in US-born black women (27/100,000), whereas Haitian immigrants have high rates of ICC (33–42/100,000). We interpret the high rate of ICC in Bushwick, where the black community has the lowest socioeconomic status in the borough, but where the proportion of immigrants is only 12%, as reflecting the poverty in this community. However, the incidence of ICC in the Flatbush health district (30.1/100,000) may be falsely high, since the population denominator is probably too low. The population of Flatbush includes a high proportion of counted immigrants (49%) and therefore probably has a high proportion of immigrants who were not counted in the census. The high incidence of ICC in the Bedford district (27.4/100,000) may reflect a combination of low socioeconomic status and undercounting.

Correlating the incidence of ICC among white women with the sociodemographic data in Table I makes it apparent that the high incidence districts (Bushwick, Fort Greene, Red Hook, and Williamsburg) are those with the largest proportions of Hispanic women, and that Bushwick and Williamsburg also have the lowest socioeconomic indicators.

Brooklyn-wide, the incidence of ICC in non-Hispanic white women is similar to that in SEER white women, as is the incidence in Bay Ridge where the population is predominantly of Italian origin. However, in the Gravesend health district, the incidence of ICC is significantly low, and in non-Hispanic whites in Flatbush and Brownsville, it is also below the SEER rate. In these three districts, large proportions of the non-Hispanic white population are Jewish. Low rates of ICC in Jews compared to non-Jews in NYC were documented by Haenszel and Hillhouse in 1952,[13] and have recently been reported by Mack et al[14] in Los Angeles.

In summary, while geographic variation in incidence primarily reflects socioeconomic status, ethnicity and immigration play local roles.

Carcinoma in Situ of Cervix. The incidence of CIS reflects both the prevalence of CIS in a population and the level of cytologic screening—the method by which most cases are identified. The ratio of the incidence of CIS to ICC is not a precise measure of screening efforts, but it is noteworthy that in Brooklyn the ratio is less than two in the black and Hispanic districts, but more than two in the more affluent non-Hispanic white areas.

In our other Brooklyn-wide study[4] we demonstrated that US-born black women had an incidence of CIS (42.6/100,000) similar to black women in the SEER population, whereas both West Indian immigrants (23–30/100,000) and Haitians (17–22/100,000) had very low rates of CIS, which probably reflected limited cytologic screening. In this study, the geographic distribution of CIS in the black community in Brooklyn shows that the health districts with relatively small proportions of immigrants have CIS rates similar to SEER, which probably result from the substantial level of screening in US-born black women. The high incidence of CIS in Bushwick probably reflects the low socioeconomic status of this community combined with the high screening activity in US-born women. The low incidence of CIS in Flatbush probably reflects the low screening levels in immigrants who comprise half the black population in this district. The CIS incidence in Flatbush may be even lower because of the undercount of the immigrant population.

The incidence of CIS in the white community is low in both Hispanic and non-Hispanic health districts with the exception of Bushwick. In the heavily Hispanic districts the below-average incidence of CIS combined with the above-average incidence of ICC suggests limited screening. However, in Gravesend, the low incidence of both ICC and CIS probably reflects low prevalence of cervical neoplasia in the Jewish community. Among the non-Hispanic white community in Bay Ridge, where almost 50% of the population is of Italian origin, there is an average incidence of ICC but an unusually low incidence of CIS. This may reflect insufficient screening of older women in this community. In 1980, more than 20% of non-Hispanic white women in Brooklyn were born in Europe, with the largest national group coming from Italy. Anecdotes suggest that immigrant women from Italy or from Ireland may be more reluctant to undergo preventive gynecologic care after the reproductive years than their US-born counterparts.

In summary, in Brooklyn, the low incidence of CIS probably reflects both the lack of cytologic screening in the large immigrant population and also low prevalence in Jewish women.

Cancer of the Corpus Uteri. Burnett[1] reported that the incidence of corpus cancer in white women in NYC in 1976–1980 was lower than the incidence in upstate New York and also in the Detroit metropolitan area. Our data for 1978–1982 for Brooklyn are consistent with Burnett's and also demonstrate that the age-specific incidence peaks above age 65 are consistent with other low incidence US-born and foreign populations, but in contrast to the SEER distribution. Furthermore, whereas in the 1977–1982 SEER population, 78% of corpus cancer was diagnosed in the local stage,[15] in Brooklyn only 70% of white women and 59% of black women had local disease. A low incidence of corpus cancer in the Hispanic community in Brooklyn had been anticipated from national SEER data on Hispanic communities in Puerto Rico, California, and Arizona. In Wayne County, Michigan (Detroit), only 2% of the population is of Spanish origin, in contrast to 18% in Brooklyn. In upstate New York only 2.4% of the population is of Spanish origin. Thus, a low rate of corpus cancer in the Hispanic community in NYC may account for much of the discrepancy observed by Burnett[1] and by us. The incidence of corpus cancer is consistently low in developing countries[16] and among women of low socioeconomic status.

However, this report shows that the triad of features—low incidence, older peak age-specific incidence, and advanced stage—is also characteristic of corpus cancer in the non-Hispanic white community in Brooklyn. The age-specific incidence in non-Hispanic white areas such as Gravesend and Flatbush peaks above age 65 as in other low incidence populations. Haenszel and Hillhouse[13] reported that in 1952 in NYC, the incidence of corpus cancer in non-Hispanic, non-Jewish white women in NYC was 14.2/100,000 (adjusted to the 1950 US standard population) and in Jewish women was 15.6/100,000, somewhat, but not markedly, lower than in other areas in the United States. The gap in incidence between Brooklyn and the rest of the country appears to have increased in the last 30 years.

Possible explanations of the discrepancy between non-Hispanic white women in Brooklyn and white women in Wayne County, Michigan, include the following, separately or in combination: low socioeconomic status, the high proportion of immigrants, low usage of medical care. These three variables may all be interrelated.

Although about 12% of white family incomes in Gravesend, Bay Ridge, and Flatbush are below poverty compared to only 6% in Wayne County, this may reflect the relatively elderly population in these Brooklyn districts (>20% over 65 years) compared to Wayne County (only 14% >65 years). Since poverty is often associated with old age, it is impossible to compare relative socioeconomic status without age standardization.

The large proportion of European immigrants in the non-Hispanic white population in Brooklyn may contribute to a low incidence of corpus cancer for two reasons. First, immigrant women may carry the lower risk of corpus cancer associated with their country of origin, as has been demonstrated for breast cancer, where it takes two to three generations for the risk in immigrants to rise to the high rate in the United States.[17] Second, immigrants are probably conservative in seeking perimenopausal gynecologic care and therefore may be less likely to receive estrogen replacement therapy.

Low physician utilization for gynecologic care or peri- and postmenopausal problems might lead both to failure to diagnose indolent endometrial cancers and to a low utilization of exogenous estrogens. However, exogenous estrogens are less frequently prescribed in the Northeast of the United States than in the Midwest, West, and South.[18] Estrogen-associated endometrial carcinomas are more likely to occur in the early postmenopausal years and are also likely to be more localized at diagnosis.

The possibility that the low corpus cancer rate is due to an unusually high hysterectomy rate, reducing the number of uteri at risk for developing cancer, can be excluded. Brooklyn-specific age-adjusted hysterectomy rates are not available, but hysterectomies are less frequent in the northeast of the US than in the Midwest (including Detroit), West, or South,[19] while the hysterectomy rate in upstate NY is consistent with a relatively low rate in the Northeast.[20] Furthermore, Brooklyn has a low average surgery rate compared to other counties in New York State.[21]

In summary, the low incidence of corpus cancer in Brooklyn probably reflects both low socioeconomic status and the high proportion of immigrants in the borough. However, despite a low incidence, the relatively advanced stage at diagnosis means that corpus cancer deserves serious attention in Brooklyn.

Cancer of the Ovary. Brooklyn-wide, the incidence of cancer of the ovary is significantly high in white women, primarily because of above-average rates in the non-Hispanic health districts. In Bushwick and Williamsburg, with a large low-income Hispanic population, the incidence of ovarian cancer is below the SEER rate. In all health districts where more than three quarters of women over 40 years of age are non-Hispanic and relatively affluent, the incidence is average to above average, consistent with a direct association between socioeconomic status and ovarian cancer incidence. In Gravesend and Flatbush, where a large proportion of the population is Jewish, the incidence is significantly higher than among white SEER women. In European and American-born Jewish women in Israel, the incidence of ovarian cancer (age-adjusted to the standard world population) is 17.2/100,000, in contrast to 12.8/100,000 in Detroit whites, 11.3/100,000 in Italy, and 11.4/100,000 in Warsaw.[16] The equivalently adjusted rates for the white populations of Gravesend and Flatbush are 14.9 and 15.2 per 100,000, respectively. However, our suspicion that this high incidence of ovarian cancer in Brooklyn might be associated with Jewish ethnicity is not confirmed by Mack's Los Angeles study,[14] in which he failed to observe an unusually high rate of ovarian cancer among Jews.

Patterns of Gynecologic Cancer in Brooklyn. Summarizing these incidence data for ten geographic areas and three anatomic cancer sites, two dominant patterns emerge, as shown in Table VII.

The first pattern is in the five northern health districts (Bedford, Bushwick, Fort Greene, Red Hook-Gowanus, and Williamsburg-Greenpoint), where the population is relatively young, low-income, and predominantly black and Hispanic. Here the rates of ICC are consistently high, while the rates of CIS and corpus cancer are average to low.

The second pattern occurs in the three southern health districts (Bay Ridge, Flatbush, and Gravesend), where the population is older, relatively affluent, and predominantly non-Hispanic white. This population has average to below-average rates of ICC, below-average rates of CIS and corpus cancer, and above-average rates of ovarian cancer.

The two remaining health districts, Sunset Park and Brownsville, have mixed populations including both younger, low-income minority communities and relatively affluent, older, non-Hispanic whites. Here there are average to low rates of CIS and corpus cancer, and average rates of ICC and ovarian cancer.

These basic patterns reflect the established associations between age, socioeconomic status, and the incidence of gynecologic cancers. However, at the local level, incidence is also affected by characteristics of specific ethnic groups and patterns of immigration.

Sustained and Extended Preventive Care for Gynecologic Cancer. To date, the main occasion for cytologic screening for cervical cancer has been in the course of reproductive health care. After 20 years of screening in obstetric and gynecologic facilities in Brooklyn, including the abortion

TABLE VII. Patterns of Gynecologic Cancer in the Brooklyn Health Districts

Health District	>50% white	>20% elderly[*]	>30% Hispanic[†]	>50% high school[‡]	>80% above poverty[‖]	Cancer Incidence Compared to SEER			
						ICC	CIS	Corpus	Ovary
Pattern 1									
Bedford	–	–	+	+	–	High	ND[§]	ND	ND
Bushwick	+	–	+	–	–	High	ND	ND	ND
Fort Greene	–	–	+	+	–	High	Low	Low	ND
Red Hook	+	–	+	+	–	High	Low	Low	ND
Williamsburg	+	–	+	–	–	High	Low	Low	Low
Pattern 2									
Bay Ridge	+	+	–	+	+	ND	Low	Low	ND
Flatbush	+	+	–	+	+	ND (W)[§] High (B)[§]	Low	Low (W)	High
Gravesend	+	+	–	+	+	Low	Low	Low	High
Intermediate									
Brownsville	–	–	+	+	–	ND	Low	Low	ND
Sunset Park	+	+	+	–	–	ND	Low	Low	ND

[*] More than 20% of white females aged 65 years or more.
[†] Hispanic women comprise more than 30% of white women aged 20 years or more.
[‡] More than 50% of persons over age 24 have 12 or more years of education.
[‖] More than 80% of families have incomes above the poverty level.
[§] ND = no difference; W = white; B = black.

services available since legalization in 1970, most younger US-born women have had several Pap tests. Clearly, with a large and growing, relatively young, low-income population in Brooklyn, sustained screening in all reproductive health facilities remains the key to cervical cancer prevention. However, screening must be followed by treatment of whatever intraepithelial neoplasia is detected. During the multiple steps involved in diagnosis and treatment, patient contact is often lost. Furthermore, many younger women treated conservatively (by cryotherapy, laser therapy, or conization) continue to be at risk of recurrence. Efforts to treat and follow women with intraepithelial cervical neoplasia must also be sustained and intensified.

In contrast to US-born women, immigrant Caribbean women have a low frequency of screening and knowledge about Pap smears and may be less likely to use contraception.[22,23] Although not necessarily at higher risk of developing cervical neoplasia, immigrants often come from countries with limited cytology services and have less access to health services in Brooklyn. Therefore, specific health education programs for immigrants are warranted.

Preventive gynecologic care after the reproductive years is less available than reproductive health care, especially in low-income communities. Increasingly, young women choose sterilization for birth control and no longer use the reproductive health services where they would receive a Pap test. A study of ICC patients in Brooklyn indicated that the major missed opportunity for screening was at sites providing ambulatory, nongynecologic medical care.[24] Therefore, efforts to extend cytologic screening to medical services and clinics, geriatric clinics, and even urgent care and emergency services are critical to reaching women at risk and further reducing cervical cancer mortality in Brooklyn.

A major effort to extend preventive gynecologic care to peri- and postmenopausal women is also warranted by the advanced stage of corpus cancer and the high incidence of ovarian cancer in Brooklyn. Simple screening tools for the early detection of corpus and ovarian cancer are not yet available, but preventive gynecologic care can alert women to early warning signs, familiarize them with pelvic examination, and thus reduce delays in reporting symptoms. Routine check-ups also provide opportunities for endometrial sampling and pelvic sonography in women who may be at high risk for cancers of the corpus and ovary.

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Missed appointments among patients new to a general medical clinic

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ABSTRACT. Ninety-nine new patients in the general medical clinic of a large municipal hospital were followed over a period of 12 to 18 months. The patients were interviewed at the time of their initial visit to obtain demographic and medical information. Based on their early appointment-keeping behavior they were divided into groups. Those who missed any of their first three appointments were younger, had fewer diagnoses per patient, and presented with problems of a more acute nature than those who kept each of their first three appointments. Retrospective review of the charts 12–18 months after the initial visit demonstrated that patients in the former group were more likely to drop out of the clinic before the completion of the workup for their presenting complaint than those patients in the latter group.

It is concluded that some patients new to a general medical clinic are more likely than others to miss appointments and to drop out prematurely. Although such behavior has not been shown to have adverse medical consequences, it poses major problems in terms of clinic efficiency and must be addressed by clinic managers. An approach to dealing with this problem is suggested.

(NY State J Med 1988; 88:473–475)

During the past two decades, much has been written about the appointment-keeping characteristics of patients in various ambulatory care settings, and a variety of solutions have been proposed to reduce the adverse effects of the 15–30% "drop out" or "no-show" rate.[1-9] More than a decade ago, Hertz and Stamps[1] pointed out that most studies emphasized demographic characteristics such as socioeconomic status, race, age, sex, and educational levels. Occasionally, additional factors have been studied, such as the effects of weather and distance, and variables associated with the health care provider organization such as the appointment system, staffing patterns, and information flow. These authors attempted to eliminate the myth reported in many studies that "low income patients do not keep appointments as well as people in middle-class socioeconomic groups." Their own study suggested that many broken appointments are at least partially caused by provider institutional factors such as physician continuity, communication systems, and type of appointment system. In their literature review of dropouts and broken appointments, Deyo and Inui[3] point out that facts such as patient beliefs, sociobehavioral characteristics, medical aspects of the disease and therapy, patient-provider interactions, and organizational features of the providing facility have been less well studied but are probably more important than patient demographic features. Some reports[2,4,6] ignore the cause of the problem but suggest ways of circumventing it by adjusting the scheduling of appointments to reduce the deleterious effects of broken appointments.

We studied the appointment-keeping behavior of patients new to this facility from their initial appointment through their first 12–18 months. This group of patients was of interest because of the large amount of time spent by physicians, physicians' assistants, and nurses with these patients when compared with "revisits," and the observation made by many of the providers that a significant number of new patients failed to return for their scheduled follow-up visits.

The failure to return after initial visits has significant impact on the utilization of resources in terms of provider and support staff time and the cost of diagnostic and preventive health procedures and tests that may never be utilized by the provider and patients. Also to be considered are the difficulties in patient flow and productivity that are the inevitable result of such behavior. It was of interest to us to see if those factors associated with poor appointment-keeping behavior identified by other investigators were true for this patient population, and if early appointment-keeping behavior would be a good predictor of future appointment-keeping patterns and premature dropout.

Patients and Methods

The study took place at the Queens Hospital Center Adult Medical Clinic in Queens, New York. The clinic is based in a

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large municipal teaching hospital complex and is staffed by faculty internists, internal medicine house staff, and physicians' assistants. There are about 26,000 visits per year to the clinic.

All new patients seen in the clinic on Tuesday afternoons from March 1985 to August 1985 were interviewed by one of the authors after their initial visit with the medical provider. (Tuesday afternoons were selected solely for the convenience of the interviewer.) During the interview, the following information was obtained: date of initial visit, name, address, phone number, age, sex, race, language spoken, prior history at the hospital, mode of transportation, employment status, chief complaint, satisfaction with the visit, medical provider's diagnosis, treatment rendered, date for return to clinic.

Only those patients who required a follow-up visit for review of laboratory data, diagnostic test results, and/or evaluation of treatment were included in the study. Patients seen on initial visit and not given a follow-up date were excluded from the analysis. In all, 99 patients were entered into the study.

The charts of those patients who kept their next scheduled appointment were reviewed for diagnosis, treatment rendered, and subsequent appointment date. Those patients who missed their first or second scheduled appointments were contacted by telephone. Three unsuccessful attempts by phone were followed by a letter. These no-show patients were asked the reason for the missed appointment and were given the name of their medical provider and the clinic phone number so they could make another appointment if desired. The same procedure was followed for the patients' third visits to the clinic.

The charts of the study patients were reviewed in order to tabulate the number of appointments and missed appointments for each patient. Based on the information gathered, the study patients were divided into two groups. Those patients who kept both of their revisit appointments were placed in the return group (N = 35). Those patients who missed either of the first two revisit appointments were placed in the no-show group (N = 64).

Differences between study group characteristics were determined by using the chi-square test with Yate's correction for categorical data of large sample size, Fisher's exact test for categorical data of small sample size, and Student's t-test for continuous data.

RESULTS

Table I shows the characteristics for the two groups studied. The no-show patients were more likely to be younger (38 vs 52 years, $p < 0.001$), to be Hispanic ($p < 0.01$), and to be either employed, students, or housewives ($p < 0.05$) than were the patients in the return group. There was no significant difference between the groups in terms of provider type, sex, spoken language, referral source, mode of transportation, degree of satisfaction with the initial encounter, or length of time to the first follow-up visit. No-show patients had a significantly longer length of time to the second follow-up visit (80.9 days vs 60.7 days, $p < 0.05$).

There were also differences in treatment characteristics between the groups. No-show patients were less likely to have been prescribed a special diet (17% vs 40%, $p < 0.025$) or medications (56% vs 89%, $p < 0.005$) than patients who were compliant with all early appointments.

The return group of patients were characterized by diagnoses of chronic ailments such as hypertension, diabetes, coronary artery disease, congestive heart failure, anemia, osteoarthritis, and thyroid disease. Patients in the no-show group had more diagnoses of an acute nature (headache, abdominal pain) or no diagnosis at all (ie, a normal examination). This more frequent occurrence of diagnoses of chronic ailments in the return group was significant at the $p < 0.001$ level. In addition, the return group had an average of two diagnoses per patient, while the no-show group had 1.4 diagnoses per patient ($p < 0.001$).

In order to control for confounding variables, a stratified analysis was performed by dividing the study group into two populations—those with chronic disease and those with only self-limited disease or no disease at all. Employment status and age were then reexamined within these groups for associations with appointment-keeping behavior (because of the small number of Hispanic patients in each group, after stratifications we were not able to include race in this analysis). Being employed or less than 45 years of age correlated with poor visit compliance only for patients with chronic disease ($p < 0.001$ for each). When these same two variables were examined for the subgroup without chronic disease, the associations were no longer present ($p > 0.9$ for each).

TABLE I. Characteristics of 35 Patients who Returned After Their Initial Clinic Visit and 64 Patients Who Failed to Return for at Least One Follow-up Visit

Characteristic	Returned N (%)	Failed to Return N (%)	*P* Value
Average age (yr)	52	38	<0.001
Sex			
Male	9 (26)	25 (39)	NS
Female	26 (74)	39 (61)	
Race			
Black	26 (74)	38 (59)	NS
Hispanic	2 (6)	20 (31)	<0.01
White/other	7 (20)	6 (10)	NS
Employment status			
Employed/student/housewife	11 (31)	36 (56)	<0.05
Unemployed/retired/disabled	24 (69)	27 (42)	
Diagnoses			
Chronic	27 (77)	22 (34)	<0.001
Acute/self limited	8 (23)	42 (66)	
Average per patient	2.0	1.4	<0.001
Treatment			
Diet	14 (40)	11 (17)	<0.025
Medication	31 (89)	36 (56)	<0.005
Referral	17 (49)	20 (31)	NS
Return to clinic (weeks)			
Revisit #1	5.6	5.7	NS
Revisit #2	6.3	10.8	<0.05

NS, not significant.

The most common reasons given for a missed appointment were failure to remember, and work, school, or other conflicts. Only three patients stated that finances was their reason for not returning to clinic. We were unable to contact 21 (33%) of the 64 no-show patients either by telephone or by letter.

Ninety-four of 99 charts were available for long term follow-up review. After a follow-up period of 12 to 18 months, 17 (54%) of the patients in the return group were still active in the clinic (visit pending or last visit within six months) or had been discharged, and 15 (47%) were inactive (absent for more than six months despite a previously scheduled appointment and no pending appointments). In the no-show group of patients only ten (16%) were classified as active or appropriately discharged, and 52 (84%) were classified as inactive. This difference was statistically significant ($p < 0.001$) and yielded a relative risk for premature drop out in the no-show group of 1.8.

Poor visit compliance during the first two follow-up visits was associated with poor long term appointment compliance. Patients who were initially noncompliant with their appointments but who remained active in the clinic kept only 28 of 41 appointments (68%), whereas the group of patients who were compliant early on and still active at the time of the follow-up kept 97 of 108 appointments (90%). The difference in long term appointment-keeping behavior between the two groups was highly significant ($p < 0.005$).

DISCUSSION

Patients who neither keep nor cancel their scheduled clinic appointments are known as no-shows. They constitute about 15–30% of all clinic patients and represent a major persistent problem in general hospital clinics,[3] primary care centers,[6,8] family practice centers,[9] and other ambulatory care settings.

The present study validates the work of previous investigators regarding patient characteristics associated with poor appointment-keeping behavior. Those patients who missed either of their first two follow-up visits fit the already well-defined profile of the no-show patient: minority individuals who are younger, employed, and have fewer and less chronic diagnoses. Two of these four variables (age and employment status) maintain their statistical significance when the presence of a chronic ailment is controlled for.

The presence of chronic diseases is associated with age. Similarly, the presence of many of these diseases with their attendant complications could affect the patient's ability to be gainfully employed. This "less sick" profile of the no-show patients may explain why Bigby et al[10] were unable to find any deleterious medical consequences of missed appointments.

Other authors[4-6] have reported that previous appointment-keeping behavior is a strong predictor of a patient's future habits. Dove et al[5] found that patients who miss an appointment are more likely to miss future appointments. Goldman et al[6] developed a simplified approach to predicting the likelihood of patients' keeping follow-up appointments based primarily on the percent of "kept" appointments over the previous 12 months. In the present study, a patient's initial appointment-keeping behavior was an accurate indicator not only of future appointment-keeping patterns, but also of that patient's potential for premature dropout from the clinic. A new patient who missed either of the first two revisits had twice the risk of dropping out than a patient who kept both early revisits.

The results of this study demonstrate that at least in settings similar to this one, there is a very high dropout rate among certain new patients. One approach to this problem is to develop systems to predict patient behavior, thereby limiting the impact of poor health-seeking behavior on the clinic. Many such systems have been devised.[2,4-6] We favor an approach that examines institutional behavior and attitudes toward patients. For example, it might be argued that the definition of dropping out is inaccurate, and that in fact many of those patients who dropped out after their initial appointment should never have been given follow-up appointments in the first place. This argument is bolstered by the fact that the second most common reason for patients missing their appointments in this study was conflict with work or school. It would seem that patients (justifiably) place more emphasis on generating income than on follow-up of health problems or preventive medicine. Such actions are consistent with the health belief model of patient behavior,[11] which states that an individual's compliance with health regimens and programs depends on that individual's perception of susceptibility to disease and its severity, or the effectiveness of health prevention or treatment strategies. If susceptibility or effectiveness are perceived as sufficiently high, the person will choose to participate in the health program or treatment regimen. Patients with chronic disease would thus find it easier to identify with the need for follow-up appointments in the clinic (eg, maintenance of health, control of disease, prescription refill, etc). Patients with self-limited diagnoses or no diagnosis at all would not necessarily share this view. This latter group would perceive the need to return to review successful short term treatment strategies or normal laboratory data as relatively less important than other aspects of their daily lives.

We propose that a general medical history be obtained and a physical examination be performed on all new patients. Health maintenance and diagnostic procedures appropriate for age, sex, and chief complaint should also be performed at the time of the initial visit whenever possible. The most urgent problems should be dealt with at this time, with others noted for later evaluation. A return appointment should be scheduled only if medically necessary (not just institutionally convenient); otherwise, the provider should discharge the patient and, at some later date, review the results of the tests ordered. The patient should then be contacted by phone or letter and given a summary of the results and an appropriate next course of action if any is needed. Such a strategy allows for optimal patient care at the time of the initial visit and eliminates unnecessary return appointments. Patient tracking efforts could then be focused on a more appropriate group of no-shows in an attempt to improve health-seeking behavior.

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REVIEW ARTICLE

Alcohol dependence and its medical consequences

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Many theories exist to explain the cause of alcohol dependence.[1] Significant disagreement abounds about the diagnosis and treatment of alcohol dependence (addiction). Uncertainty that it is even a disease that requires medical consideration prevails. Alcohol addiction still remains an often unspeakable, hidden, and provoking subject.

More people drink alcohol today than ever before. The medical and psychosocial problems associated with the drug alcohol are larger and more pervasive than ever before.[2,3] The age range of those affected has dramatically enlarged, so that alcohol-associated problems in teenagers and the elderly have become commonplace.

Social stratum, ethnicity, occupation, and financial status provide no barriers to the onset of alcohol addiction and its complications. What was once the province of Skid Row is now affecting many families and whole communities.

Some studies indicate that between 25% and 50% of patients seen in an average general medical practice will have significant medical and psychosocial problems associated with alcohol use.[3,4] These percentages are increased if other drug problems are included.[2] It is imperative that physicians be skilled not only in the diagnosis of the conditions that are associated with alcohol use but also in the diagnosis of the cause, which is often an addiction to alcohol.

The diagnosis of alcohol addiction can be suspected by detecting the presence of a medical or major life problem related to drinking. The diagnosis is confirmed by identifying the essential features of addiction. The behaviors of addiction are a reliable and consistent means of diagnosing problem drinking. A preoccupation with the acquisition of, compulsive use of, and relapse to alcohol are the most sensitive indicators that alcohol is a consistent problem of central importance.[5]

Tolerance and dependence may not be essential symptoms for the diagnosis of alcohol addiction. These adaptive, homeostatic body functions probably occur in every individual who uses alcohol, whether addicted or not. Dependence can occur after a single dose of alcohol, and tolerance varies according to genetics, as well as dispositional (size), behavioral (learned), and pharmacodynamic mechanisms. The criteria for tolerance and dependence vary widely and can be difficult to confirm clinically.[5,6]

HISTORICAL CONSIDERATIONS

The diagnostic features of alcohol addiction have many parallels to those of syphilis in the prepenicillin era. Many years ago, before penicillin prevented its secondary and tertiary stages, syphilis was considered the "great masquerader." Manifestations of the disease were often confused with a variety of other medical and psychiatric disorders. The diagnosis was frequently overlooked if the suspicion of syphilis was not adequately high. A good clinical differential diagnosis included one of the three stages of syphilis. The reasons for considering the disease were varied and manifold. The long course, systemic and neurologic involvement, inadequate serologic testing, and social and moral stigmata all conspired to obscure and distract physicians from making the diagnosis.[7] Currently, because of early detection and adequate treatment, the many masks of syphilis are no longer frequently seen. Alcohol addiction also has many varied medical and psychiatric expressions.

DISEASE CONCEPT

Morality and Causality. Alcohol addiction remains a moral dilemma for those who have it and for those who try to understand it. A popular view of the cause of alcohol addiction is that it is a mysterious and metaphysical affliction that originates in an "immoral" individual who lacks character. This view also holds that only willpower is needed to overcome excessive or abnormal drinking. Other common beliefs are that underlying conditions, either mental or physical, are primarily responsible for the cause of alcohol addiction. Current medical and psychiatric evidence supports the conclusion that none of the above is true. Recent genetic, clinical, neurochemical, and neuropharmacologic studies suggest that alcohol addiction is a primary disease of the mind and central nervous system.[1,2,4,8-11] The addiction is initiated when alcohol is ingested by individuals with a genetic predisposition to addiction.[12-17] A pattern of pathologic signs and symptoms of a behavioral, psychological, and physical nature ensue from an interaction between alcohol and the brain.[6] Primary alcohol dependence (addiction) by itself has a characteristic clinical course that is predictable and identifi-

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able. Addiction to alcohol can stand alone and does not need other conditions to support and define it.[4,5,9,18]

Denial. A few fundamental points need to be recognized and accepted before the diagnosis can be successfully made. The first and most important point is that denial is essential to the propagation of the alcohol addiction. This denial is present not only in the alcoholic or problem drinker but also in those associated with the addict. The second point is that there is resistance by the addict and those affected by the addict to consider that alcohol may be a serious problem. The third point is that excellent treatment is currently available for alcoholism and often provides a solution to its related medical and personal problems.[8,19]

ESSENTIALS FOR THE DIAGNOSIS

Cause and Effect. The most important criterion for diagnosing alcohol dependence or problem drinking is that drinking alcohol is affecting the person's ability to function either psychologically or physically.[4,9] The American Medical Association's (AMA) National Council on Alcoholism's diagnostic criteria include impairment in social and vocational functioning as well. Caution is urged throughout the process of establishing a diagnosis, because frequently abnormal alcohol use is attributed to the effect of a problem rather than to the cause of it.[4,9,10]

For instance, someone complaining about having difficulty with sleep and who believes himself to be drinking because of it is more likely to be suffering from the effects of alcohol on sleep. Alcohol suppresses REM sleep, so alcohol consumption may cause insomnia.[20] Electroencephalographic studies have confirmed many characteristic neurophysiologic changes that underlie behavioral effects from acute and chronic alcohol use. Acute administration of alcohol produces diffuse slowing of background activity, whereas withdrawal from chronic alcohol administration results in diffuse, fast activity, suggestive of an activated or anxious state of arousal.

Depression is a common consequence of drinking alcohol.[21,22] Anyone who drinks more than one or two ounces of ethanol on any regular basis rarely escapes having a disturbance of cognition and irritability of mood due to alcohol. However, the relationship between alcohol and mood is often incorrectly reversed, in that drinking is attributed to depression. Studies have demonstrated that initially depressed individuals do not necessarily drink more alcohol and may even drink less.[16,17] Alcoholics appear to drink in spite of the depression worsened by alcohol, and even report a dysphoric response to ethanol ingestion.[21,22]

People appear to drink to excess because of a susceptibility to the reinforcing effects of alcohol. Studies suggest that alcoholics inherit a genetic predisposition to develop alcohol dependence.[12-17] People may be motivated to drink, but not to excess, because of stresses and related life problems. Alcohol use in the setting of stress is not abnormal, unless its consumption is marked by a persistent pattern of loss of control.[5]

Most individuals, whether alcoholic or not, appear to consume alcohol for the same reasons, ie, happiness or sadness, success or failure, weddings or divorces, birth or death, or simply to enhance social interaction. Only a casual perusal of drinking practices is needed to confirm these patterns of alcohol consumption. An extremely crucial and difficult notion to comprehend is that a certain percentage of the population drinks too much alcohol in the presence of these life events only incidentally. The reason alcoholics drink is because alcohol affects them differently and apparently provides them with an unusual psychic reinforcement that those without a vulnerability to alcohol do not need.[4,10,11] In short, life problems, emotions, circumstances, and events may lead to alcohol drinking but do not explain abnormal drinking. These life problems are the frequent consequences of abnormal drinking.

Addictive Drinking. Once a personal or social impairment exists in the setting of active drinking, the diagnosis of addictive drinking or alcoholism can be made.[4,18] Abnormal or addictive drinking is defined as loss of control that is manifested by preoccupation, compulsiveness, and relapse with regard to alcohol use.[5]

Preoccupation with alcohol is illustrated in a variety of ways, but the essential feature is the persistent presence of alcohol in the individual's pattern of living. Alcohol occupies a high priority in the individual's routine in spite of problems related to drinking. The importance of alcohol may be evident by regular or intermittent use. Other responsibilities in the individual's life are relegated to a secondary status. Loss of control regarding the importance of alcohol in the alcoholic's life underlies the preoccupation with alcohol.

Compulsiveness is manifested by continued use in the presence of or in spite of alcohol-related consequences. Regular or repetitive use is frequent, but is not a requirement for compulsiveness. The distinguishing feature is the continued abnormal use of alcohol when common sense or logic dictates moderation or abstinence. The pitfall to avoid is attributing the consequences to the cause for compulsive drinking, ie, "I am drinking abnormally because I have marital difficulties." The reverse is more likely to be true. Loss of control of the use of alcohol is particularly evident in the behavior pattern of compulsive use.

Relapse is a critically important and revealing feature of addictive behavior. Coincidental associations between abnormal alcohol use and its consequences may occur without addiction to alcohol, but usually not repetitively in a recurrent identifiable pattern. Relapse is often confirmatory of a suspected or presumptive diagnosis of alcohol addiction. A return to alcohol use in spite of previous adverse consequences can confirm the presence of preoccupation and compulsiveness.

Need for Historical Corroboration. The nature of the disease, with inherent denial directed at self and others, requires a departure from the traditional one-to-one patient-physician assessment for diagnosis and treatment. Often, additional corroborative historical information regarding alcohol intake and related consequences is needed to identify behaviors that suggest an addiction to alcohol. Those who know the patient should be approached with care and concern. Caution is urged even at this early stage in the diagnostic process, since denial and rationalization may exist among those who know the alcoholic. Fear, intimidation, and disdain plague many physicians who attempt to tread the waters of denial and protection within and surrounding the alcoholic. These critical elements represent part of the entire spectrum of the disease of addiction.

SELECTED MANIFESTATIONS OF DEPENDENCE

Medical Disorders—Gastrointestinal Symptoms. Gastrointestinal disturbances commonly occur with alcohol use. Esophagitis (heartburn), peptic ulcer disease, and acute pancreatitis often result from regular alcohol consumption.[23,24] Vague abdominal discomfort, diarrhea, constipation, guaiac positive stools, and gastric cancer are also associated with alcohol use. A further history for alcohol consumption is indicated and can be profitable when gastrointestinal symptoms are the presenting complaint.

Hypertension. Hypertension and tachycardia are common sequelae of alcohol ingestion. Virtually anyone who drinks alcohol in any amount on a regular or irregular basis will have a blood pressure elevated over his or her baseline during the acute withdrawal period.[25-27] Elevated blood pressures usually occur during the first few days of acute withdrawal from alcohol.[25-27] The elevation may be sustained for weeks after chronic use or for only hours after acute ingestion. The hypertension and tachycardia which occur need not be precipitated by many years of chronic drinking, since they represent a normal physiologic response by the sympathetic nervous system to the alcohol withdrawal that follows a single episode of consumption or dose of alcohol.[28] An elevation in the first 24 to 72 hours of 20 to 30 mm Hg in systolic blood pressure and 10 to 20 mm Hg in diastolic blood pressure over that of baseline is typical for a given individual. The blood pressure may return to normal without specific therapy. Individuals with a history of hypertension should be screened for alcohol use before being diagnosed as having essential hypertension. A significant percentage of patients diagnosed as having essential hypertension will turn out to have chronic alcohol ingestion as the cause of their problem,[25-27] and the remainder will probably require less antihypertensive medication.

Heart Disease. Alcoholic cardiomyopathy is probably a common condition. Heretofore, many cases of alcohol-induced cardiomyopathy may have been mistakenly diagnosed as idiopathic cardiomyopathy, secondary to viral infection. If accurate alcohol histories are obtained in the presence of the diagnosis of cardiomyopathy, alcohol may be found to be the culprit.

Congestive heart failure from any cause may be precipitated by a combination of the toxic effects of alcohol on the heart and the hypertension and tachycardia induced by the sympathetic nervous system in response to alcohol ingestion.

Liver Disease. Laennec's cirrhosis occurs in only about 5–10% of all alcoholics,[23,24] usually in the advanced stages of alcohol consumption. Serious sequelae associated with cirrhosis of the liver are esophageal varices, hepatorenal syndrome, and hepatic coma. Transient elevations of liver enzymes such as alkaline phosphatase, serum glutamic oxaloacetic transaminase, and others can be useful if present, to indicate recent alcohol use, alcoholic hepatitis, and acute fatty infiltration of the liver.[24] These enzyme markers return to normal within days to weeks after cessation of alcohol consumption.

Hematologic Manifestations. In the setting of regular alcohol consumption, an elevated mean corpuscular volume (MCV) of the peripheral blood erythrocytes often indicates either a folate or B_{12} deficient state that is not uncommon in alcoholics. Alcohol can selectively or universally suppress the bone marrow to produce subsequent leukopenia or pancytopenia. These hematologic deficiencies frequently revert to normal after cessation of alcohol use.[24]

Other medical conditions that result from or are significantly aggravated by regular alcohol use include glucose intolerance or diabetes mellitus. Management of diet and weight control is made difficult because of chronic alcohol use in which there is poor compliance with diet. Ethanol is used as an energy source in preference to other food sources. The direct toxic effects of alcohol may produce chronic pancreatitis with insulin deficiency and subsequent glucose intolerance.[23,24]

Neurologic Disease. Neurologic complications such as the dementia syndrome, amnesic states (blackouts), seizures, hallucinosis, and peripheral neuropathy are relatively common with chronic alcohol consumption.[28] Some degree of dementia manifested by a global impairment of intellectual function in areas of concentration, memory, and abstraction abilities is a frequent accompaniment of regular alcohol use.[29] Direct toxicity to the brain that includes destruction of or damage to neuronal cell bodies, dendrites, and axons by the chemical ethanol has been well documented in animals and humans as a cause of dementia. The Wernicke-Korsaff syndrome involves a specific loss of recent memory out of proportion to other cognitive deficits attributable to thiamine deficiency, and should be differentiated from the global, intellectual impairment in alcoholic dementia.

As little as two ounces per day of 80 proof whiskey can result in measurable cognitive deficits that are often reversible with complete abstinence from alcohol.[29] Depressed IQ scores in the areas of concentration, memory, and abstractions measured in the majority of alcoholics continue to show improvement as long as 13 months following cessation of alcohol consumption and maintenance abstinence.[29]

Psychiatric Complications. Alcohol produces a multiplicity of subtle and overt psychiatric symptoms. These symptoms can and do cluster in a constellation to produce psychiatric syndromes.[10,30-32] The manifestations of any of the psychiatric syndromes can be indistinguishable from and identical to those produced by causes other than alcohol.[31,32]

Depression. One of the more common symptoms and syndromes produced by alcohol is depression.[10,21,22,30-32] Depression that is secondarily produced by alcohol can be severe and is associated with suicide.[33,34] Alcohol dependence as a primary disorder represents the second most important risk factor in suicide, next to age.[34] Idiopathic affective disorder as a primary cause of depression is of only low relative importance in a discriminant function analysis of risk factors for suicide.[33,34]

The subjective symptoms and objective signs of depression induced by alcohol are identical to those required for the diagnosis of a major depression in a primary affective disorder. An individual may complain of sadness, dysphoria, profound hopelessness, a sense of worthlessness and self-blame, which at times can be of delusional proportions. The chemical effect of alcohol tends to produce poor control of emotions, tearfulness, crying spells, lethargy, poor initiative, lack of energy, and general mental and spiritual demoralization. An individual may demonstrate a depressed affect, psychomotor retardation, and disturbances in appetite and sleep.[4,10,21,22] The initial treatment of the secondarily induced depression is abstention from alcohol use.[21,22]

Many studies indicate that consumption of alcohol initiates and sustains a depression.[10,21,22] Few studies actually support the popularly held notion that depression causes alcoholism. Initially, depressed individuals may drink more, the same, or less and do not show a consistent pattern of drinking in the presence of depression. An alcoholic continues to drink persistently in spite of the drug-induced depression for reasons attributable to addictive drinking. In most alcoholics, the preoccupation with, compulsive use of, and relapse to drinking appear to be related to alcohol and not to result from depression. However, some studies suggest that in women 20% of cases of alcoholism are secondary to affective disorders, while in men only 5% are secondary.[10,21]

Anxiety. Anxiety is a cardinal manifestation and clinically useful indicator of frequent or excessive alcohol use.[31,32,35] The symptom of anxiety is produced by the discharge of the autonomic nervous system largely through the contribution of the sympathetic neurons located in the hypothalmus that are excited during alcohol withdrawal.[28] These symptoms can range from apprehension, tension, overreactivity to internal and external stimuli, excessive response to stress, sweating, and tremors. The alcohol blood level cannot be practically maintained at a steady state, since this would require a constant infusion over time unachievable by oral intake, especially in the presence of a development of tolerance to alcohol. A decline in the blood level of alcohol triggers the firing of the sympathetic nervous system to produce the onset of withdrawal symptoms. The spectrum of the presentation of anxiety may range from a subtle, mild, pervasive anxiety to a strikingly evident state such as in delirium tremors, which is an extreme manifestation of anxiety in alcohol withdrawal.[10,28]

Sexual Problems. Sexual dysfunction and various forms of impotency are commonly associated with alcohol use. Complaints in the area of sexual performance should suggest taking an alcohol history. Decreased desire (libido) regularly occurs in men and women who frequently consume alcohol. Paradoxically, alcohol may induce uninhibited, aggressive sexual behavior that may be offensive and even dangerous. Alcohol use may dull the emotions and impair the ability to experience the intimacy required for mutual satisfaction in sexual performance.[36]

Excessive sympathetic discharge during alcohol withdrawal may lead to failure to maintain an erection and/or premature ejaculation in men. In men, alcohol intoxication produces difficulty in achieving and maintaining an erection and experiencing ejaculation, while in women it may cause absence of orgasm.[36] Chronic persistent alcohol intake may result in testicular atrophy and subsequent feminization of unopposed estrogen effects in men.[24]

Personality. Personality disturbances that result from alcohol dependence are also common. The types of maladaptive behaviors and mental and mood disorders that develop in alcoholics are many and varied. Hysterical and antisocial attitudes and behaviors are often evident in those alcoholic individuals who might not ordinarily manifest them in a sober or treated state. Alcoholics may become very attention-seeking, demanding, aggressive, emotionally labile, immature, and blaming of others. The defense mechanisms of denial and projection that are operative in alcohol addiction extend to many other facets of the alcoholic's life. A refusal to admit and accept responsibility for one's actions and attribution of one's faults to others are favorite ploys of the alcoholic. Antisocial acts and attitudes of varying types and degrees frequently present in the alcoholic include disrespect, lack of concern, assault upon others, driving while intoxicated, and other indications of poor judgment and self-centeredness.[4,8,9]

It is important to recognize and emphasize that a variety of personalities are represented among alcoholics before the onset of dependence. No specific type of personality appears to be predisposing to the development of dependence. As discussed, a common superimposed pattern of personality abnormality in the alcoholic may develop during the course of alcohol dependence.

Trauma. Trauma of all kinds is frequently associated with alcohol use. Visible evidence of trauma, such as bruises, new and old wounds, and fractures, is an indication of intoxication. The intoxicated state that affects mental judgment and motor coordination lends itself to vulnerability to accidental trauma. Also, a toxic effect of alcohol suppresses certain clotting factors that increase susceptibility to bruising and bleeding. Accidents are a leading cause of death at any age, and alcohol often plays a prominent role in these accidents.

Cancer. Various types of cancer have been associated with chronic alcohol use. These include carcinomas of the oral pharynx and gastrointestinal tract, such as the esophagus, stomach, and liver. Alcoholics frequently smoke cigarettes, so that lung cancer is also common in alcoholics.[23,24]

Respiratory Problems. Alcoholics frequently have chronic pulmonary problems because of associated cigarette smoking and suppression by alcohol of the immune system.[23,24] More specifically, chronic obstructive pulmonary diseases such as chronic bronchitis and emphysema are commonly found in alcoholics. Regular drinkers will often have a productive, irritative, sometimes nauseating cough in the morning, especially if they smoke cigarettes. Pneumonia and tuberculosis are more common in alcoholics.[23,24]

Skin Manifestations. A large array of skin abnormalities may be present on physical examination of the alcoholic. These may include cigarette burns, decreased hair, gynecomastia, increased flushing or vascularity of the face, livedo reticularis (reddish-blue mottling), multiple contusions, abrasions and cuts in various stages of healing, nicotine stains on fingers, palmar erythema, major rubars, rhinophyma, spider angiomas, unexplained edema, and evidence of poor personal hygiene.

Sociologic Complications. Interpersonal relationships are the most fundamental and severely affected area of an alcoholic's personal life. The difficulties in establishing and maintaining productive relationships with others often begin with those close to the alcoholic, particularly family, fellow employees, and significant others. Frequently, marital discord, separation, and impending divorce are symptoms of the underlying disturbance in interpersonal relationships. Often the alcoholic will project his problems in interpersonal relationships onto others, and only relate how "misunderstood" he is by spouse and friends, displaying the characteristic denial of his responsibility for the difficulties.

Employment problems may or may not be obvious. The alcoholic may or may not be significantly affected depending on the measurements of performance. He or she is often dishonest in presenting the history associating alcoholic intake and employment-related consequences. Performance may gradually decline and the capacity for productivity diminish as the dependence progresses. When the alcoholic is able to meet his or her job-related responsibilities, it can prompt rationalization that alcohol is not having a significant effect on work performance.

Legal problems may exist or develop. Offenses often take the form of driving while intoxicated, assaults, or other expressions of impropriety. Such indiscretions and poor judgment indicate loss of control over alcohol.

Other Drugs and Medications. The pure alcoholic, in whom alcohol is the only or clearly the predominant drug used addictively, is vanishing. Studies indicate that 80% of the alcoholics under the age of 30 years use at least one other drug regularly and addictively.[2,8] The propensity to freely exchange one drug for another, or to use them simultaneously in an addictive way, is common and alarming.

In obtaining a history of alcohol intake and related consequences, a careful screen for other drugs is mandatory.[37] The list of drugs to which alcoholics may become addicted is large. Cross-tolerance and dependence between alcohol and many other drugs exist. The vulnerability for abuse and addiction to alcohol appears to extend to significant numbers of other drugs, suggesting a generalized susceptibility to addiction to chemicals possessed by alcoholics and drug addicts.

Because drug seeking behavior appears to be common in alcoholics, careful, judicious and sparing use of any medications should be exercised. Drug seeking behavior for any drug or medication should be met with skepticism and scrutiny, and may require confrontation of the motivations behind medication requests. Common illicit drugs and prescription medications regularly used by alcoholics include marijuana, cocaine, phencyclidine, hallucinogens, benzodiazepines, barbiturates, anticholinergics, and others.

TREATMENT OF ALCOHOL DEPENDENCE: DETOXIFICATION

Many effective schemes for detoxification during the acute alcohol withdrawal period exist. Most approaches include a benzodiazepine, antihypertensives, magnesium sulfate, and vitamins, as indicated by the severity of the withdrawal measured by blood pressure, pulse, and tremors. The majority of alcoholics today experience only mild withdrawal and require minimal amounts of medication.

A thorough and complete medical history and physical examination are mandatory, as well as a neurologic examination as indicated by the clinical presentation. Extensive laboratory testing is an essential screen to determine clinically inapparent, medical, neurologic, and psychiatric diseases and to confirm the presence of alcohol-related or unrelated conditions.[37] A battery of tests, including a complete blood count, liver and renal function, electrolyte, and serologic tests, urinalysis, and drug screen for additional drugs is often essential for a complete evaluation. Further evaluation of the neuroendocrine status, such as dexamethasone and thyroid stimulation tests, may be indicated for coexisting psychiatric, neurologic, and medical problems.[37]

Treatment. Alcohol dependence is a chronic addiction that is associated with progressive medical, psychiatric, and social deterioration. Severely affected alcoholics may require hospitalization and specific inpatient treatment for their alcoholism before recovery can be initiated. Hospitalization is a particularly powerful intervention that can effectively interrupt the cycle of addiction, so that the denial and rationalization can be confronted and dissipated.[19] In addition, a full medical, neurologic, and psychiatric evaluation can be undertaken to investigate coexisting illness and disorders.[19]

Outpatient treatment is indicated for less severe forms of alcohol dependence, in which the course and consequences of alcohol addiction are not long and particularly serious, and the system of social and economic support for the alcoholic is adequate. Drug problems in addition to alcohol addiction tend to undermine outpatient treatment.

Effective inpatient and outpatient treatment programs utilize a 12-step approach to recovery based on Alcoholics Anonymous (AA). Additional individual and group psychotherapies are often helpful in assisting the alcoholic in achieving full recovery. An intervention and treatment program directed towards the family is essential for the optimal success of recovery for the alcoholic and the entire family.

Direct referral of the alcoholic and family to Alcoholics Anonymous and Alanon (for the nonalcoholic) is also suggested. Although AA is a highly desirable program for alcoholism, a significantly greater initial success rate may be achieved by instituting inpatient or outpatient treatment before attending AA and Alanon.[8,11,19]

CONCLUSIONS

The definition of alcohol addiction contains three critical elements: preoccupation with the acquisition of alcohol, compulsive use of alcohol, and relapse or recurrent use of alcohol. Difficulties with interpersonal relationships involving others or employment or legal problems with alcohol may exist, prompting a reduction or elimination in alcohol use in nonalcoholics. Alcoholics, on the other hand, continue their compulsive use of alcohol in spite of and not as a result of these difficulties.

Most alcoholics or problem drinkers can stop drinking and do. There may be varying periods of days, weeks, months, or even years without any alcohol consumption. Relapse or return to drinking often occurs. Again, various circumstantial reasons are given to explain relapse.

Other associated manifestations of alcohol use are medical, psychiatric, neurologic, traumatic, neoplastic, and sociologic. Alcohol must be diagnosed as the causative agent to adequately prevent and treat these conditions. The alcoholic possesses a generalized susceptibility to develop addiction to other drugs and medications. This vulnerability to universal drug addictions should be considered in prescribing medications to alcoholics.

Denial and rationalization are present in the alcoholic and those surrounding the alcoholic. The traditional one-to-one, doctor-patient interview is often inadequate to overcome the denial. Additional, corroborative history is needed to acquire information to identify the behaviors of addiction. Caution is urged even in this step, since the denial and rationalization are so pervasive.

A multifaceted treatment approach with the diagnosed alcoholic is recommended. Detoxification, local, regional, and national treatment centers (inpatient and outpatient), and regular meetings of Alcoholics Anonymous have become the standard treatment approaches for alcoholism.

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Temporary insanity and premenstrual syndrome: Medical testimony in an 1865 murder trial

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Medical arguments used during the trial of Mary Harris, who murdered Adoniram J. Burroughs on January 30, 1865, are still being debated. Semantics may have changed over the years, but the intent is certainly the same. Premenstrual syndrome (PMS) is still being suggested as the cause of antisocial acts. Temporary insanity is still being used as a defense to explain criminal behavior, although in 1865 the terms used were "paroxysmal insanity" or "moral insanity." [1]

Aspects of the Mary Harris case may yield clues as to the persistence of the attitudinal stances of the physicians, the legal profession, the mass media, and the public. In this paper the medical practices of the period are also elucidated regarding temporary insanity and dysmenorrhea.

BASIC FACTS ABOUT THE MURDER AND THE INSANITY DEFENSE

At 4:00 on the afternoon of January 30, 1865, Mary Harris stepped out from behind the floor-to-ceiling grandfather clock on the second floor of the Treasury Building in Washington, DC. She fired two shots from her 1859 Sharps' four-barrelled .32 caliber derringer at Adoniram J. Burroughs as he walked down the hall leaving work for the day. Tearfully, she explained that he had promised to marry her, ruined her, and then married someone else.

At the trial, Harris' chief defense counsel, Joseph H. Bradley, Sr, presented a hypothetical case[1(pp 101-105)] for the reaction of physician witnesses, using their medical expertise. Even granting the defense lawyer's probable bias in the presentation, the basic facts in the extract that follows are generally accurate in terminology and fact.

> Mary Harris was not more than ten or eleven years of age when she attracted the attention of Adoniram J. Burroughs, who was at least twice her age. While Harris had very few cultural advantages, Burroughs was an educated man, experienced in business and affairs of life. They were in daily association since he was in [the] mercantile business and she was employed in a nearby millinery and fancy store. Burroughs plays with her as a child; she sits on his knee and receives and returns his caresses. This intimacy continues for about two years and he becomes a trusted friend of Harris' employer and is daily at her store. After he fails in business, Burroughs comes to keep the books of Harris' employer.
>
> He is a Baptist; she a Roman Catholic. Prepared by his culture and instruction, Harris is admitted into the best and most refined and cultivated society in Burlington, Iowa. Burroughs leaves Burlington to seek employment elsewhere and corresponds with Harris from November 1, 1853 until the Spring of 1863. Harris' parents discover that this correspondence is going on. Her father is enraged. Then, Burroughs' visits are prohibited. However, a Burlington woman consents to permit them to meet at her house. She is formed, molded, trained by Burroughs in her habitudes of thought, morals, and manners. Eventually, they are engaged to be married. However, his plans do not materialize and he is too poor to marry. He prevails on Harris to leave her father's house to go to Chicago to seek employment there near him.
>
> In Chicago, Harris has few friends, principally married ladies older than herself. Her chief pleasure is her correspondence with Burroughs. Thus nearly five years are passed. In the meanwhile, three different times had been chosen for their marriage and deferred.
>
> Burroughs is about to leave Chicago to come to Washington, D.C. in search of employment. When they were last seen togeth-

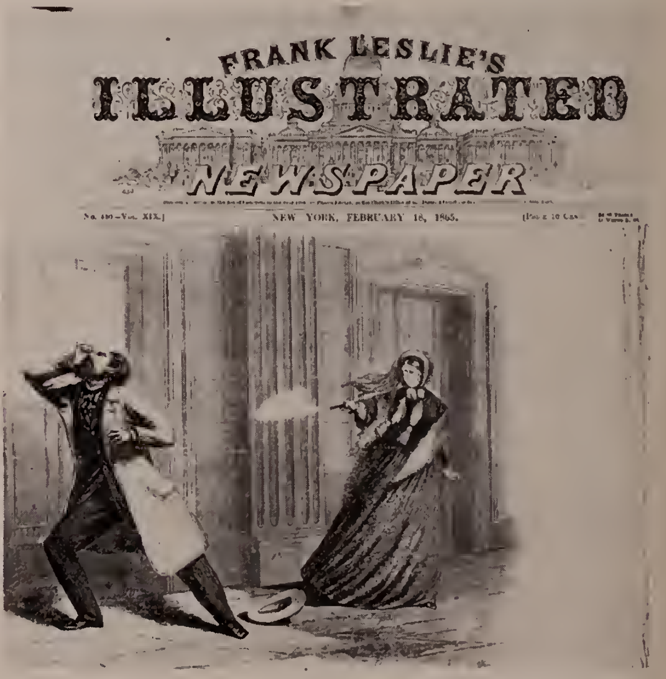

FIGURE 1. The caption of this illustration, from *Frank Leslie's Illustrated News* (February 18, 1865), reads as follows: "Tragedy in the Treasury Department at Washington, D.C., Jan. 30—Mr. Burroughs, a Clerk in the Currency Bureau, Shot Dead by Miss Mary Harris."

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er, Harris was sitting on his knee and he was playing with her curls.

Six months elapse. In the meanwhile, Burroughs obtained employment in the U.S. Treasury Department in Washington, D.C. as a clerk.

On August 7th, 1863, Harris received a letter from Burroughs asking where he could see her. They met in Chicago, Burroughs holding her hand while they talked, and they seemed to part as ever—friends. On September 8th, 1863, Harris received a letter signed by J. P. Greenwood. On September 14th, 1863, Harris received another letter from Greenwood begging her to meet him at an address, later discovered to be a house of ill-fame. Inquiring at the Post Office, Harris found clear proof that these letters were written by Burroughs. On being convinced of this fact, she was greatly distressed and became wildly excited. A few days after this, Harris discovered that four days after the receipt of the first of these letters, and one day after the receipt of the second, Burroughs was married to Amelia L. Boggs in Chicago by his brother, Rev. John C. Burroughs, then President of the University of Chicago.

Within less than a week after this discovery, Harris was sick enough to require medical attention. Dr. Calvin M. Fitch was called and he treated her for painful dysmenorrhea. However, Dr. Fitch did not know her personal history and did not witness any mental disturbance. Her sickness lasted a few days. She became silent, moody, and melancholy. Her nights were spent in sleeplessness and tears. She went about her daily duties routinely. That continued for two or more periods. At last, Dr. Fitch directed that she should lie in bed till after she had had her breakfast. She then slept as she had from the first of May 1863, in the same bed with Louisa Devlin, in whose employment she lived as clerk, and in the same room with Jane Devlin. At times, she would get up from her warm bed in the inclement mid-winter climate of Chicago, and go into an adjoining room in her nightclothes and sleep on the bare floor. During one of these periodical sicknesses, Harris stealthily got up from the bed, leaving Louisa Devlin supposedly asleep, softly dressed herself, approached the bedside of her friend, and said in a low tone, "I must leave you." Louisa threw her arms around Harris' neck and said: "Why, where are you going?" Harris answered, "I wanted to take a walk on the lake shore." Louisa restrained her forcibly and prevailed upon her to undress and go to bed.

At another of the periods of her sickness, Harris was sitting at a table with her two friends, her employers. Harris was sitting nearest the younger sister, Jane, who was nearer her own age. While thus sitting, Harris reached out toward Jane remarking, "Don't you want to read some fine letters?" Jane recognized the handwriting of the man who had so long corresponded with Harris and had been engaged to marry her. Jane was familiar with all the facts and replied, "No; I never want to see any of his writing, or any other such a fellow's; and I never wish to hear Burroughs' name mentioned again as long as I live." In an instant, Harris snatched up a carving knife and attacked Jane, but Louisa restrained her. After a severe struggle of several minutes, Louisa succeeded in getting the knife from Harris.

After a while, Harris insisted upon and attempted to get out of the window and to go into the street and was forcibly restrained by the two sisters. Thinking that Harris would be relaxed quicker by yielding to her, Louisa opened the door and let Harris go into the street. Jane followed and kept Harris in sight. After wandering around two or three blocks, Harris stopped a street car, put her foot on the step, then turned away and walked quietly down the street. Harris entered the private ladies' entrance of the principal hotel in Chicago. Jane and Louisa then went for a gentleman, whose wife was a very warm friend of Harris, and who himself had much influence with her. All three went to the hotel and endeavored to prevail upon Harris to return home. They failed and left. As night approached, Harris came home alone, composed, and "clothed in her right mind." This exhibition of violence was the longest in duration that had occurred up to that time.

FIGURE 2. Mary Harris (from *Harper's Weekly*, March 4, 1865, p 140).

In another return of her periodical sickness, without cause or provocation of any kind, Harris struck Jane several repeated blows over the head with the window-brush—a heavy brush used to cleanse the store windows.

During another sickness, Harris struck a lady customer with a pincushion that had a piece of brick in it.

At another time in Chicago, Harris purchased a small-sized Sharps' revolving pistol with a case of cartridges. When asked by Louisa why she purchased the gun, Harris replied, "Many ladies carry pistols." In addition, Harris said she feared that Burroughs and his brother had a plan to seize her and carry her off and she had the gun for her defense.

Harris employed an attorney in Chicago to sue Burroughs for breach of his promise to marry her. A writ was issued, but Burroughs could never be found. Her counsel urged and advised her to compromise. Harris refused. She said she didn't want money but wanted her character and reputation upheld. She urged her counsel to come to Washington with her and sue Burroughs there. He declined because he told her that her love seemed to have turned to hate.

Then, Harris came to Washington from Chicago alone to institute a lawsuit there. She visited the Treasury Department and learned that Burroughs had gone that day with his wife to Chicago. Traveling without stopping, Harris took the return train to Chicago and found that his wife had arrived there, but he had not.

Louisa and Jane Devlin moved from Chicago to Janesville, Wisconsin. Harris accompanied them. But her life was the same; a brooding melancholy pervaded it. She performed all her duties as clerk and saleswoman, but she shunned society, and her spirits were gone. Periodically, sometimes not every month, but in two months, these exhibitions were revived, but with less violence. In the latter part of December 1864, Harris seized an expensive patchwork silk quilt and began to cut and tear and destroy it. It required a great force to get it from her. Harris was taken to her room and locked in. Prior to that, Louisa, in hope that it would bring relief to her, agreed to furnish Harris with money to come to Washington to institute a lawsuit there for breach of promise.

On January 1st, 1865, Harris left for Washington, by way of Baltimore, where friends of the Devlins resided. She traveled alone. On reaching Baltimore, she went to a respectable boardinghouse, where she was unwell with a bad cold, and was detained for three weeks, and then had her periodical sickness. On

FIGURE 3. Mary Harris' Chief Defense Counsel, Joseph H. Bradley, Sr (L. C. Handy Studios, Washington, DC).

Saturday, January 28th, Harris told a lady who occupied the same room with her the history of her case. Harris showed her a large package of letters from Burroughs. She told of his attempt to get her to that bad house; she read parts of the letters; she said that she was coming to Washington to see for herself whether he was here before she consulted counsel; she had the name of a lawyer here whom she was to employ; she said her sole object was to vindicate her fame and reputation. Harris continued talking and reading and handling the bundle of letters long after two o'clock Saturday night. On Sunday night the same thing occurred.

On Monday morning, the small Sharps' pistol and the bundle of letters were lying on the bureau. Harris had shown the pistol to the lady who occupied the room with her and did not conceal it. While making her preparations, Harris was called suddenly and told she was late. She threw the bundle of letters into the trunk and put the pistol into her pocket instead and hurried downstairs. Harris came to Washington alone on the train. From the station, Harris walked to the Treasury Department, inquired for Burroughs, and opened the door of the room in which he was and saw him distinctly.

An hour or more after this, as the clerks were leaving the office, Burroughs came near her in the hallway. Harris drew the pistol and fired. No one saw her fire, but there were three persons nearby who saw her instantly after the shot was fired. That shot hit Burroughs, who turned, saw her, exclaimed, "Oh my God!" and fled. She then cocked her pistol, leveled it in his direction, and fired a second time as he disappeared around a corner of the hall. Then, Harris turned and walked quietly downstairs and out of the building. Just outside of the building, Harris was arrested and taken back and placed in a room with a policeman. By this time, a Justice of the Peace arrived and Harris immediately handed the pistol to him. Up to that time, Harris had not shed a tear. She paced the room in violent agitation; tore her hair; knelt on the floor and sprang up; knelt to the Justice; and her face was convulsed, but she shed no tear. Hugh McCullough, the Comptroller of the Treasury, came in and spoke to Harris. She asked if Burroughs was dead. He said he had often seen representations of mental agony on the stage, but he never witnessed the reality till then. She was still tearless. McCullough asked Harris two questions: one whether Burroughs had wronged her in any other way; the other whether she was a virtuous woman. Harris answered both rapidly and relapsed instantly into the same excitement. To the latter question she replied, solemnly and clearly, "As God is my judge, I am." She was on her knees, clinging to his clothes. He raised her more than once. Her exclamations were, chiefly, "Why did I do it?" or "How could I do it;" "I loved him better than my life;" "I would have died for him;" etc., etc.

Harris was committed to jail. On her way to the jail, Harris told the policeman that Burroughs had caused her to be driven from home and friends; that he had taken her to a bad house and had seduced her; that she had procured that pistol and came here to avenge the injury, and that she had done so. He understood that Harris said she got the pistol just before she left home, and came directly here for that purpose. The policeman cautioned her against making any statements; yet she persisted in doing so. During the whole time she was greatly excited. When they reached the jail she was so exhausted that she had to be supported by him and others into the jail.

For days after her commitment she paced the jail cell in violent agitation. By the latter part of February she had calmed down. Two friends, a gentleman and his wife from her old home at Burlington, came to see her. Harris was so changed they would not have known her had they not conversed with her. She was changed in appearance, mind, and manner. This was during her periodical sickness. In the latter part of March, during that condition, Harris' pulse was about 110 and her hands were cold. She spoke incoherently of the death of Burroughs.

On recurrence of her period in April, as on the previous occasion, she showed great insensibility to cold. Her pulse was nearly 120; the back part of her head very warm, and her hands as cold as if they had been in water. When speaking of any matter connected with the shooting, the pupil of the eye was so dilated as almost to cover the iris. At times the face was fixed, and the eye fixed on vacancy, as in, or similar to, cases of catalepsy. She did not believe Burroughs was dead; she said she saw him there; had frequently seen him in her cell. With difficulty, she was calmed down, and then she became cheerful. After a brief time, the excitement returned; the pulse rose to near 120; the same condition of the face and eyes returned; she talked incoherently. This continued for half an hour or more, when she became composed. On the return of her period in May, the same physical condition existed as in April, but in a much more excited form. Still greater mental disturbance was disclosed in her language and acts. She said she would not stay any longer in that prison; that she was going out; that bars could not restrain her. She exhibited great violence of manner. She fancied she heard dreadful cries and voices and shrieks. This occurred again in a lesser degree on the next day, and on the third and fourth days after that. Her conversation and language on these occasions showed that she thought and spoke of Burroughs as then alive, and she spoke of him in terms of endearment.

In June, when her period returned, there were but slight changes from her normal condition. During the intervals, Harris was entirely possessed of her faculties, but was generally very quiet, and oftentimes melancholy—with a temperament altogether changed from what it was up to the time of her disappointment in love.[1(pp101-105)]

As Mr Bradley put it, Burroughs "formed, shaped, and molded her mind, feelings, habits, tastes, and her intellectual and moral character." [1(p10)] Burroughs wrote 92 letters to Harris during almost five years of corresponding, often mentioning stolen kisses, using many endearing terms, and frequently expressing his love and admiration for her.

At the murder trial, Harris' defense lawyer claimed her actions were caused by "paroxysmal insanity from moral reasons." A forensic physician testified that Harris was temporarily insane from the combination of her being "crossed in love" and her painful dysmenorrhea at the time of the shooting. In opposition to the medical expert, "common sense" physicians testified that Harris was not insane, merely hysterical.

Among the press and the public during the trial, the popular impression was that Harris was not insane and that "a cunningly devised fiction was being foisted on the jury regarding her mental condition." [2]

On July 19, 1865, the jury took only five minutes to acquit Mary Harris. She was set free without any restrictions with a "not guilty" verdict.

MEDICAL TESTIMONY AT THE TRIAL

Defense attorneys alleged that after the notice of the marriage of A. Judson Burroughs to Amelia L. Boggs appeared in the *Chicago Tribune*[3] on September 16, 1863, Mary Harris' disposition drastically changed. Amelia Boggs was the daughter of a wealthy merchant from Chicago and Harris believed that Burroughs had deserted her for financial reasons. In November, a bogus notice about Harris' marriage appeared in the *Chicago Tribune*[4] and Harris made sure that Burroughs received a copy.

Harris' condition worsened and Dr Calvin M. Fitch was called to treat her toward the end of September 1863. In his testimony, Dr Fitch concluded that Harris suffered from severe congestive dysmenorrhea due to irritability of the uterus and that such uterine irritation always affects the nervous system. "In some instances it develops insanity. . .uterine irritability is one of the most frequent causes of insanity." He gave her an anodyne to quiet the nervous system and to induce sleep. His testimony also noted that "disappointed affection is one of the most frequent" of the moral causes of insanity. Furthermore, Dr Fitch stated that the combination of "uterine irritation" and "disappointed affection" produces a greater effect. On cross examination, Dr Fitch told the jurors that "there are many cases on record of paroxysmal insanity." [1(p51-52)]

In medical practice for 22 years with a specialty of the study of the mind for 18 years, Dr Charles H. Nichols was the major expert medical witness for the defense. In fact, Dr Nichols was the only witness examined as an expert on insanity. Dr Nichols was then superintendent of the Government Hospital for the Insane in Washington, DC, and had been so ever since the asylum opened in 1852.

Dr Nichols visited and observed Mary Harris in jail four or five times. During one of their conversations, Dr Nichols wrote a question on a card, so not to offend Harris' "womanly delicacy," asking about her dysmenorrhea at the time of the shooting. Harris replied, in writing, that she was unwell three days before coming to Washington.[1(p74)]

In addition, Dr Nichols was present for most of the testimony at the trial. His own observations were reinforced by the testimony of the comptroller of the Treasury, Hugh McCulloch, who observed and spoke with Mary Harris immediately after the shooting and again when he and his wife visited her in jail. On the stand, McCulloch described in vivid detail Harris' intense emotional distress, her wild looks, and her uncontrollable behavior, such as her pulling out her hair.[1(p24)]

Dr Nichols was also in the courtroom when, in an unusual legal twist, chief defense counsel Joseph H. Bradley, Sr, was also allowed to testify as to his observations on visits to his client. Although not a medical expert, Bradley commented that his interest in moral insanity, paroxysmal insanity, and paroxysmal insanity from moral causes went back some 20 years and continued to this day. In detailed descriptions, containing medical references and psychological inferences, Bradley recounted a number of incidents that clearly illustrated to the jury Mary Harris' "paroxysmal insanity." In concluding his testimony, Bradley said that there were "three occasions in which she was undoubtedly insane. She was insane from moral causes aggravated by disease of the body." [1(p17)]

Based on his own interviews and the testimony of McCulloch and Bradley in particular, Dr Nichols concluded: "Mary Harris' brain and nervous system are large and active." Her dysmenorrhea had existed since autumn 1863 to the present time. He said that she had had her menstrual period at the time of the murder. In his opinion, she was "unusually susceptible to either a physical or moral cause of insanity." Dr Nichols stated that her "disappointment in love" shocked her delicate moral sensibilities. Dr Nichols strongly stated that Mary Harris was "unquestionably insane at times." He cited her fearing harm from Lincoln's assassins while in jail as an example of her delusions. Referring to the *McNaughton* rule, District Attorney Edward C. Carrington asked Dr Nichols whether a knowledge of right and wrong was any longer

FIGURE 4. Dr Charles H. Nichols, expert medical witness for the defense.

considered a test of insanity. Dr Nichols said, "I do not consider a knowledge of right and wrong, in the abstract, as a test of insanity." Dr Nichols diagnosed Mary Harris' condition as "paroxysmal mania." [1(p23)]

Common-Sense Medical Testimony

To counter the expert testimony of Dr Nichols and the conclusions of Dr Fitch as he attended to Harris, the prosecution called upon five prominent "common-sense" physicians in the DC area.[5,6] Interestingly, defense lawyers commented during the trial that even the "common-sense" doctors supported their contention that Mary Harris was insane at the time of the murder.

Dr John Frederick May had practiced surgery and general medicine since 1834. He was the first to perform an ovariotomy, and did most of the major surgery in the District of Columbia. At one time, Dr May was a professor at Columbia College, holding the Chair in Principle and Practice of Surgery.[5(pp149-150),6(pp229-230),7(pp770-771)] Shortly before the Harris trial, Dr May had been called in to identify the body of John Wilkes Booth since he had removed a carbuncle from Booth's neck and was familiar with the resultant scar.[8,9(p38)] Although not an expert on mental diseases, Dr May categorized insanity into four types: mania, monomania, dementia, and idiocy. At first, Dr May testified regarding the condition labeled "hysteria." He said, "I consider hysteria as a disease emanating from the uterus." Yet Dr May also stated that, "Unquestionably [disappointment in love], I should say is a very powerful cause [of mental disturbance]." [1(p106)] In reaction to the hypothetical case presented by defense attorney Bradley, Dr May concluded: "I have no hesitation in saying. . .that the person labored under a deranged intellect, paroxysmally deranged, produced by moral causes, and assisted or increased by a physical cause, derangement of the uterus." [1(p106)]

In Dr Thomas Miller's reaction to the hypothetical case, he stated that he agreed completely with Dr May.[1(p108)] Dr Miller was a professor of anatomy at Columbia College and an attending surgeon at the Washington Infirmary. He had been president of the District of Columbia Board of Health and was a founder of the Washington Medical Institute in 1830. A possible rationale for including Dr Miller may have been his prior activity in providing care for the insane and establishing the Government Hospital for the Insane.[5(pp169-174),6(pp226-227),7(pp794-795)] However, on October 21, 1852, Dr Miller had protested strongly against "importing a physician [referring to Dr Nichols' coming from New York] to take charge of the Government Hospital." It was rumored that Dr Miller wanted the position for himself.[10(p140)]

Dr William P. Johnston, who had been professor of obstetrics and diseases of women and children at Columbia University since 1845,[5(pp178-179),11] described the differences between hysteria and dysmenorrhea. He concluded from the hypothetical case that the patient suffered from dysmenorrhea and hysteria. In addition, Dr Johnston said, "We consider an individual suffering from hysteria as irresponsible for any act which she might commit." [1(p108)] He was the first physician in the District of Columbia to devote special attention to diseases of women, and had the reputation of being the most successful and popular obstetrician in the District of Columbia. He was also a founder of the Pathological Society of Washington. Secretary of War Stanton had given him permission to care for Confederate prisoners in the Old Capitol Prison.[6(pp231-232)]

Dr Noble Young was the physician at the prison, where he observed Mary Harris daily during her confinement. His credentials included holding the Chair of Theory and Practice of Medicine at Georgetown Medical College for 25 years and the presidency of the faculty.[5(pp162-164),6(p227),12] Dr Young testified that he prescribed porter for Harris' sleeplessness. However, he did not see any indication of insanity or of dysmenorrhea. He stated that her last period had been June 16 to 19, 1865.[1(p106)]

Finally, the jury heard from Dr Flodoardo Howard. Dr Howard was professor of obstetrics and diseases of women at Georgetown Medical College and had been a pharmacist before graduating in medicine at age 30.[13(p34),6(p152)] In reaction to the hypothetical case Dr Howard said, "I would suppose the patient thus described to be subject to mental alienation, and that she was subject to insane impulses—possibly suicidal or homicidal mania." [1(p109)]

Clearly, the district attorney's "common-sense" doctors did not destroy the insanity proposition advanced by the defense. While there were shades of differences, all the physicians agreed on the medical possibility of Mary Harris' derangement.

In his closing arguments, District Attorney Carrington acknowledged Judge Andrew Wylie's instructions to the jury that Dr Nichols was an expert medical witness and they should be bound by his opinions if they believed him credible. However, Carrington contested the credibility of Dr Nichols. He stated that Dr Nichols made false assumptions in arriving at his decisions. Those false assumptions and their corrections were enumerated by the district attorney[1(p173)]:

1. A marriage contract existed between Mary Harris and Adoniram J. Burroughs.
2. Mary Harris was melancholy due to disappointed love.
3. A. J. Burroughs made a dishonorable proposition in the letters signed by J. P. Greenwood.
4. Mary Harris suffered from dysmenorrhea at the time of the shooting.

Responding to each false assumption, Carrington bolstered the supposition of a deliberate murder:

1. There was no evidence that a marriage contract existed. Even if a contract existed, it was violated by Mary Harris.
2. Mary Harris went to parties, played cards, and engaged in actions that did not indicate a melancholy mood. Mary Harris never loved A. J. Burroughs.
3. Reverend John C. Burroughs, brother of the murdered man, said that the J. P. Greenwood letters were not written by A. J. Burroughs. Reverend Burroughs was a Baptist minister and the president of the University of Chicago at the time.
4. Mrs Flemming, Mary Harris' landlady when she stayed over in Baltimore on her way to Washington,

> DC, said that Mary was not sick when she left for DC that fateful morning to seek out Burroughs.

Talking about Dr Nichols' theory in his closing remarks, District Attorney Carrington was quite harsh in his personal characterization of the defense's expert medical witness: "...this propagator of the new and dangerous doctrine—this modern philosopher of the humbug of paroxysmal insanity." [1(p180)]

Carrington forcefully attacked: "...This is a case of murder, and this woman is not scientifically insane; she is not legally insane; she is not periodically insane; she is not spasmodically insane; she is not even hypothetically insane; but she is as sound today as she was before the murder; and this idea of insane impulses is a modern humbug."[1(p172)]

Jurors may have read Dr Nichols' testimony in the local newspapers when he was called as a defense expert at the trial of Lincoln's assassins in June 1865.[14,15] Nichols was to testify regarding the insanity of Lewis Payne, one of the conspirators along with John Wilkes Booth who attacked Secretary of State Seward and his son and was later hanged for Lincoln's murder. In the questions and answers printed in the newspapers, Dr Nichols defined and gave the symptoms of moral insanity and responded to hypothetical examples. Payne's defense lawyer, William E. Doster, told Dr Nichols that Payne shouted, "I'm mad, I'm mad," after attacking Secretary of State Seward. Answering, Dr Nichols stated that madmen seldom say that they are mad and therefore he would suppose the attacker to be feigning madness. Owing to his wife's death on June 12, 1865, Dr Nichols was not able to complete his examination and to be present for the defense's efforts to introduce an insanity plea for Payne.[16] Dr Nichols' reputation as a medical expert on insanity was certainly enhanced by the numerous newspaper stories about the assassination trial.

It is doubtful that District Attorney Carrington's efforts to belittle the credibility of Dr Nichols as an expert medical witness had an impact upon the jury. In stark contrast to the district attorney's rationales, defense lawyer Bradley simply told the jury to "...let this poor, blighted, afflicted, ruined, and persecuted girl go. The law has no claim on her." [1(p138)]

Aware of the reactions of spectators in the packed courtroom and the jurors, prosecutor Carrington acknowledged the obvious popular sentiment. "If you acquit her upon the ground of insanity, it will be a pretext only...if you wish to acquit her, do it because you want to do it; but do not render yourselves ridiculous by listening to this nonsense of insane impulse." [1(p177)]

Apparently, Carrington judged the courtroom climate correctly. Jury foreman John Scrivener quickly led his fellow jurors back after a few minutes of deliberation. He faced Mary Harris and rendered a "not guilty" verdict. There was not a verdict of "not guilty by reason of insanity." [1(p181)] That verdict was met with cheers and applause in the crowded courtroom.

NEWSPAPER AND PROFESSIONAL REACTION TO THE "NOT GUILTY" VERDICT

Mary Harris' "not guilty" verdict was soundly denounced in newspaper editorials, stories, and in comments. "The public tended to suspect fraud in defendants' pleas of insanity, particularly as such pleas became more frequent. Newspaper stories fanned these feelings." [17(p198)] At a May 1867 meeting of the Association of Medical Superintendents of American Institutions for the Insane, Dr Nichols appraised existing sentiments: "The public press pretty universally condemned the verdict in this case, and in a few instances, the medical press united in that condemnation." [18]

MEDICAL/LEGAL PROFESSIONALS' REACTIONS

Major articles appeared in the *American Journal of Insanity* in 1866. One was by Dr Isaac Ray[19] on "The Insanity of Women Produced by Desertion or Seduction." Dr Ray was Superintendent of the Butler Hospital for the Insane (Providence, Rhode Island), a founder of the Association of Medical Superintendents of American Institutions, and famous as a theoretician of medical jurisprudence. In 1838, Dr Ray published *A Treatise on the Medical Jurisprudence of Insanity*.[20]

Another article was entitled "The Trial of Mary Harris." [21] Although the second article's author was not given, it was most likely written by Dr John P. Gray, superintendent of the Utica (NY) Lunatic Asylum and editor of the *Journal* for 30 years.

In later years the *American Journal of Insanity* became the *American Journal of Psychiatry*, the official publication of the American Psychiatric Association, the new name for the Association of Medical Superintendents of American Institutions for the Insane.

Dr Ray defended the moral insanity theory as put forth by Dr Nichols and agreed with the concept. In support, Dr Ray noted that Dr Nichols presented the psychological and pathological aspects in the Mary Harris case with a force and clearness seldom exhibited on the witness stand.[19(p265)] Ray also commented about the community's feelings that the insanity plea was merely "a dodge whereby a villain is saved from merited punishment." [19(p127)]

On the other hand, Dr Gray was decidedly opposed to the philosophy of moral insanity. He stated that in the medicolegal sense of the term, no delusion was exhibited by Mary Harris at any time in her history. Her temporary visual and aural hallucinations in Chicago and in jail were the "natural result of excitement and not worthy to be mentioned...Any degree of mental enfeeblement is also disclaimed for her...There remains, then, the plea of moral insanity, which we maintain has not, and cannot have, any place in the sciences of law or medicine." [21(p356)]

Dr Gray claimed that periodical or paroxysmal insanity are two species of disease and not a single entity. Furthermore, Dr Gray stated that neither dysmenorrhea nor disappointed affection causes homicidal insanity. That cause and effect was merely a popular error of attribution. In fact, Dr Gray said that during the trial Dr Fitch described hysteria alone. Dr Gray concluded that there were no grounds for a diagnosis of insanity for Mary Harris. With a flourish, Dr Gray commented that moral insanity is indistinguishable from moral depravity.[21(p356)]

In response to the reading of Dr Ray's article on the insanity of women at a meeting of asylum superintendents, Dr Gray commented[22]:

> Mary Harris, though belonging to the general class discussed, does not come within the category of typical cases described, and certainly is not such a case as the one detailed. . .I can hardly conceive the phrases "paroxysmal fury" and "uncontrollable criminal impulse" as the result of disease, applicable to the Mary Harris case.

Those sentiments reinforce the assumption that Dr Gray was the author of "The Trial of Mary Harris" in January 1866. In addition, the author of the article referred to "our respected" friend, Dr Nichols—another clue.

In those same proceedings, Dr Thomas S. Kirkbride, then president of the Superintendents' Association, took the argument one step further while trying to pacify the divided audience[22(p97)]:

> In regard to the Mary Harris case, as much as we may differ in respect to her condition of mind, I am very sure we shall not differ in the fact that a great outrage was committed upon the community when she was acquitted on the ground of insanity and then immediately discharged. It seems to me a perfect farce of judicial proceeding.

At a later date, Dr Gray expressed similar feelings when he testified that Charles J. Guiteau, President Garfield's assassin, was not insane, while Dr Nichols argued for obvious insanity.[23] (Interestingly, that Gray/Nichols confrontation took place in the same courtroom where the testimony of Dr Nichols had saved Mary Harris.) In addition, Dr William W. Godding,[24] the superintendent of the Government Hospital for the Insane who succeeded Dr Nichols, also pointed out that "he [Dr Nichols] was more widely known and respected than any other expert in the courtroom; probably every member of the jury knew him personally."

At a later date, the Government Hospital for the Insane was renamed St Elizabeths Hospital, as it is known today. However, St Elizabeths is now administered by the Commission on Mental Health Services of the District of Columbia's Department of Human Services.

Ironically, Dr John P. Gray fell victim to an unpredictable "irresistible impulse" after he returned to Utica from Washington after the Guiteau trial. Gray's career was cut short on March 16, 1882. On that evening, a man named Henry Remshaw, "a manipulator at a Turkish bath in the city," was allegedly seized by a fit of insanity. Remshaw burst into the Utica Asylum and, without a word, shot Gray in the face. Gray never recovered his health after that attack, and died four years later.[25–27]

Another important article appeared in the *Quarterly Journal of Psychological Medicine and Medical Jurisprudence* in 1867. Dr R. L. Parsons,[2] superintendent of the New York City Lunatic Asylum, enumerated the following eight principal points to consider regarding the insanity of Mary Harris:

1. Conduct of the defendant at the time of the homicide
2. Natural physical and mental characteristics
3. Facts possibly acting as causes of insanity
4. Physical causes which may have led to insanity
5. Facts regarding disturbance of intellectual faculties
6. Facts regarding disturbance of affective faculties
7. Physical condition relative to insanity
8. Insane acts of the defendant

By the time Parsons wrote his article, he was able to say that Dr Nichols had been proven correct in his conviction about the insanity of Mary Harris. Parsons noted that Mary Harris was now in the Government Hospital for the Insane.

WHAT HAPPENED TO MARY HARRIS AFTER THE TRIAL?

On May 25, 1867, the 21st Annual Meeting of the Association of Medical Superintendents of American Institutions for the Insane convened in Philadelphia, Pennsylvania. Dr Nichols responded to inquiries from attendees and brought the membership up to date about the Mary Harris case.[18]

Using another name, Harris moved to Richmond, Virginia, shortly after the trial and continued to live and work with the Devlin sisters, her employers ever since she moved to Chicago. After several hostile incidents, Harris attempted to stab a man named Collins, who had married Louisa Devlin. While Harris did no serious harm, she did actually cut his clothes. Harris was taken to the District of Columbia by Louisa Collins (Devlin) to meet with her former defense lawyer, Joseph Bradley, Sr. Bradley asked Dr Nichols to examine Harris as to her mental condition. Dr Nichols found Harris' "physico-pathological condition to be that of catalepsy. Her positions were fixed, rigid and continued, though often times quite awkward and such as would be painful to a person in health. Considerable force was necessary to raise her arm or change her position, but a limb or her body would remain in any position to which it was brought. It was also difficult to obtain from her answers to questions." [18(p124)]

Louisa Collins told Dr Nichols that Harris had eaten and slept very little for several months. In addition, Dr Nichols was told that Harris exhibited suicidal tendencies and had spoken about wishing to die and even the methods that she would use. Dr Nichols did not relate the suicidal propensity to any remorse for the Burroughs murder. Harris had often stated that she did not desire to live and had previously left the house under circumstances giving rise to the suspicion that she intended to commit suicide.

As she always had, Harris "denied distinctly that she was insane, and that she needed any such treatment as insane persons require; or, in fact, that she needed any treatment at all, on account of ill health." [18(p125)]

At the end of the examination, Dr Nichols told Mr Bradley that he "regarded her [Harris'] insanity as dangerous to herself and others. . .unless she was put under restraint, she would either commit a homicide or suicide." [18(p125)]

After persuasion by her lawyer, Harris reluctantly came to the Government Hospital for the Insane with Mr Bradley. Two physicians from the hospital examined Harris and made out the necessary certificate, under oath, for her admission as a free District patient.

ASYLUM COMMITMENTS

On February 1, 1867, Harris was admitted to the Gov-

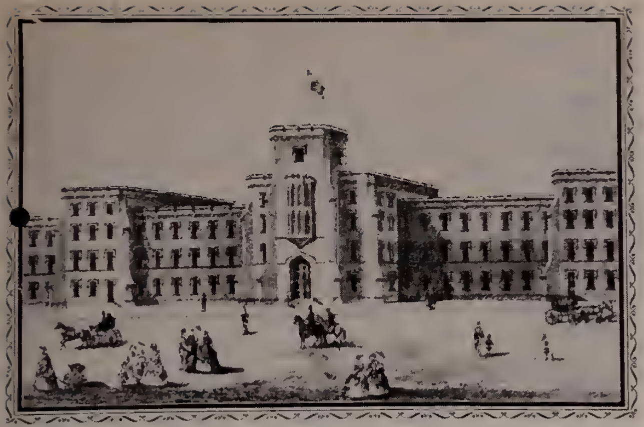

FIGURE 5. The Government Hospital for the Insane, Washington, DC (from the cover of *Mental Hospitals*, May 1855, Association of Medical Superintendents of American Institutions for the Insane, Elmira, NY).

ernment Hospital for the Insane in Washington, DC, with a diagnosis of periodic homicidal mania. In 1867, Dr Nichols reported that Harris' health was considerably improved. She was sleeping soundly, eating regularly, and even gaining weight. On the other hand, Dr Nichols' account of her aberrant behavior is telling in its graphic detail[18(pp125-126)]:

> She has been nervous and irritable during her catamenial periods, and on several occasions she had displayed a tendency to violence. She frequently expresses the desire to leave the Hospital, and always maintains that she is not insane and never has been. When asked why she does things that are so extraordinary, she usually answers she really does not know, and is only partially aware of what she has done. On other occasions, she says she knows she has done the strange things of which she is reminded, but could not help doing them. She usually lies in bed during her monthly sickness. She appeared to be passing through a recent sickness favorably, when being in bed and alone in her room, she suddenly got up and broke everything that was destructible in the room. It appeared that no external exciting cause could have induced this act.

From 1867 to 1881, Harris had three admissions and discharges from the Government Hospital for the Insane: from February 1, 1867, to September 2, 1868 (one year, seven months); from March 8, 1873, to April 10, 1875 (two years, 1 month); and from September 13, 1875, to July 1, 1881 (five years, ten months).[28,29]

Additionally, the hospital's patient register noted that Harris was born in Ireland, had a common education, and resided in the District of Columbia. She was single and white, her station in life was civil and indigent. While Harris was known to be Catholic, her second and third admissions listed her religion as Protestant. Furthermore, there was a pencilled notation that the patient was named Harris or Harrison, an alias that she had used previously.

In addition to those commitments, Harris also enjoyed a brief escape from July 5 to July 26, 1873, until she was recaptured in Philadelphia and returned to the Government Hospital in Washington, DC. Dr Nichols immediately discharged the attendant who had helped Harris over the wall using a ladder.[30]

MORAL TREATMENT

In all her time at the hospital, Harris' moral insanity was treated with a prescription of moral treatment. If an individual becomes paroxysmally insane due to moral causes, it is homeopathic logic to assume that moral treatment will rectify the situation. In today's terms, moral treatment may be considered akin to the concept of the therapeutic milieu. A perusal of the moral treatment at Bloomingdale Hospital (NY) as described by Dr Pliny Earle in the mid-1800s illustrates the therapeutic concept of the times[31]:

> In the moral regimen at this institution, every practicable effort is made to pursue that system, at once gentle, philosophical, and practical, which has resulted from the active and strenuous endeavors of many philanthropists, in the course of the last half century, to meliorate the condition of the insane. The primary object is to treat the patients, so far as their condition will possibly admit, as if they were still in the employment of the healthy exercise of their mental faculties. An important desideratum for the attainment of this object is to make their condition as boarders, as comfortable as possible; that they may be the less sensible of the deprivations to which they are subjected by a removal from home. Nor is it less essential to extend them the privilege, or the right, of as much liberty, as much freedom from personal restraint as is compatible with their safety, the safety of others, and the judicious administration of other branches of curative treatment. The courtesies of civilized and social life are not to be forgotten, extending, as they do, to the promotion of the first great object already mentioned, and operating, to no inconsiderable extent, as a means of effecting restoration to mental health.

In 1847, the superintendent of the Utica (NY) Lunatic Asylum, Dr Amariah Brigham, also offered a definition of moral treatment[32]:

> The removal of the insane from home and former associations, with respectful and kind treatment under all circumstances, and in most cases manual labor, attendance on religious worship on Sundays, the establishment of regular habits of self-control, diversion of the mind from morbid trains of thought, are now generally considered as essential in the moral treatment of the Insane.

Both Drs Earle and Brigham believed in the therapeutic value of a teaching program. Patients in these institutions could attend lectures on subjects such as natural philosophy; chemistry; animal physiology; astronomy; physical, intellectual, and moral beauty; recitations of poetry; history and description of Malta; Greece as it was in 1838; and characteristics of the Americans and the Europeans. Dr Nichols presented concerts with experts using the improved musical instruments, particularly the new organs.

Moral treatment was not a single specified technique but a combination of a variety of activities. All the activities combined sought to produce an improvement in the patient's condition.

Thus, "moral treatment may be defined as organized group living in which the integration and continuity of work, play, and social activities produce a meaningful total life experience in which the growth of individual capacity to enjoy life has maximum opportunity." [33]

MARY HARRIS' CODA

Mary Harris spent more than nine years out of the 14 years between 1867 and 1881 as a patient at the Government Hospital for the Insane. Under Dr Nichols' administration, Harris participated in a moral treatment regimen similar to those described. Between her admissions, Harris worked as a clerk in the Philadelphia post office using the name Mary Harrison.[34-35] It is probable that her chief lawyer, Bradley, helped her to secure the job since his father had been a high level administrator in the post office for many years.

In addition, Harris may have worked in the Washington, DC, law office of Judge Charles Mason, another one of her former defense lawyers, and also a former resident of Burlington, Iowa, as was Harris.[36] Since Mason was a former Commissioner of the US Patent Office, he may have also helped Harris to get a job in the Patent Office at one time.[37]

Harris continued working for about two years after her last discharge from the Government Hospital for the Insane. Suddenly, newspapers in Washington, DC, Chicago, New York, and elsewhere[38-41] reported on a romance of the court that culminated in Philadelphia, Pennsylvania, on October 31, 1883. Mary Harris married Joseph H. Bradley, Sr, the chief defense lawyer who defended her in 1865. He was over 80 and Harris was about 40. Bradley died four years later in 1887, and his obituary notices recounted his career including the Mary Harris trial and the subsequent wedding.[42-44]

At this point, the trail of Mary Harris came to a dead end. Several possibilities occur. She may have returned to Burlington, Iowa, to live with her family or she may have moved to Philadelphia where she previously worked and was married. On the other hand, the trauma of Mr Bradley's death may have resulted in her return to an insane asylum. In any event, nothing more was uncovered about what happened to Mary Harris.

Surviving members of the Bradley clan residing in the Washington, DC, area were asked for information. Only a few people responded to questions about what happened to Mary Harris. Typically, they said: "We don't know anything about Mary Harris and we don't want to know anything about her." Not surprisingly, it is highly likely "that the family disapproved of Mary Harris all the while." [45]

THE CONTINUING AND CONTINUOUS CONTROVERSY

Today, there are still arguments about the concept of moral insanity, the causal relationship of premenstrual syndrome to temporary insanity, and the use of the insanity defense in criminal proceedings.

Moral Insanity. On May 19, 1863, the Association of Medical Superintendents of American Institutions for the Insane held its annual meeting at the Metropolitan Hotel in New York City. With Dr Nichols and Dr Gray leading the verbal exchange, five of the members acknowledged their belief in the moral insanity concept while eight rejected the idea; one of the eight had previously supported the theory.[46] Tracing the battle back even further, an article in a professional journal[47] noted that the debate began when Dr Isaac Ray "espoused such revolutionary ideas as moral insanity and irresistible impulse as defense against criminal responsibility."

As late as 1975, a lengthy historical review revisited the moral insanity controversy, explicitly illustrating the scope of the differences of opinions during the years when the theory was popular.[48] Even Stendhal's literary classic, *Le Rouge et Le Noir*, utilizes the relation of legal insanity to moral insanity.[49]

It is apparent that concern over moral causation leading to violent actions still remains with us. Typically, after John Hinckley shot President Reagan, he was immediately given a psychiatric examination seeking extenuating circumstances that could have initiated a mental condition.

Premenstrual Syndrome. As early as 1953, researchers wrote about the medicolegal aspects of the premenstrual cycle, commenting on its relation to crime. Attention was given to premenstrual syndrome in the courts, PMS as a special defense, and the use of PMS as medical evidence in criminal trials.[50] By 1980, the same researcher discovered that 44% of 50 women charged with violent crime had committed the act during paramenstruum. There were 26 violent acts against people including four manslaughter charges, one attempted murder, five grievous bodily harm acts, five actual bodily harm actions, and eight assaults. Significantly, there was a lack of offenses during ovulatory and postovulatory phases.[51] Another researcher presented three case studies of cyclical criminal acts in premenstrual syndrome: manslaughter, arson, and assault.[52] Historically, one book even discussed a PMS rationale for the behavior of Mary Todd Lincoln during her husband's presidency.[53] Mary Harris was also mentioned in a Canadian perspective of the legal implications of premenstrual syndrome.[54] In addition, a comprehensive historical review looked at women, menstruation, and 19th century medicine and commented on happenings during the "catamenial week." [55]

Moving into the late 1900s and contemporary history, the *New York Times* published an editorial in 1982 that was headlined "Raging Hormones." That editorial dealt with the fitness of women to hold high executive offices, such as the US presidency. An allegation was made that "raging hormonal influences" due to premenstrual tension rendered women unfit to hold high office.[56] During that same year, the Brooklyn, New York, District Attorney, Elizabeth Holtzman, prosecuted an arson case in which the defense opted for PMS as its rationale. Holtzman responded: "PMS is a defense without merit. . .There is no scientific evidence that there is any such thing as a syndrome which causes women to become insane or violent in connection with the menstrual cycle. . .There was nothing to justify an insanity defense." [57] In 1986, PMS became a matter of "psychofashion" according to one commentator. It was noted that ". . .elevating PMS to official recognition would perpetuate myths that menstruating women are pathological cases." To offset that recognition, it was suggested that PMS be renamed "periluteal phase dysphoric disorder (PPDD)." [58] In keeping up with current events, the *New York Times* commented that PMS keeps the doctors guessing, looking for the causes and the treatments.[59] Interestingly, between January 1982 and May 1984, the National Library of Medicine reported 155 articles on PMS in the professional literature and stated that 5–10% of the female population was severely afflicted.[60] In continuing to focus upon the issue, *U.S. News & World Report* in 1986 published an article, "Is PMS Mental Illness? Debate Grows." [61]

Insanity Defense. Similar to the attitudes expressed during the Mary Harris trial, there continues to be an undercurrent of disbelief when the insanity defense is used in criminal proceedings. An article headline in an 1984 issue of *Psychology Today* sums up the feeling: "Innocent Until Proven Crazy." [62] Another article asked, "Are All Women on Trial?" with respect to PMS in the courts.[63] Still another, in 1986, commented on the bias against women in defining mental illness: "Crazy by Definition." [64]

Obviously, the insanity defense is not relegated solely for use by females. In fact, correspondence from 1911 to 1922 between Dr William A. White, superintendent of the Government Hospital for the Insane, and a law school professor, Edwin R. Keedy, discussed many aspects of criminal responsibility for all the mentally ill.[65]

Furthermore, a number of articles have thoroughly reviewed the insanity defense, noting the misconceptions about overuse in the courts, the battle of medical experts in testimony, the characteristics of the defendants, the necessity of punishment, the disposition of the acquitted defendants, and even the moral basis of the special defense of insanity.[66-70] Questions requiring research and suggested reforms are included in these analyses of the insanity defense in the judicial system.

An often used French proverb succinctly ties the pieces together: Plus ça change, plus ça reste la meme (The more things change, the more they remain the same). Proceedings in the Mary Harris case in 1865 demonstrated the legal usage of concepts such as "moral insanity," "paroxysmal insanity from moral causes," "crossed in love," "disappointment in love," periodical sickness," and "dysmenorrhea as the causal factor." These concepts continue to evoke intense pro and con responses today as professionals and the public discuss the premenstrual syndrome and temporary insanity.

Acknowledgments. While working on a novel dealing with details of a character's confinement in a mental institution, I came upon the *American Journal of Insanity*. Turning pages rapidly, looking for ideas, I came across a short, six-sentence notice about the murder trial of Mary Harris for shooting A. J. Burroughs. A brief note mentioned that Burroughs knew Mary when she was 12 and he was 24. Those few sentences intrigued me and I made a note to myself that here was a good idea for a novel. When the original novel was completed, I turned to researching the Mary Harris story. That was seven years ago, and the search still continues.

As often happens in research, serendipity plays a major role in the collection of information. Many days spent reading magnified images of the local newspapers of the 1800s yielded clues in direct proportion to the level of eyestrain incurred. In turn, those serendipitous findings led to others and to a chain of additional sources. Each finding also resulted in a euphoria akin to winning at an Atlantic City casino or discovering a missing anthropological link.

In the course of my sporadic research efforts, a large number of people and/or organizations took the time to talk with me, to suggest additional sources, to write to me, and to send me material. I want to thank:

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Jehovah's Witnesses and the transfusion of blood products

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Jehovah's Witnesses are Christians who hold the Bible to be the complete Word of God. The sect was founded in the 1870s as a nondenominational study group by a Pennsylvanian named Charles Taze Russell.[1] Similar Bible study groups joined in 1881 to form the Watchtower Bible and Tract Society, which by 1909 was an international organization with headquarters in Brooklyn, New York.[2] In 1931, the organization adopted the name Jehovah's Witnesses. The name comes from the book of Isaiah, where the Lord, Jehovah, said, "You are my witnesses."[3] The organization first referred to the sanctity of blood in December 1927 in its biblical publication, "The Watchtower." Seventeen years later the same publication referred again to the biblical law forbidding blood transfusion on penalty of loss of eternal life in God's kingdom.[4] Jehovah's Witnesses currently number approximately 3.2 million worldwide, 25% of whom reside in the United States.[5] Jehovah's Witnesses comply with the vast majority of modern medical and surgical treatment. Some are physicians, even surgeons.[6] They do not smoke, use recreational drugs, or have abortions, claiming that they cherish and respect human life too much to waste it.[7] Why then the seemingly contradictory objection to life-preserving blood transfusions? What is in the Bible that Jehovah's Witnesses interpret as forbidding, under the penalty of loss of eternal life, the use of blood products, which were not even imagined possible at the time the Bible was written?

BIBLICAL BASIS FOR RELIGIOUS BELIEFS

Blood is frequently mentioned in the Bible. In one passage from Genesis after Noah survives the flood, God tells him, "Every moving animal that is alive may serve as food for you. As in the case of green vegetation, I do give it all to you. Only flesh with its soul—its blood—you must not eat."[8] This passage, according to Jehovah's Witnesses, states that blood is equated with the soul. They then claim that since animal blood was of sacred significance to God, human blood has even greater significance, so the eating of human blood was also strictly forbidden by God to Noah. Since all mankind is Noah's family, this same restriction applied to everyone. The penalty for disobeying this rule was clarified when God said, "I shall certainly set my face against the soul that is eating the blood, and I shall indeed cut him off from among his people."[9] Jehovah's Witnesses interpret this to mean the loss of eternal life. No exceptions were granted for emergencies. In the days of King Saul some battle-weary, famished soldiers of Israel slaughtered sheep and cattle and "fell to eating along with the blood." This was considered sinning against Jehovah.[10] As far back as the second century, the Romans would try to force Christians to consume blood, sometimes disguised in the form of sausage. When the Christians refused, it often cost them their lives, analogous to dying secondary to refusing blood transfusion today. However, the issue is whether or not there is a difference between ingesting blood and receiving a transfusion.

ATTITUDE TOWARD TRANSFUSIONS

Obviously, the Bible never specifically mentions blood transfusion, so why is that prohibited? Jehovah's Witnesses believe that blood transfusions were covered in principle. They note the command that Christians "keep abstaining. . . from blood"[11] means that blood should not be accepted orally, parenterally, or otherwise. They draw the analogy that if a physician says one will have a dangerous reaction to an oral medication, one should not take it by vein. The same pertains to blood: God forbids it. According to Psalm 19, "The judicial decisions of Jehovah are true; they have proved altogether righteous. . . In the keeping of them there is a large reward."[12] The reward for refusing blood was specified by Jesus when He said, "I am the resurrection, and the life. He that exercises faith in me, even though he dies, will come to life."[13] Jehovah's Witnesses faithfully believe this and will allow themselves or their children to die before consenting to a transfusion. They are not just paying lip service; they firmly believe it. Nor do they want anyone else, whether a judge, administrator, or physician, to reverse this decision.

Does this refusal to accept blood constitute suicide? Jehovah's Witnesses very much want to live, but not through the use of blood transfusions. In fact, they welcome all other conventional forms of medical therapy. According to Professor Robert M. Byrn, "Rejection of lifesaving therapy and attempted suicide are, and should be, as different in law as the proverbial apples and oranges."[14] Jehovah's Witnesses point out that the average Jehovah's Witness is no more likely to die of hemorrhage than from a

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motor vehicle accident or various forms of cancer or heart disease. Yet few seriously claim that driving a car or smoking is actively suicidal, or that these activities justify the usurpation of one's rights by the courts or by physicians. It is not unusual for people to die for their beliefs. It is even expected behavior in some circumstances. Therefore, in dealing with Jehovah's Witnesses the physician should take them seriously when they say no to transfusions. They will not change their minds just because their physician recommends that they do so. Herein lies the conflict between the patient's rights based on religious convictions, and the physician's responsibilities based on years of training and the oath taken to preserve life.

In addition to the ethical dilemma, there is also the issue of liability.

LEGAL ASPECTS

Often there is sufficient time for the physician to discuss with the patient whether a transfusion is indicated and what alternatives exist. Some alternatives include autologous transfusion, autotransfusion, and the use of blood substitutes. Currently no consensus exists regarding the acceptability of these alternatives among Jehovah's Witnesses. If satisfactory, however, their use may eliminate potential conflict.

If a patient will likely require transfusion, and alternatives are either unavailable or unacceptable, the physician should be aware of legal precedents that may influence his decision whether or not to authorize transfusion without consent.

Justice Louis D. Brandeis was in the minority when he wrote, "The makers of our constitution sought to protect Americans in their beliefs, their thoughts, their emotions and their sensations. They conferred as against the government the right to be let alone the most comprehensive of rights and the right most valued by civilized man."[15] In support 36 years later, Justice Warren Burger added, "Nothing in this utterance suggests that Justice Brandeis thought an individual possessed these rights only as to sensible thought, reasonable emotions or well-founded sensations. . . he intended to include even absurd ideas which do not conform, such as refusing medical treatment even at great risk."[16]

In viewing this issue from a first amendment standpoint, the strength of one's convictions, one's competency, and the interests of the state all become important. In *In re Brooks Estate* a competent patient refused to consent to blood transfusion on religious grounds. The Illinois Supreme Court supported her, saying, "Even though we may consider a patient's beliefs unwise, foolish or ridiculous, in the absence of an overriding danger to society, we may not permit interference therewith in the form of conservatorship established in the waning hours of her life for the sole purpose of compelling her to accept medical treatment forbidden by her religious principles and previously refused by her with full knowledge of the probable consequences."[17]

If there is a question of competency a transfusion can be authorized despite the patient's objection.[16] Even if the patient is competent, he may be forced to accept the transfusion if the state can demonstrate a compelling, overriding interest. In *In the Matter of Melideo* the court ruled that promoting the welfare of children justifies compulsory transfusion in pregnant women and mothers of young children.[18] The court took this issue a step further in *In re Osborne* where it was determined that a male Jehovah's Witness could be compelled to receive a transfusion if the loss of his income could lead his offspring to become wards of the state.[19]

Therefore, based on first amendment protection, a competent adult with no dependents probably cannot be compelled to receive a blood transfusion against his religious convictions if he makes these beliefs known to the physician. The physician must use his judgment and may order a transfusion if the patient is not a competent adult, has dependents, is pregnant, or there is reasonable doubt about the strength of the patient's religious convictions. The patient or his family should be informed in advance of a pending transfusion, rather than finding out after the fact. When time permits, the patient may elect to have his care transferred to another physician.

A study of Jehovah's Witnesses revealed that two-thirds would bring suit if transfused against their will.[1] While statistically litigation is more a threat than reality since such lawsuits are rare, the physician must realize that he could be sued whether or not he compels transfusion. Following the above guidelines can help prevent misunderstandings which may destroy the physician-patient relationship and lead to legal difficulties.

CONCLUSIONS

Physicians whose patient populations may include Jehovah's Witnesses should understand that Jehovah's Witnesses sincerely believe God forbids their receiving blood products. They are not suicidal but neither are they likely to accept transfusions to avoid death. In some circumstances, a physician may legally authorize transfusion of a Jehovah's Witness regardless of his or her convictions. However, the physician should make this decision in advance and so notify the patient. Alternatives to transfusions exist and may be acceptable to the patient.

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A cluster of cases of penicillinase-producing *Neisseria gonorrhoeae* in an adolescent detention center

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Beta lactamase (penicillinase)-producing *Neisseria gonorrhoeae* (PPNG) have been common in the Far East but are relatively rare in the US.[1,2] In New York City, however, the number of cases of PPNG reported to the Department of Health of the City of New York has been increasing steadily over the past several years.[3] As a result, the percentage of gonorrhea cases attributable to PPNG rose dramatically, from 1.33% (67 of 5,034 gonorrhea cases) in January 1985 to 9.8% (391 of 3,993 cases) in November 1986.

In the New York City correctional facilities on Rikers Island, only a few cases of PPNG appeared (2% of positive cultures in February 1985; 3.5% in December 1985) until June 1986. In that month, there were ten cases (8%). Thereafter, the monthly incidence of PPNG began to rise. In October 1986, there were 15 cases of PPNG; in December there were 18 cases. The proportion of pencillinase-producing gonorrhea isolates had risen to 14.3% by December 1986.

At the Adolescent Reception and Detention Center (ARDC) on Rikers Island, about 1,200 to 1,500 recently arrested adolescent and adult males are admitted each month; 50% stay for less than three weeks. The total inmate population is 1,700. Inmates are predominantly from inner-city neighborhoods and most have limited contact with medical care providers when not incarcerated. They are therefore at high risk for diseases of many sorts, including sexually transmitted infections. Sexually transmitted diseases among new admissions are a concern because, although sexual activity is not permitted in the jail, it cannot be completely eliminated. The concurrence of these conditions—the high PPNG prevalence in New York, the high volume of new admissions to Rikers Island, the high risk of sexually transmitted diseases among new admissions, and the existence of sexual encounters between inmates—is conducive to outbreaks of PPNG among inmates.

A cluster of PPNG cases among adolescent detainees was reported in 1983,[4] but all individuals involved had worked as prostitutes and were infected before entering incarceration. We present here a report of a PPNG outbreak that occurred within ARDC in December of 1986.

CASE REPORT

Information on subjects was obtained by review of medical charts in the ARDC clinic and from Department of Correction (DOC) records.

On December 26, 1986, an 18-year-old inmate who was an admitted homosexual presented at the ARDC clinic with a pharyngeal exudate. A culture taken of his pharyngeal exudate grew PPNG. Five other inmates presented at ARDC clinic on December 26. Three had a purulent penile discharge; cultures of urethral swabs from all three grew PPNG. The other two inmates complained of "gonorrhea" but had no symptoms on physical examination; no swab was taken for culture from either one.

Specimens were treated according to the standard procedure of the Rikers Island Health Services laboratory: gonorrhea cultures were plated on Martin-Lewis-Jembec medium and incubated at 37°C. Plates were examined after 24 and 48 hours. *Neisseria gonorrhoeae* are identified by Gram stain morphology plus Phadebact (Pharmacia; Piscataway, NJ) coagglutination assay; if the latter is negative, identification is made by differential oxidation of sugars in cystine tryptic medium (Gibco; Madison, Wisc). Beta lactamase production was assayed using beta lactam reagent disks (Marion Scientific; Kansas City, Mo).

All four inmates with PPNG—the index case and the three with urethral gonorrhea—were treated with spectinomycin (2 g, intramuscularly) and vibramycin (100 mg, orally, twice a day, for seven days). Subsequent cultures were negative.

Evidence that these four cases might be linked was sought from medical charts and DOC records. All four culture-positive inmates had been housed in the same unit in ARDC up to December 26. In this unit, one inmate is housed per cell, but inmates are not locked in during the day; all share a common dayroom, shower, and toilet facilities.

A mental health clinic report from December 28 quoted the index case as alleging that he had been forced to have receptive oral sex on three different occasions by six different inmates. Three of the inmates named were the three with urethral PPNG infections. According to laboratory records, three additional inmates, who had also been implicated in the sexual incidents, had been called to the clinic on January 2, 1987, so that urethral swabs could be taken for culture. All cultures were negative.

DISCUSSION

We were unable to prove that the four PPNG cases represented a chain of transmission. Auxotyping, which could have demonstrated that the isolates were identical, was not performed on the PPNG cultures. The remaining evidence, although persuasive, is circumstantial.

We hypothesize that the index case transmitted the infection to the urethrae of at least three inmates with whom he had receptive oral sex. The suspicion of two unaffected inmates that they might have gonorrhea suggests that a sexual incident involving several residents of the housing area had indeed occurred.

No new cases occurred, as evidenced

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by a failure to culture *N gonorrhoeae* from contacts of the index case (three inmates cultured one week later) and the absence of PPNG isolates from ARDC in the subsequent three weeks.

The probability that the oropharynx will serve as a source for transmission of *Neisseria* sp is disputed. However, data collected in the late 1970s and among prostitutes and US servicemen in the Philippines in 1981 suggest that this mode of transmission does occur,[5,6] and Hutt and Judson[7] supported the likelihood of transmission from the oropharynx in their more recent work.

This outbreak demonstrates the potential for spread of infectious organisms in incarcerated populations. Sexual activity, although not condoned, does occur in these settings. Treatment of infected individuals with appropriate antibiotic therapy is imperative. It is notable that spectinomycin, 2 g intramuscularly, although appropriate for urethral gonorrhea in an area with high PPNG incidence, is not optimal for oropharyngeal infection.[7] Rikers Island Health Services has since amended its sexually transmitted diseases treatment protocol to call for use of 250 mg of ceftriaxone, intramuscularly.

Where PPNG prevalence is high and when follow-up is uncertain because of either frequent turnover of institutionalized persons or difficulty in tracing frequently transferred individuals, it is a mistake to wait for culture results to demonstrate whether a gonorrhea infection is penicillin-sensitive; all suspected cases of gonorrhea should be treated as if they were PPNG. The Centers for Disease Control has recommended that this be done whenever more than 3% of strains are PPNG.[8] The Department of Health of the City of New York has recommended that all cases of gonorrhea be treated with spectinomycin (2 g) or ceftriaxone (250 mg) without waiting for culture results. This is becoming increasingly imperative as the PPNG prevalence in the general population rises, as more individuals enter incarceration, and as turnover and transfer of inmates continue at high rates. It is also important to educate institutionalized individuals about the potential for spread of sexually transmitted diseases and the means to prevent transmission.

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Recovery of beta-cell function postpartum in a patient with insulin-dependent diabetes mellitus

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The phenomenon of temporary postpartum decrease in insulin requirements dramatically below prepregnancy levels has been observed frequently, but is not well understood. We investigated parameters of endogenous beta-cell function (C-peptide and proinsulin) in connection with postpartum insulin requirements in a patient with insulin-dependent diabetes mellitus (IDDM).

CASE REPORT

The patient was a 17-year-old woman with onset of IDDM at the age of 12 years. She became pregnant at the age of 16. She had previously received treatment in a hospital for ketoacidosis seven times in a four-year period. Immediately prior to pregnancy she was using NPH and regular insulins in a split, mixed regimen of 52 units per day.

She required a hospital stay for glycemic control once in the first trimester of pregnancy and again in the third trimester. Her hemoglobin A_{1c} was 6.6% in the first trimester, averaged 5.1% in the second trimester, and averaged 5.9% in the third trimester (normal, <6.2%). Her maximum insulin dose during pregnancy was 137 units per day, seven weeks prior to delivery. Subsequent episodes of hypoglycemia led to decreases in the patient's insulin dose, so that on admission for delivery, she was using 104 units of insulin per day.

The patient delivered a 6 lb, 2 oz boy, with a five-minute Apgar score of nine, after 37 weeks of gestation. Oxytocin was used for an otherwise normal vaginal delivery without complications. The infant's neonatal course was unremarkable.

Following delivery, the patient was given regular insulin coverage based on capillary blood glucose determinations using Chemstrips bG and the Accu-Chek bG blood glucose monitor. On day three postpartum, she received five units of NPH insulin before breakfast and required five units of regular insulin coverage.

The patient's postpartum hospital course was marked by six separate episodes of hypoglycemia in eight days, usually occurring before her morning NPH insulin dose and associated with symptoms. During this period she received an average of 15 units of insulin per day.

On day five postpartum, the patient received 17 units of insulin, and her capillary blood glucose ranged from 220 to 280 mg% without hypoglycemia. That morning her postprandial serum C-peptide level was 4.0 ng/dL (normal fasting, 0.5-4.2 ng/dL) and

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her postprandial serum proinsulin level was 6.2 ng/dL (normal fasting, less than 0.5 ng/dL). On her ninth postpartum day she was discharged from the hospital taking eight units of NPH and three units of regular insulin in the morning and three units of regular insulin in the afternoon.

Following discharge, the patient's insulin requirement increased quickly. On postpartum day 26 her insulin requirement was 42 units per day, maintaining her blood glucose in the 100–200 mg% range. On that day, her serum postprandial C-peptide was 0.6 ng/dL and her serum proinsulin was 3.9 ng/dL. By postpartum day 106, her serum C-peptide was undetectable, her serum proinsulin was 3.4 ng/dL, and her insulin requirement was up to 50 units per day (Fig 1).

The patient breast-fed her baby for the first two weeks postpartum.

Proinsulin was measured by radioimmunoassay at the Nichols Institute (Los Angeles, Calif), using an immunoprecipitation technique to avoid interference with insulin antibodies. C-peptide was also measured by radioimmunoassay at Roche Laboratories (Raritan, NJ) or Damon Laboratories (Trevose, Penn), following precipitation of insulin antibodies.

DISCUSSION

Spontaneous remission of IDDM is observed in 30% of young patients and invariably occurs within the first year after diagnosis.[1] Partial and full remissions have also been induced within one year of diagnosis using cyclosporine,[2,3] an inhibitor of the cell-mediated immune system.

The phenomenon of dramatic postpartum decrease in insulin requirements has frequently been noted.[4-6] Patients with IDDM may require no insulin for 48 hours postpartum after receiving more than 100 units per day prior to delivery.[4] A 25% decrease in insulin requirements during the last weeks of pregnancy is not unusual, but remains unexplained.[6]

Although there is speculation in the literature about the roles of placental factors and breast-feeding, we could find only one study that examined postpartum decrease in insulin requirements.[5] In this study, a resumption of prepregnancy insulin requirements on the third postpartum day, in two patients, corresponded to a return of normal growth hormone response to insulin-induced hypoglycemia. In three other subjects, growth hormone levels failed to respond to insulin-induced hypoglycemia on repeated testing up to six to eight weeks postpartum. Insulin requirements in these patients were not reported. The presence or absence of lactation was of no correlative significance.

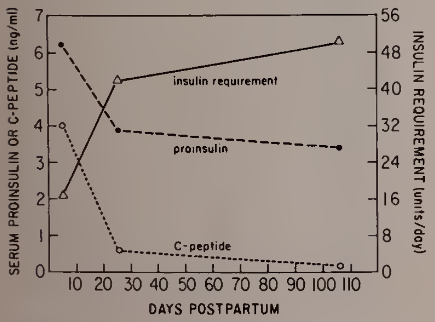

FIGURE 1. Serum C-peptide, serum proinsulin, and insulin requirements postpartum.

In the patient presented here, there was already evidence of a decreasing insulin requirement during a seven-week period prior to delivery, when insulin requirements dropped from 137 units to 104 units per day. This calls into question the role of placental factors and lactation. Also, this patient stopped breast-feeding two weeks postpartum, yet her C-peptide secretion and somewhat lower insulin requirement persisted for several weeks thereafter.

Endogenous insulin production, as measured by C-peptide, has been followed during pregnancy and postpartum in patients with IDDM in several studies.[7-9] In one group of patients with initial fasting C-peptide less than 0.25 ng/dL, a significant increase was observed during weeks 32–33 of pregnancy. Postpartum, however, there was no significant change in C-peptide compared to initial C-peptide values. There was no mention of dramatic postpartum decreases in insulin requirements in patients in this[7] or the other studies.[8,9]

The patient with IDDM presented here is the first, to our knowledge, in whom postpartum diminution of insulin requirements was correlated with increases in C-peptide and proinsulin levels. Five years after the onset of IDDM, with multiple hospital admissions for diabetic ketoacidosis, this was clearly not anticipated.[1]

Because C-peptide is secreted from the pancreas with insulin in equimolar quantities, it is commonly used now to assess endogenous beta-cell function in patients receiving exogenous insulin.[2,3] This patient's C-peptide level was within normal limits on day five postpartum and decreased to an undetectable level by day 106. The proinsulin level showed a similar trend, while the patient's insulin requirement was correspondingly low postpartum and increased to her prepregnancy level by day 106. Thus, after five years of IDDM, this patient demonstrated a temporary resumption of significant beta-cell function concurrent with a partial remission of clinical diabetes.

It is generally assumed that IDDM involves an irreversible destruction of the pancreatic beta cells. An autopsy study of the pancreas showed that 50% of young patients with IDDM whose duration of disease was between six months and ten years had beta cells present, whereas 18% of patients with IDDM for more than ten years had beta cells present.[10] A study of residual beta-cell function, using C-peptide levels, included patients with IDDM who had onset of disease between ten and 20 years of age. C-peptide immunoreactivity was present in two thirds of patients who had had diabetes for less than five years, but was detectable in only 15% of patients with diabetes for five to ten years.[11] Thus, the notion that pancreatic beta cells in IDDM have been totally and irreversibly destroyed should be modified: Pancreatic beta cell function commonly persists, to some degree, even years after the onset of disease.

The mechanism underlying resumption of beta-cell function in this patient is of considerable interest and invites speculation. It is known that pregnancy is a state of suppressed immune function, after which autoimmune diseases such as thyroiditis, rheumatoid arthritis, and systemic lupus erythematosis may flare. Much evidence, including the cyclosporine trials, implicates the immune system as a major factor in the pathogenesis of IDDM.[2,3]

One can imagine pancreatic beta cells of a patient with IDDM undergoing continuous autoimmune destruction, while simultaneously there is a second process of repair or regeneration. In the patient with clinical IDDM the destructive process predominates over the regenerative one. With a change in immune status, as occurs during pregnancy, the balance of forces may shift in favor of repair or regeneration, allowing some recovery of beta-cell function. The metabolic demands of pregnancy, including the development of increasing insulin resistance,[6] easily overwhelm the improved beta-cell function, though in some patients a decreased insulin requirement may be observed several weeks prior to delivery. Postpartum, when the metabolic burden of pregnancy is lifted, this resumption of beta-cell function may be uncovered. Then the immune system reverts to its prepregnancy state, autoimmune destruction of the pancreas predominates, and insulin requirements return to their prepregnancy levels.

Another possibility that may explain the decreased insulin requirements observed in these patients, peripartum, is the excellent glucose control that occurs during pregnancy. Studies have shown that strict glucose control improves beta-cell function in IDDM.[12] Insulin resistance is also ameliorated or reversed in IDDM that is adequately treated with insulin.[13] This patient had numerous episodes of ketoacidosis prior to pregnancy, but was maintained nearly euglycemic throughout most of her pregnancy. Thus, tight control of glucose during pregnancy may have contributed to recovery of beta-cell function and decreased insulin resistance in the third trimester and postpartum.

Clarification of the underlying mechanism for these phenomena requires further study and may provide insights useful for treatment of insulin-dependent diabetes mellitus in the nonpregnant patient. Meanwhile, clinicians should be alert to the occurrence of postpartum decreased insulin requirements in type I diabetes and should monitor their patients' blood glucose closely to avoid hypoglycemia.

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Löffler syndrome and ancylostomiasis brasiliensis

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Ancylostomiasis brasiliensis is widespread throughout the world. A number of distinct but as yet unidentified and closely related species are probably involved. The disorder is endemic in the southern part of the United States,[1] but sporadic cases have been reported as far north as Massachusetts. It is present along coastal regions of Texas, Mexico, Central America, Colombia, Venezuela, the Guianas, and Brazil. It is also found in many parts of tropical Africa and Asia. In an era of increased worldwide travel, physicians unfamiliar with this disorder are often required to treat patients with ancylostomiasis and its complications. Löffler syndrome is uncommon with this disease.

CASE REPORT

A graduate student conducting field studies of the biology of sea turtles in the West Indies, the coastal areas of Venezuela, and the Guianas returned home after several months and sought medical care for intensive itching throughout his body and "creeping eruptions." Local inhabitants told him to try to burn the eruptions out with cigarettes. There were several burn marks over his skin. Physical examination revealed erythematous, serpiginous, and elevated lesions (Fig 1). There were many scratch marks, but no signs of local infection. There was moderate eosinophilia and radiographic evidence of some pulmonary infiltrates suggesting Löffler syndrome (Fig 2). Differential diagnostic considerations included infection with *Strongyloides myopotami* (nutria strongyloid),[2] *Gastrophilus* or *Hypoderma* species (horse botfly or cattle warble fly). *Ancylostoma brasiliense* infection is very common in the areas the patient visited and is also most likely to produce Löffler syndrome. *Ancylostoma canium* (dog hookworm) was not considered since the lesions produced by this parasite usually disappear in about two weeks. The diagnosis of ancylostomiasis was established by microscopically examining the parasites. The patient was treated with thiabendazole. Stone and Mullens[3] reported successful treatment of ancylostomiasis brasiliensis with oral thiabendazole and Eyser[4] recommended local therapy. We decided to initiate local therapy and to consider sys-

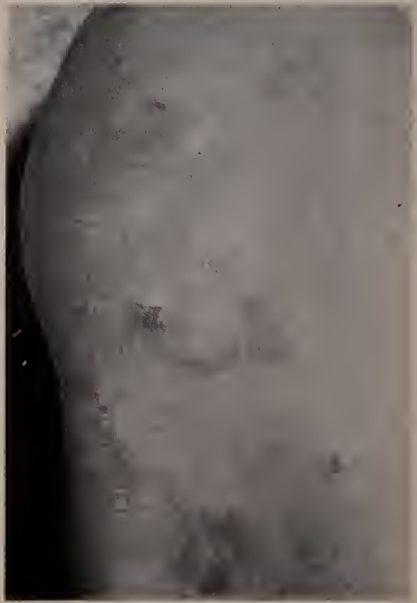

FIGURE 1. Erythematous, serpiginous, and elevated skin lesions.

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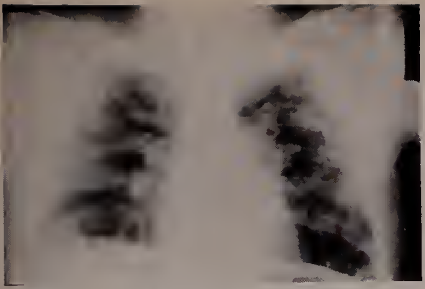

FIGURE 2. Anteroposterior chest film showing infiltrates consistent with Löffler syndrome.

temic therapy only if the treatment failed.

However, no thiabendazole ointment is available in this country. Therefore, a thiabendazole cream in a 0.1% dexamethasone cream was made by incorporating 10 mL of a pediatric thiabendazole suspension (containing 1 g active ingredient) into 20 g of cream. The patient was told to apply this cream to all lesions three times daily and to superimpose a polyethylene film wherever possible to occlude the medication. The patient returned two weeks later with all skin lesions completely cleared. The Löffler syndrome also disappeared. The chest film was normal. The patient reported that itching disappeared within an hour after applying the ointment.

DISCUSSION

Creeping eruption is treated in many parts of the world by trying to burn the lesions out with cigarette butts or glowing cinders. This results in much scarring and is highly unsatisfactory therapy. Probably the medical equivalent of this type of treatment is an attempt to freeze the area with ethylchloride spray or carbon dioxide snow. This is particularly unsatisfactory if numerous lesions are present. The oral administration of promethazine hydrochloride has been tried, but oral thiabendazole is considered the best therapy at present. Usually 25 mg/kg is given two times per day for two successive days and repeated in about a week.

Thiabendazole is available as an oral medication, including a pediatric suspension. At present, no form is available for local application in the United States. It appears worthwhile to consider making available local preparations, possibly under the "orphan drug" regulations. It may be of interest to develop ointments and creams with and without dexamethasone. This material is occasionally applied with dimethylsulfoxide and intracutaneously with jet injectors.

The most important preventive measure is to deworm domestic cats and dogs periodically. In these animals, the worm travels to the heart, lungs, and gastrointestinal tract, when it matures, reproduces, and passes eggs in the stool, thereby contaminating soil. Under adequate conditions of temperature and humidity infective forms develop in the soil. They penetrate the skin of man and produce typical creeping eruptions. The life cycle of the European dog hookworm (*Uncinaria stenocephala*) is similar although the lesions produced in the skin of patients are usually less extensive. Our previous experience suggests that the response to therapy of *Ancylostoma brasiliense*, *Uncinaria stenocephala*, and *Ancylostoma caninum* is similar.

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Papulonecrotic tuberculid

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In 1896 Darier[1] reported that patients with tuberculids have the following key findings: a positive tuberculin skin test, tuberculous involvement of lymph nodes and/or internal viscera, absence of tubercle bacilli from skin biopsy and culture, and skin lesions that heal upon the remission of the tuberculous infection. In the modern era, rapid resolution of the skin lesions invariably follows the institution of antituberculous antibiotic therapy.

Papulonecrotic tuberculid is a rare cutaneous manifestation of tuberculosis in the United States. It is most commonly seen in populations with a high prevalence of tuberculosis and usually affects children or young adults; in the United States it usually affects recent immigrants. Papulonecrotic tuberculid is a symmetric eruption of crops of papules, often on the extensor surfaces of the extremities, that heals with necrosis and scar formation.

We report a case of papulonecrotic tuberculid in a diabetic New Yorker with pulmonary tuberculosis.

CASE REPORT

A 55-year-old man suffering from alcoholism and insulin-dependent diabetes mellitus was evaluated for a three-week history of a slightly pruritic, nontender rash. He had had fever, dyspnea, and a productive cough for two months. He had a history of intravenous heroin abuse more than 25 years earlier. The history also included alcohol-induced pancreatitis, hepatitis A and B, and a total hip replacement for degenerative joint disease, but none of these processes were currently active. The patient denied arthritis. He was born in New York City and had always lived in the United States. There was no known history of prior purified protein derivative (PPD) testing.

Physical examination revealed numerous 2- to 4-mm diameter erythematous papules predominantly on the shins and the dorsa of the feet (Fig 1). Scattered lesions were noted on the extensor surfaces of the patient's forearms, arms, and anterior chest. Many of the papules had necrotic centers or focal

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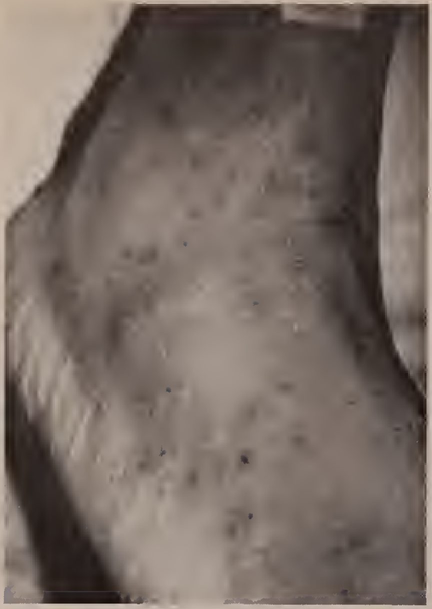

FIGURE 1. Papules on the foot of a patient with papulonecrotic tuberculid.

hemorrhagic crusts.

A chest film revealed a right upper lobe infiltrate and thickening of the left pleura. A sputum examination for acid-fast bacilli was positive and culture of sputum later grew *Mycobacterium tuberculosis*. Sputum culture for fungi and all blood cultures were negative. A PPD was positive as were controls placed to rule out anergy. Serologic testing for human immunodeficiency virus (HIV) was refused by the patient.

Skin biopsy showed edema and a neutrophilic infiltrate in the papillary dermis (Fig 2). There was focal alteration of the subjacent reticular dermis with a mononuclear perivascular infiltrate. Other specimens showed focal necrosis of the epidermis with neutrophils in the ulcer base. No mycobacteria were detected by Fite stain of the skin specimens. Periodic acid-Schiff stain of the skin biopsy did not reveal fungi.

Treatment with isoniazid (300 mg/day), rifampin (600 mg/day), and ethambutol (1.2 g/day) was begun and the skin lesions resolved within two weeks.

DISCUSSION

Papulonecrotic tuberculid has been reported rarely in the English language medical literature during the past two decades. Case reports have described the entity in immigrant populations (Southeast Asia) and less developed countries where tuberculosis is more prevalent.[2-4] In 1974, Morrison and Fourie[5] reported 91 cases of papulonecrotic tuberculid occurring in South Africa during a 17-year period. Two thirds of the patients were younger than 30 years of age. In approximately one third of their cases, a tuberculous focus was determined. Of these, adenopathy that was often combined with scrofuloderma was more common than a known pulmonary focus.

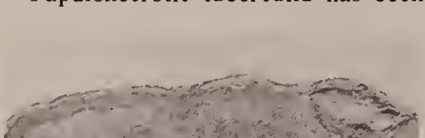

FIGURE 2. Biopsy of papule from patient's foot revealing foci of dermal edema and collagen change and a mixed-cell infiltrate (hematoxylin-eosin stain; original magnification × 4).

It has been proposed that the papulonecrotic tuberculid represents an Arthus reaction followed by a delayed, hypersensitivity reaction to mycobacteria.[5] Bacilli from a tuberculous focus presumably seed the systemic circulation and come to reside in the microcirculation of the skin in an opsonized form. Phagocytosis of opsonized bacilli and resultant destruction of neutrophils releases proteolytic enzymes with ensuing necrosis and destruction of vessels. The consequent mononuclear cell response presumably then destroys the bacilli.

Papulonecrotic tuberculid may perhaps be thought of as evidence of a normal, protective immunologic response. This is in contradistinction to lupus vulgaris which is a postprimary form of skin tuberculosis in individuals with only moderate immunity. Unlike papulonecrotic tuberculid, untreated lupus vulgaris usually is progressive and complete healing is only rarely observed.[6] Pulmonary tuberculosis associated with lupus vulgaris actually exhibits a higher than expected mortality rate.[6] Like papulonecrotic tuberculid, lupus vulgaris develops from noncutaneous tuberculous infection and in about one third of cases is preceded by scrofuloderma.

Other mycobacterial organisms besides *M tuberculosis* can initiate papulonecrotic tuberculids. A case of papulonecrotic tuberculid secondary to *Mycobacterium bovis* flared each time a patient with an *M bovis* lymph node infection received prednisone for temporal arteritis.[7] The papulonecrotic tuberculid also may occur following vaccination with bacillus Calmette-Guerin.[8,9]

Papulonecrotic tuberculid is uncommon even in populations with a high prevalence of tuberculosis. It occurred in ten of 222 cases of tuberculosis in South Vietnam[2] and was diagnosed only nine times during a 15-year period in the clinic at the Osaka University School of Medicine in Japan.[4]

In recent reports, Wilson-Jones and Winkelmann in 1986[10] described the papulonecrotic tuberculid in 12 young patients with symmetric, scattered "papulopustular" necrotic lesions of the extremities. All had strongly positive Mantoux reactions and prompt resolution of the lesions with antituberculous therapy; eight of the 12 had documented tuberculous foci. Of the 12 patients, ten were immigrants from continents where tuberculosis was much more prevalent than in England, where the reports originated. A case report from Israel[11] described papulonecrotic tuberculid of the glans penis of a Jew of Moroccan descent who had emigrated to Israel. He exhibited a strongly positive tuberculin test and a Ghon complex on chest film, and his skin eruption cleared completely while on antituberculous medication. Papulonecrotic tuberculid has rarely been reported in Americans.[12]

Classically, tuberculids were said to contain tuberculoid granulomas. However, reports suggest that caseating granulomas are uncommon; they were found in only four of the 91 cases presented by Morrison and Fourie.[5] The most suggestive histopathologic finding for diagnosis of the papulonecrotic tuberculid is a wedge-shaped area of necrosis with the broad base toward the epidermis found in older lesions,[2-5,7-10] but earlier lesions demonstrate less specific pathologic changes ranging from a nonspecific infiltrate[3,4] or a mild perivascular infiltrate[9] to true fibrinoid necrosis and thrombotic occlusion of individual vessels.[9]

Morrison and Fourie[5] suggested that papulonecrotic tuberculids begin as Arthus reactions and then proceed as delayed-type hypersensitivity reactions. This would be compatible with early lesions showing a leukocytoclastic vasculitis followed by a perivascular infiltrate of mononuclear cells and subsequent necrosis with epithelioid giant cell formation. Therefore, on histologic grounds alone, the papulonecrotic tuberculid has a wide differential diagnosis that reflects the evolution of the underlying process.

Classically, papulonecrotic tuberculid occurs in childhood or adolescence. With the concomitant rise in cases of tuberculosis and AIDS, we may encounter more cases of papulonecrotic tuberculid in older, susceptible patients. The histopathology of this tuberculid may be so atypical for mycobacterial infection that the correct diagnosis could be overlooked unless clinical correlation is properly stressed. Despite its rare occurrence in countries with a low prevalence of tuberculosis, the papulonecrotic tuberculid should be considered in the differential diagnosis of symmetric chronic papulopustular necrotic lesions on the extremities. Because the incidence of mycobacterial infections in the United States is increasing,[13,14] any such eruption on the extremities may herald an undiagnosed tuberculous focus. Likewise, the possibility of papulonecrotic tuberculid caused by infection with atypical mycobacteria should be considered and appropriately investigated in these patients.

Papulonecrotic tuberculid will resolve promptly after institution of appropriate medical therapy. The current recommended regimens for *M tuberculosis* infection include daily isoniazid, 10 mg/kg (maximum 300 mg daily), and rifampin, 10 mg/kg (maximum 600 mg daily), for nine months or, alternatively, six months of isoniazid and rifampin in the same doses but supplemented with pyrazinamide, 15–30 mg/kg daily, during the initial two months.[15]

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PREGNANCY AND DIABETES; REPORT OF CASES

Now that death and even marked ill health has been so definitely postponed in well cared for, severe diabetic patients by the use of insulin, and that in these patients physiological processes appear to proceed in quite a normal manner, the advisability for a diabetic woman to become pregnant becomes a question demanding consideration. These individuals who pass in a crowd as being healthy, are not, even though their metabolic defect appears to be stayed; they are still on a diet that is not normal, though it may be sufficient; there may be a disturbance in a vital system, especially the vascular, which is the result of diabetic toxaemia, and they may be more susceptible to certain infections.

Prior to the use of insulin, the occurrence of pregnancy, especially in a severe case of diabetes, was looked upon with alarm; we had not the tools to cope with the situation, and so these patients usually died in coma. With the use of insulin it was only reasonable to expect a better prognosis in such cases. Fortunately the physician does not bear this responsibility in its entirety because pregnancy more often occurs without there having been any previous consultation as to its advisability. In the series of cases reported below we were consulted in but one instance, when consent was given, on condition that the patient be under hospital supervision at least for the last month of gestation. In three of the cases the termination of pregnancy was urged but was refused.

J. Whitridge Williams reports that in his group of sixty-six cases of pregnancy in diabetic patients, twenty-seven per cent died during delivery, and twenty-three per cent succumbed within two years following the pregnancy. Also about one-third aborted and one-third of the foetuses were born dead. DeLee's experience was rather similar. This is such a gloomy state of affairs that it is no wonder that pregnancy in diabetics has been frowned upon. Fortunately, however, this condition adjusted itself to some extent, as the large majority of diabetic women became sterile. This is commented upon by DeLee who quotes 114 cases collected by Lecorche in whom there were but seven gestations. Insulin, however, has changed this condition, as the majority of amenorrhoeic patients have a return of their menstruation and become fertile. This has recently been discussed by Fitz and Murphy who have reported one case. In our group of seven cases, three had amenorrhoea, with a return of menstruation after prolonged insulin treatment. Joslin, who has had such a complete experience in diabetes, gave his full consent for pregnancy in but one case before insulin. He believes that pregnant women are so unstable that acidosis may come on in a few hours, thereby making it unsafe. . . .

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(*NY State J Med* 1923; 23:351–358)

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Utilizing former tuberculosis sanatoria as hospitals for the treatment of AIDS

TO THE EDITOR: I was very interested in K. W. Wright's article, "A history of the Onondaga Sanatorium for the Treatment of Tuberculosis," in the March issue of the *Journal*.[1]

My interest in sanatoria dates from my residency in 1946 at the Bedford-Hills Sanatorium, which was under the direction of Dr Max Pinner of Montefiore Hospital. All we had available then to treat active tuberculosis patients was time-honored bed rest, plus pneumothorax, pneumoperitoneum, and thoracoplasty. The surgical patients were sent to Montefiore Hospital in the Bronx. It was not until streptomycin was found to be effective in treating tuberculosis that a new era of therapy through medication dawned. The effectiveness of antituberculosis drugs resulted in the closing of sanatoria all over the country.

It strikes me that we now have an analogous situation with the acquired immunodeficiency syndrome (AIDS) epidemic. An incurable disease is rapidly spreading. Advanced cases are treated, more or less reluctantly, in general hospitals, to the discomfort of the staff and the financial loss of the institution. My suggestion is that the old tuberculosis sanatoria, with specially trained staff, be reopened as AIDS hospitals. This would effectively isolate infectious cases, a first principle of public health, and humanely treat seriously ill patients.

It is only a question of time until a vaccine, chemotherapeutic agent, or both, will be found to treat AIDS. However, neither is available at present. Therefore the suggestion to hospitalize AIDS patients in reopened tuberculosis sanatoria seems an eminently practical one.

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Chlamydial infections in children and adolescents

TO THE EDITOR: The recent article by Goldenring et al[1] raises some important points about the significance of rectovaginal chlamydial infections in children and adolescents. However, some caution needs to be exercised about this significance with regard to sexual abuse and the use of the nonculture antigen detection methods (MicroTrak and Chlamydiazyme) in prepubertal children. Although rectal and vaginal infection with *Chlamydia trachomatis* can be acquired after sexual assault,[2] persistent perinatally acquired infection may persist beyond 18 months.[3] We do not know the endpoint. Asymptomatic vaginal infection lasting at least three years has been documented in some children after sexual abuse. Who is to say that it could not occur after birth? Unfortunately we will not have this longitudinal data because we are treating asymptomatically infected infants.

The other potentially very serious problem is the performance of MicroTrak and Chlamydiazyme. Neither test has Food and Drug Administration approval for use with rectogenital specimens from prepubertal children. Evidence has accumulated documenting a significant lack of both sensitivity and specificity when used at these sites in children.[4-6] MicroTrak has been approved for use on rectal specimens from "symptomatic" individuals, which actually refers to adults with proctocolitis. One well-respected laboratory recently studied MicroTrak for rectal specimens from sexually active adults, and although they found the test to be sensitive and specific, they stated that over 7% of the smears contained artifacts close in size and staining to chlamydial elementary bodies.[7] Even with cervical specimens, wide variations in performance can be found from one laboratory to another; there are no mandatory quality controls or proficiency testing.[4] The medical and legal implications of a false-positive MicroTrak or Chlamydiazyme are quite different for a sexually active adolescent than for a nine-year-old boy or girl.

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In reply. I thank Dr Hammerschlag for her letter which allows me to discuss two areas on which we have some disagreement.

First, Dr Hammerschlag raises the question of how long *Chlamydia trachomatis* can be carried after neonatal transfer. While she is correct in saying that we do not have a complete answer, we do know some useful facts. A longitudinal study of newborn vaginal and rectal chlamydial carriage clearly indicates that in the majority of such cases, chlamydia disappears by the age of one and a half years.[1] Although cases are now reported where chlamydia has persisted to age three years before spontaneously resolving,[2] it is unlikely that chlamydia is different from other infectious organisms. The carrier rate clearly decreases over time. If chlamydia does persist into preadolescence, this must be an extremely rare event; certainly rarer, in my opinion, than transmission via sexual assault. I suggest that in those less than one and a half years of age the likely mode of transmission is neonatal. There is some difficulty in interpreting the mode of transmission in those one and a half to four years of age. Chlamydia carriage alone is not pathognomonic of sexual abuse and must be corroborated by physical or historical evidence. Sexual abuse is the likely mode of transmission in those over four years of age. It should be possible (as in the case presented[3]) to obtain solid corroborating physical and/or historical evidence of abuse.

I persist in believing what I have said before,[4] that outside of the first year of life, when sexually transmitted diseases (STDs) are encountered in children they have usually been sexually transmitted. I consider chlamydia only slightly more problematic than other STDs in this regard.

Dr Hammerschlag also raises the question of use of direct fluorescent antibody (DFA) testing, as opposed to culturing for *C trachomatis*. In this respect, there are two issues, false positives and false negatives.

As to false positive DFAs, which Dr Hammerschlag has recently reported,[5] I must point out that the cause of the "falseness" is readings by inexperienced microscopists.[6,7] Thus, the problem can be remedied by referring samples which are difficult to interpret for adjudication by more experienced observers, much as one would do with any pathologic sample about which there is some doubt. It should be possible to distinguish cross-fluorescing microorganisms from chlamydia because the patterns of fluorescence are very different to the experienced eye. Since DFA slides provide permanent evidence that can be reviewed by experts, there is no reason why they cannot be used in medicolegal cases.

I do agree with Dr Hammerschlag that the other nonculture method of testing for chlamydia by ELISA cannot be used in sexual abuse cases because there is no permanent evidence which can be reviewed, thus it is not possible to distinguish false from true positives.

As for false negatives, I must agree that cultures are more sensitive than DFA testing,[8] although there is ample evidence that occasionally the DFA is truly positive when the culture is negative.[9-13] My own experience, and that of others,[8] in comparing DFA and culture in children is far better than that cited in Dr Hammerschlag's reference numbers.

At any rate, a greater problem than missing some cases would be missing all cases. In Los Angeles at least one third of our sexual abuse examiners operate in private practice or at small hospitals where facilities for chlamydia culture are not readily available. Chlamydia cultures also cost twice as much as DFAs and are fragile (they require refrigeration during transportation to central laboratories). For these reasons, many examiners do not even attempt to test for chlamydia, which I think both Dr Hammerschlag and I would agree is an error, considering that *C trachomatis* is the most prevalent STD in most areas of the United States.

Unless all sexual abuse evaluations are centralized at large institutions with the capability of doing chlamydia cultures easily and at lower cost, I suggest that DFAs can be used where cultures are not easily available, though the clinician must be aware of the test's limitations.

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A critical area for problem solving and decision making

TO THE EDITOR: If membership is the ultimate driving force to enable an organization to survive and thrive, how do we fuel it? If the headquarters of the Medical Society of the State of New York (MSSNY) at Lake Success is not to become an empty Taj Mahal, how do we solve the problems of membership and what decisions do we make? With a wisdom born of understanding the problems of increasing membership, the leadership of our society has mandated that the new membership committee focus particular attention on women physicians, foreign medical graduates, minorities, young physicians, residents, and medical students.

What are the statistics that underlie those concerns? Whereas the MSSNY's market share among physicians in general, excluding residents, is 47%, certain groups of doctors are less likely to be members. Our market shares of these segments are as follows: women, 28%; foreign medical graduates, 38%; minorities, 32% (estimated); students, 24%; young physicians, 33%. The committee on membership, whose members include representatives of these various constituencies, has worked vigorously to turn this around. Its members have articulated their concerns to a responsive council.

Regrettably, certain suggestions have not been favorably received by the organization, for example, that of differential membership dues for physicians in underserved areas and retired physicians, as well as for physicians serving the military at Camp Drum. This makes it all the more important that we do not ignore other reasonable proposals. We can agree that change is not easy, but sometimes it is necessary if we are to grasp opportunities. A medical society can be entrepreneurial or intrapreneurial in pursuing its "business." We can seek astute investment strategies or develop internal projects for the benefit of the organization. Regardless of the path the MSSNY chooses, a high priority must be placed on membership if we hope to remain a dynamic, progressive organization with vision and a mission.

Expanding membership means reaching out to potential constituencies and offering them tangible reasons for joining and remaining members. The welcome mat must be out in front of every county society as well as the state society.

We have asked members of these underrepresented groups to tell us what they expect of the MSSNY. We are told that they want to be an integral part of the mainstream, with an equal opportunity to be chosen for elected office and as delegates to the American Medical Association. One response is that members must "pay their dues" for a number of years before being selected. This is certainly an understandable contention, but if we limit ourselves in this fashion we risk losing those who can find other areas in which to devote their energies and skills. An intuitive, pragmatic medical society will seek a balance between long-standing, accomplished members and talented newcomers. This mix can certainly enrich our endeavors and provide the soil for creative efforts.

We will continue to pursue the techniques of marketing the MSSNY to members by surveys, focus groups, computer simulations, demographics, psychographics, etc, but among the things that we need to provide is a marketable product. The image and credibility of the organization should appeal to both members and nonmembers. Our concerns for all physicians and all patients must be clearly spelled out. Special targets of opportunity such as those highlighted here must be encouraged in every possible way to join us.

Like many mature organizations, we are at a critical point in our growth curve. Unless we take more steps that will assure continued expansion, we will miss out on the chance to remain a key player in health care. The future is in our hands. How we solve this problem and the decisions we dare to make will say a great deal about our character and our fate as a medical society.

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Patient use of metered dose inhalers

TO THE EDITOR: Metered dose inhalers (MDI) may be the preferred delivery method for beta adrenergic medications.[1] Unfortunately, the proper technique of using MDI is not mastered by all patients.[2] Spacer devices have been presented as a solution to this problem; Newhouse and Dolovich[1] state that ". . . reliable aerosol delivery by metered-dose inhalers can be ensured with use of an inhalation aid that attaches to the inhaler."

I asked ten consecutive asthmatic outpatients who use an InspirEase spacer with their MDI to demonstrate their technique with the device. All patients had received instruction in and a demonstration of the proper technique, and were told to read the illustrated instructions that come with the device. In spite of this, seven patients used the device improperly. Three patients inhaled too rapidly, invariably sounding the warning whistle; two patients actuated the MDI only while inhaling from the bag; one patient actuated the MDI with the bag closed, then exhaled into it; and one patient never actuated the MDI at all. After these patients were again instructed on the proper use of the InspirEase, however, and were able to satisfactorily demonstrate it, their performance was excellent on subsequent office visits. To ensure reliable aerosol delivery from an MDI, even utilizing a spacer device, careful instruction involving actual demonstrations by patients is necessary.

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Bicycle safety

There are over 78 million bicyclists of all ages in the United States. An estimated 93% of children ages 6–16 ride a bicycle. However, fewer than 5% of children who ride bicycles wear helmets. In the United States, more than 1,000 people a year die from injuries sustained while riding a bicycle. Nearly half of these deaths occur among children. The US Consumer Product Safety Commission estimates that nearly 400,000 children ages 1–14 were treated for bicycle-related injuries in hospital emergency rooms in 1985. More than 600 of these injured children died—80% of these deaths were attributed to head injuries. This toll exceeds the death rates from either accidental poisoning, falls or firearm injuries to children.[1]

Head Trauma

Traumatic head injury is the single most frequent cause of death and the most frequent serious (nonfatal) injury sustained in bicycle accidents. Over 75% of bicycle-related deaths involve head trauma and nearly 70% of hospitalizations from bicycle-related injuries are for treatment of head trauma.[2] A Canadian study found that of 73 head-injured bicyclists, none had worn a bike helmet at the time of accident.[3]

Autopsies were performed between March 1956 and December 1979 in all but two deaths occurring from bicycle accidents in Dade County, Florida. Of the 173 autopsies, 81% revealed evidence of brain injury. The median age of the bicyclist killed was 14. None of the victims were reported to have been wearing a bike helmet. Males predominated (139 males, 34 females) in all age groups within the range of ages 3–92.[4] Other studies show a common age-sex association; males have a much higher incidence of bicycle accidents and the under-14 age group has the highest incidence rate for bicycle-related injuries.

From 1980 to 1984, the number of bicyclists in the central business district of Manhattan rose by 42%. This growth in bicycle usage has been associated with a high incidence of bicyclist injuries and fatalities in New York City. In 1983, 20% of all bicycle fatalities and 43% of all bicycle injuries in New York State occurred on New York City streets. By 1985, this increased to 31% of fatalities and 48% of injuries. With the number of bicycles used on roadways steadily rising, a continued increase in injuries and fatalities may be expected, warranting greater efforts to educate all roadway users.[5]

Alcohol Involvement

In 1986, the New York State Department of Motor Vehicles (DMV) reported 58 bicyclists killed and 10,281 injured according to police accident reports. Alcohol was involved in 14 (24%) of the 58 fatalities and 213 (2%) of the 10,281 injuries. According to the accident records, 3% of all highway deaths involved bicyclists, and 4% of all people injured on highways were bicyclists. Previous research indicates that in the majority of bicycle/motor vehicle mishaps it is the driver of the vehicle for whom alcohol is specified as a contributing factor.

Contributing Factors

Identifying some of the causes of bicycle accidents will help to determine intervention strategies to reduce the number of injuries and fatalities. As noted in New York State DMV police accident reports, alcohol use is a major factor in vehicle/bicyclist crashes. Another factor involves the close passing of bikes by trucks and vans whose oversized mirrors have been reported to strike bicyclists. The risk of collision is compounded by darkness and nonuse of reflective clothing and gear.

The National Highway Traffic Safety Administration's (NHTSA) Fatal Accident Reporting System (FARS) reported that from 1975 to 1982, the annual number of bicycle accidents under conditions of darkness increased from 259 to 329. Many of these nighttime bicyclist/motor vehicle accidents occurred when victims were wearing dark clothing.[6] According to one study, the most common bicyclist/motor vehicle accidents involved overtaking and striking the bicyclist along the side of the roadway.[7] In New York State, studies of bicycle accidents over a three-year period showed that most bicycle/motor vehicle accidents occur from 7 AM to 7 PM, with the greatest frequency between 4 PM and 7 PM. Six out of ten accidents occurred on local streets.

A study of the importance of bicycle safety helmets in reducing head injuries was conducted among 894 South Australian bicyclists registered in five bicycle clubs.[8] A mail questionnaire asked club members if they had recently been involved in a bike accident and if a bike helmet had been worn at the time of the accident. Of the 197 riders reporting accidents, 168 were males (85%) and the mean age for the entire group was 29 years. Of these, 38% were not using a helmet, 35% used a hairnet helmet, 19% wore a hard shell helmet of poor design and 8% used a good hard-shell helmet at the time of the crash. The crash victims without helmets were 4.2 times more likely to suffer head injury than bicyclists in all of the helmet groups combined. Although any type of helmet offers more protection than none at all, those head-injured bicyclists who wore a good hard-shell helmet suffered less severe injuries than those who wore a poor hard-shell or a hairnet type, which offers the least protection against severe injury.

Similarly, a prospective case control study conducted by the University of Washington in Seattle also found helmets to be effective in preventing bicycle-related head trauma.[9] Of 179 head-injured bicyclists, 14 (7.8%) were wearing helmets. Of 335 injured riders without head trauma, 89 (26.6%) wore helmets. Therefore, the use of bicycle helmets decreased the risk of head injury by approximately 78% (95% confidence interval, 63% to 86%).

The low rate of bike helmet usage has been attributed to the lack of information provided to children by parents, teachers and health professionals. Of 161 pediatricians and family physicians surveyed in Tucson, Arizona,[10] 30% reported they almost never discussed safety during well-child care and 29% never discussed safety, although the majority knew that bicycle-related deaths among children were more frequent than deaths from other causes of injuries. Most were aware of the low usage rate for bike helmets and believed that lack of parental awareness of the importance of using helmets was the reason.

Bicycle Injury Prevention

The studies reviewed demonstrate the relationship of bicycle accidents and head injury and indicate the need for preventive measures, such as encouraging bike helmet use. Routine use of bicycle helmets is probably the most important intervention that can be undertaken to decrease the incidence of serious bicycle-related injuries.[2] Bike safety education provided by our schools, community agencies, physicians and law enforcement officials can be influential. Such educational efforts should stress:

- proper use of a bicycle helmet to reduce the risk of head injury in a collision;
- use of lighting and reflective gear on bicycles and reflective clothing to improve visibility to motorists;
- observance of traffic safety laws;
- use of lanes and paths designated for bike use, whenever possible.

The provision of bike lanes and barriers in urban areas, as well as bicycle paths and routes in both residential and nonresidential areas can be an effective environmental intervention.

Bicycle safety programs with an emphasis on helmet use have been started in Albany and Suffolk counties. For information about starting a bicycle safety program in your area, contact the Injury Control Program at (518) 473-1143.

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Zoonoses of dogs and cats: An overview

Introduction

Zoonoses have been defined by the World Health Organization as "those diseases and infections which are naturally transmitted between vertebrate animals and man." Animal-associated illnesses have been recognized for centuries. There are at least 165 diseases that can be transmitted from animals to man. Some diseases, rabies for example, are well known, albeit infrequent. There are, however, many lesser known zoonoses present in New York State.

Toxoplasmosis

Toxoplasmosis is caused by a protozoan parasite, *Toxoplasma gondii*. Toxoplasma exists in three forms: trophozoite, cyst and oocyst. The trophozoite is the invasive form responsible for manifestation of acute infection. The cyst is responsible for persistence of latent infection in multiple organs. Only the cat family has been found to harbor the oocyst in the intestinal tract; in all animals, the parasite is found encysted in muscle or brain tissue.

After formation in the cat intestine, oocysts are excreted in the feces and undergo maturation for one to 20 days before becoming infective. The mature oocysts may remain infective in water and moist soil for months. Rodents, swine, cattle, sheep, goats, chickens and other mammals and birds serve as intermediate hosts of *T gondii*; infection can occur by the consumption of raw or undercooked infected meat or by the ingestion of infected oocysts (through water or soil contaminated by feces of an infected cat).

Infection in humans and other animals occurs primarily after ingestion of either the cyst or the mature oocyst. When the cyst is disrupted, the trophozoites invade the intestinal epithelium and are spread hematologically to all organs.

The most common symptom in people is lymphadenopathy. Spontaneous resolution usually occurs. However, in immunocompromised hosts, fatal encephalitis may develop.

The two main preventive measures are adequate cooking of meats and immediate disposal of cat feces. Household cats should be fed canned or dry cat food and not be given raw or undercooked meat or allowed to hunt for fresh meat; they could ingest the organism by eating the contaminated flesh of infected animals or birds. Because of the risk of transplacental transmission, pregnant women should avoid cleaning cat litter pans and contact with cats of unknown feeding history. Gardeners may come into contact with contaminated soil, so they should wash hands carefully before preparing food. Sandboxes should be covered when not in use by children to discourage cats from using them.

Cat Scratch Disease

Cat scratch disease was first described in 1931 and, since that time, more than 700 reports have been written about its symptoms and etiology. In 1983, a small gram negative bacillus was identified in lymph gland tissue by using a silver staining technique.[1] Clinically, the disease usually follows a scratch or bite of a cat with the development of a papule at the site of exposure, followed by regional lymphadenopathy. The nodes are often tender and painful and may remain enlarged for several weeks to months. Suppuration may occur.

Although cat bites and scratches are usually involved, up to 35% of the cases have no cat involvement. In such cases, infection may have resulted from a penetrating injury to the skin by thorns, splinters, etc, that may have carried the agent. Cats serve as mechanical carriers of the agent; they are not necessarily infected themselves.

Diagnosticians should use histopathology; specific skin test antigen may be helpful but not conclusive. Antibiotic therapy has not proven to be universally effective.

DF-2 Infection Following Animal Bites

One of the more serious consequences of keeping animals as pets is the ever growing number of bite cases. In addition to the infliction of skin damage and infection, the time spent on investigation by health personnel is a financial burden on public resources.

One of the less common bacterial infections following dog bite is that caused by a yet unnamed gram negative rod designated DF-2 (dysgonic/fermenter). A number of wound infections following dog bites have been attributed to this organism. Symptoms include fever, cellulitis, endocarditis, meningitis, hypotension and renal failure. In some cases, death has resulted.

Physicians should be aware that the organism may be a natural inhabitant of a dog's mouth. This organism may be the causative agent of illnesses with the aforementioned symptoms, especially if the patient describes a history of dog ownership. The organism may take as long as eight days to grow on media. Penicillin is the drug of choice for treatment.

Brucellosis

Human brucellosis is less common now that the incidence in cattle has been almost eliminated. However, one of the newer zoonoses is due to *Brucella canis*, which is prevalent in dogs and results in abortion. Transmission to humans is rare but has occurred with symptoms similar to, yet usually not as severe as, those caused by *B abortus*. These include fever, headache, lethargy, profuse sweating, chills, arthralgia and weight loss.

Toxocaral Visceral Larva Migrans

Toxocara canis, the common roundworm of dogs, is found universally wherever dogs exist. Although less common in older dogs, transplacental migration of larvae occurs from infected pregnant dogs to fetal pups. Roundworm ova shed in the feces require two to three weeks to mature and may infect humans through contaminated soil. This is especially the case for children with a history of pica (a craving for unnatural food such as soil). Hatching occurs in the proximal small intestine of the host, and the released larvae penetrate the mucosa, migrate to the liver via the portal circulation, follow vascular channels to the lungs and then enter the systemic circulation.

Consequences of ingestion vary with the numbers of larvae but symptoms include coughing, wheezing, pallor, malaise and weight loss. Pulmonary involvement may present as acute bronchiolitis, asthma, pneumonitis or a combination of these symptoms. Ocular involvement may be confused with granuloma and result in unnecessary enucleation.

Strategies to prevent infection in humans include adequate deworming of dogs and proper disposal of dog feces.

Sarcoptes Scabiei

The human and canine types of *Sarcoptes scabiei* are morphologically identical. However, they have been classified separately as *S scabiei var canis* and *S scabiei var hominis*, which differ in the type of human lesions they induce. The canine (*canis*) variety causes lesions in the human on the abdomen, trunk and arms but rarely on the face or genitalia. The lesions are papule-like without burrowing. Delayed hypersensitivity does not occur. Infection is due to direct contact with infested dogs. Lesions due to the human (*hominis*) variety are much more severe, and are found on hands, arms, buttocks and genitalia. Burrows are usually present. Transmission occurs via infested humans.

Q Fever

Q fever is caused by the rickettsia *Coxiella burnettii*. The natural reservoir is certain species of wild and domestic animals, especially sheep, goats and cattle which serve as the main source of human infection. Infection occurs primarily through inhalation of the rickettsiae. A recent outbreak of Q fever in Nova Scotia was traced to a cat that gave birth to stillborn kittens. Infection resulting from ingestion of contaminated meat, milk or milk products and by tick bite is rare.

A patient with Q fever may present with cough, chills, high fever, vomiting and malaise, which may be confused with influenza symptoms. Duration of illness may be as much as three weeks. Complications may include endocarditis or myocarditis. Diagnosis is made serologically. The drugs of choice are tetracycline and chloramphenicol.

Zoonosis Prevention

Since people will continue to have pets, zoonotic disease incidence will continue. Children should be taught the hazards of injury as well as risks of possible infection. Those too young to understand need to be protected from exposure to potentially dangerous animals (eg, ferrets). Physicians should make careful inquiry about a patient's animal association especially when symptoms are unusual.

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Tobacco use in New York State: 1982–1986 trends

Cigarette smoking continues to be the leading preventable cause of premature morbidity and mortality in New

York State. Nearly 30% of the adult population continues to smoke, costing the state $4.7 billion annually in health care costs and resulting in 27,000 premature deaths.[1]

There is evidence, however, that the proportion of the population that smokes has been declining. Data are available from two national surveys and an annual survey of New York State residents. The US Public Health Service conducted a survey of a random sample of the US population in 1983 and 1985—the National Health Interview Survey (NHIS).[2] Under the auspices of the Centers for Disease Control, the Office of Smoking and Health (OSH) performed a similar survey in 1986.[3] Also, the New York State Behavioral Risk Factor Surveillance System (BRFSS) provides smoking rates that can be compared with these national data.[4] (The BRFSS was described in the April 1988 issue of *Epidemiology Notes.*) Each of the surveys discussed defines a current smoker as one who has smoked at least 100 cigarettes in his/her lifetime and reports present use.[5]

All three surveys reported similar prevalence rates. In 1983, the proportion of current smokers in the US was 32.4%. In the same year, the New York State rate was 30.4%. In 1985, the rate dropped to 30.4% in the national survey but showed an increase in New York State to 31.4%, due to an increase in female smoking greater than the decrease in male smoking. However, by 1986, both female and male smoking had dropped to show an overall smoking rate of about 27% in both state and national survey data.

The US Public Health Service has established a 1990 national objective that aims to reduce to 25% the proportion of adults who smoke, and the New York State Department of Health has adopted the objective.

Smokeless Tobacco

While smoking rates appear to be declining, the use of smokeless tobacco has risen sharply, especially among male adolescents. Smokeless tobacco has been causally linked to oral cancer, periodontal disease and other problems related to nicotine addiction.[6] Serious concerns have been raised about these health effects in light of the fact that the sale of moist snuff has risen from 23.7 million pounds in 1978 to 37.1 million pounds in 1984, while sales of chewing tobacco have risen from 80 million to 87 million pounds during the same period.[7]

According to Boyd et al,[8] who reviewed a number of studies on this subject, evidence from a 1985 National Institute on Drug Abuse (NIDA) survey indicates that 25% of males and 3% of females between ages 12 and 17 have used smokeless tobacco and that 7.4% of the males report weekly or more frequent use. In most grade level-specific studies, the proportion of students who tried smokeless tobacco exceeded that reported in the NIDA study. The proportion of sixth graders who had ever used smokeless tobacco varied from 6.7% in New York City to 68.2% in rural Montana, while reports of recent use for sixth graders ranged from 1.1% in New York City to 14.9% for rural Vermont. In New York State, questions about smokeless tobacco use were added to the BRFSS survey in 1986. The BRFSS results indicate that 20.2% of males ages 18–24 have used smokeless tobacco. The rates of use decline with age except in the over 65 population. This may be attributed to the fact that, until recently, smokeless tobacco was most likely used by older men and women and certain occupational groups, such as miners, in which smoking was not allowed. Because the BRFSS collects information only from household members over 17 years old, New York State estimates for the 12–17 year age group are not available. The State Department of Health and the State Department of Education are presently working with the State Division of Substance Abuse Services to incorporate questions on tobacco use and especially smokeless tobacco into the division's 1988 survey. This survey, to be conducted in spring 1989, will gather data from more than 27,000 fifth through 12th grade students statewide.

For more information about tobacco use, smoking issues or smoking related Health Department activities, contact Stephen Onderdonk, Cancer Prevention Services/Health Risk Reduction Program, at (518) 474-1222.

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MANAGING CHRONIC DISEASE

Edited by C. Stewart Rogers, MD, and Jack McCue, MD. 439 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $29.95 (softcover)

This book is a concisely written review of the clinical approaches used in managing adults with long-term illnesses. It is organized into chapters by illness and into sections by organ systems. The editors and 19 additional contributing authors are well qualified to discuss these subjects, both by their apparent knowledge of the underlying principles of medical practice, stated here in the briefest possible form, and by their clinical experience. Most are clinical teachers associated with institutions in North Carolina, although Dr McCue is associated with Tufts University in Boston.

Explicit presentation of guidelines for therapy, including dosages and methods of administration, coupled with succinct discussions of specific conditions, is supplemented by useful summary tables. The result is a practical handbook for an area in which a sound literature is only beginning to emerge, although the area represents much of the health care administered in Western countries. The inclusion of a clinical pharmacist in the editorial board significantly adds to the value of discussions that are also oriented toward what not to do in order to avoid pitfalls of both judgment and therapy. The writing style and presentation are surprisingly consistent throughout for a work with this many contributing authors. Chapters are brief enough for convenient reading and for use as an "at hand" ready reference. References at the end of each chapter are few, but point to fundamental sources where more detailed bibliographies may be found.

The opening chapters discuss core issues and basic principles for the ongoing management of long-term adult illnesses, including when to test or treat, how to obtain consensus and compliance with the patient and family, and how to prescribe effectively while avoiding iatrogenic complications. Therapeutic principles are stated briefly enough to reinforce correct clinical decision making without any elaboration or expansion. The closing chapters similarly summarize the approach to geriatric patients by reviewing the rapidly expanding literature. Each intermediate chapter summarizes the evaluation and care of one clinical presentation with regard to minimal indicated testing, appropriate treatment, and cautions about commonly made, self-compounding errors. The perspective of the patient and family, the impact of decisions on other aspects of daily life, and the cost/benefit impact of actions taken are clearly represented.

Little other literature has yet emerged for the clinical management of chronic care, and much of it is oriented toward rehabilitation medicine or the pediatric population. A concise, clinically oriented book that is useful and inexpensive will serve multiple roles as an introductory text for third- and fourth-year medical students, as a review for resident physicians in primary care medicine, and as a reminder to the experienced clinician of how and why one makes "empiric" decisions. This is a handy shelf reference for daily use in the selection of tests and prescriptions and for the delivery of not only caring but also correct care. As the authors propose, it is up to date, realistic, and practical, positive but not unrealistically hopeful, and a fair representation of the perspectives of patient, family, and physician in what are often difficult situations.

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MEDICINE ON TRIAL. THE APPALLING STORY OF INEPTITUDE, MALFEASANCE, NEGLECT, AND ARROGANCE

By Charles B. Inlander, Lowell S. Levin, EdD, and Ed Weiner. 304 pp. New York, Prentice Hall Press, 1988. $18.95 (hardcover)

The authors of this book are all affiliated with the People's Medical Society, a five-year-old nonprofit consumer advocacy organization with a membership of about 100,000. Mr Inlander is President, Mr Weiner is Senior Editor, and Dr Levin is on the Board of Directors and a Professor of Public Health at Yale University School of Medicine.

According to Dr Levin, the People's Medical Society receives 3,000 letters a week "filled with truly heart rendering stories. People are so frustrated . . . not knowing to whom to turn for redress, or just to let out their anger at medical care."

A possible political truism states that people with a gripe or engaged in "anti" movements tend to vote and take action. Contrarywise, "do-gooders" or people satisfied with the status quo don't vote and don't take action. Even if that be so, the emotional reactions to this book, which uses professional journals to discover medical mistakes, makes it incumbent that those in the health professions be aware of what the public is being told. Readers of this book are being alerted to the following frightening potential risks when they become involved in the US health care delivery system:

- One of every six drug addicts is a physician.
- Up to 10,000 people a year die from ineptly administered anesthesia.
- Five million operations a year are unnecessary.
- On average, a hospital mistakenly gives 60 patients the wrong medicine or the wrong dosage every hour of every day, seven days a week, 52 weeks a year.
- One of every five hospital patients is likely to leave the hospital with a condition they didn't have when they checked in.

This documented list of medical malfeasance goes on and on, but the picture should be clear from the examples. In fact, an advertisement for this book shows a picture of a patient in surgery being tended to by four health professionals and asks the reader to "spot the medical mistakes." Seven deadly mistakes relate to the surgeon's having had four martinis with lunch; the resident's having worked for 36 straight hours; the use of the wrong syringe by the anesthetist; a doctor's not washing his hands; an erroneous laboratory report; defective medical equipment; and the fact that a radiologist misread the x-ray, resulting in an unnecessary operation.

Twelve chapters of evidence and expert testimony, referenced with 30 pages of small-type-size citations, lead up to a final chapter with a prescription for change. These authors suggest 12 immediate remedial actions, such as periodic testing, relicensing, and recertification of physicians and other medical care workers; development of governing mechanisms (overseen or operated by government) to replace the failed machineries of medical autonomy; creation of medical "teams" in which the physician is not the head or sole arbiter, but an equal member in a diagnostic/therapeutic group that includes continuous involvement with and input from nurses, pharmacies, and, of course, the patients; and tougher Food and Drug Administration regulations and oversight of present and emergent medical technology.

Additional immediate remedial actions involve disciplining impaired physicians; battling nosocomial infections; instituting unit dose medications in hospitals; prohibiting reuse of disposables; funding self-care and alternative care; providing public access to medical libraries; disclosing physicians' economic relations with other providers; and improving medical care press coverage.

In addition, a "Model Hospital Disclosure Act" is detailed to reveal needed quality-related data, with the suggestion that the act be adopted in each state. Furthermore, the People's Medical Society recommends that all patients be provided with a copy of the Disclosure Act.

Practitioners are also urged to provide patients with a ten-point "Code of Practice" dealing with office procedures and choices in diagnosis and treatment. The code calls for posting of fees and itemized bills; providing nonemergency telephone consultation hours; scheduling appointments with minimal waiting time; notifying patients of lab test results; allowing friends/relatives to accompany patients into the examining room; and helping patients to obtain medical records and test results. Suggested diagnosis and prescription actions deal with explaining the prognosis, discussing alternative treatments, revealing the physician's qualifications to undertake the Dx and/or Rx; recommending sources of support and information, and not proceeding until there is understanding and agreement on the Dx and Rx.

A 15-page appendix lists organizations, with addresses and phone numbers, from which individuals can seek redress regarding dentists, physicians, hospitals, and nurses. A 16-page glossary defines terms for the lay person, beginning with *acupuncture* and ending with *x-ray*.

The foreword of this strident book opens by saying: "I warn you. What you will read is not very reassuring. This book presents a grave indictment of American medicine." In closing, the last sentence in the book reads: "We are serving notice that the social charter of medicine is about to be rewritten and reaffirmed as a public responsibility."

These opening and closing statements reinforce the contention that health/medical care professionals should be aware of what the public is reading. These illustrations and examples can lead us to examine our own behavior. If we are doing well, we can applaud ourselves. If we are not measuring up to the public's expectations, we can see the need for changes and make them. Some of the material in this book may enrage physicians, some may even be inaccurate. Nevertheless, this book is highly recommended to provide a point of view from the other side of the stethoscope.

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COMPUTED TOMOGRAPHY OF THE SPINE. DIAGNOSTIC EXERCISES

By Robert Kricun, MD, and Morrie E. Kricun, MD, 290 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1987. $56.00 (hardcover)

The authors have presented a book that they hope "will benefit those interested in understanding and interpreting CT of the spine." This goal is accomplished in a very effective manner. Sixty-six cases are presented, with one to three images, primarily CT, and minimal appropriate clinical information. After studying the presented material, the reader is challenged to make a diagnosis. On the following pages the authors give a succinct explanation of the original images. In addition, a thorough, detailed presentation of the condition as well as a differential diagnosis is available. Other types of examinations (ie, myelograms, plain films, and magnetic resonance imaging) add to the completeness. Good line drawings and specimen photographs are included to enhance the text. Each case is presented in an average of five pages.

The cases presented are grouped in categories such as disc disorders, spinal stenosis, infection, trauma, and so on. However, all of the authors' cases do not represent disease processes. Also described are patients who present with artifacts, such as pseudoherniation of the disc, "cupid's bow," and basilar vertebral veins. A discussion of pseudospread of the atlas is part of the discussion on Jefferson fracture. These presentations emphasize pitfalls in diagnosis as well as the importance of methods and techniques of scanning. A brief consideration of scalloping and erosions of the spine is appropriate. All cases are presented clearly and are accompanied by excellent quality illustrations.

The text creates an excellent source both for immediate reference as well as more detailed information from the average of eight up-to-date references which accompany each case. Especially well organized is the section on disc disease, which provides a good, workable, acceptable classification and illustration of disc disorders. The congenital disorders are well assembled including syringomyelia, myelomeningocele, diastematomyelia, tethered cord, and Chiari malformation.

The section covering trauma is presented in an organized manner with appropriate illustrations. Besides a discussion of important common diseases and injuries, this book also covers less common conditions such as scleroderma with spinal calcification, extensive cervical intramedullary (holocord) tumor, and multiple intradural schwannomas.

The book resembles the syllabus productions of the American College of Radiology, but does not include an initial and separate quiz of multiple choice questions. Doctors Robert and Morrie Kircun have the qualifications, training, and experience in skeletal radiology to write this text.

The book achieves its stated objectives and this reviewer considers it excellent for radiologists.

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MAGNETIC RESONANCE IMAGING OF THE THORAX

By Miriam Sperber, MD, and Marc C. Kaiser, MD. 270 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1987. $65.00 (hardcover)

In their introduction the authors state that the purpose of this book is to serve as a guide to the understanding of the advantages as well as the limitations of magnetic resonance (MR) imaging compared to other existing diagnostic modalities, and to help the reader become familiar with the presentations of various abnormalities on MR images. In the majority of the book their intent has been met.

The book encompasses 11 chapters. It begins with an introduction and brief discussion of physics, and ends with a limited look to the near future of magnetic resonance. In between, the chapters are arranged by anatomic category, ie, cardiovascular, mediastinum, hilum, pulmonary nodules and bronchogenic carcinoma, benign pulmonary conditions, and pleural and diaphragmatic abnormalities.

The discussion of MR physics is basic and brief and can only be regarded as an overview, appropriate for this type of book. However, in the chapter on the cardiovascular system there is a fine discussion on cardiac gating techniques including gradient recalled fast scan imaging. A limitation is the lack of any discussion of respiratory compensation techniques.

The best chapters are those concerning the hilum and pulmonary nodules. Here the authors' intention of comparing MR to computed tomography and x-ray tomography is truly met. The text gives meaningful discussion of when MR or CT is the diagnostic modality of choice. In addition, relevant tables reinforce the discussion.

The section on cardiovascular diseases is brief and to the point. Anatomic lesions as well as functional abnormalities are discussed. The newer techniques of cardiac "cine," left ventricular mass, and wall thickening determination are shown. Also, magnetic resonance spectroscopy is briefly presented.

A serious limitation, which should be a strong point in a basic text on a specific topic, is the chapter on normal thoracic anatomy. This chapter, along with the normal anatomy section of the cardiovascular chapter, is too superficial. While the axial images, along with their corresponding drawings and CT scans, are adequate, the sagittal and coronal images and discussion are limited, pointing out only major structures. As the authors repeatedly acknowledge, one of the main advantages of MR is its multiplanar capability; therefore, normal sagittal and coronal anatomy should be covered in greater depth. If not in a book of this kind, where will it be presented?

Images presented are for the most part clear, well labelled, and of state-of-the-art quality. At the end of each chapter is an excellent reference list of pertinent articles.

The work-up of thoracic disease is evolving. This book is a good introduction, and a place to begin learning what magnetic resonance can offer to the diagnostic armamentarium.

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FOREIGN TRAVEL AND IMMUNIZATION GUIDE

12th Edition. By Hans H. Neumann. 86 pp. Oradell, NJ, Medical Economics Books, 1987. $12.50 (paperback)

The 12th edition of *Foreign Travel and Immunization Guide* by Hans H. Neumann represents a time-tested compact guide to emporiatrics. Dr Neumann was well qualified to write this volume, having had a long career in the field of public health and preventive medicine. He served as Director of Preventive Medicine for the New Haven, Connecticut, Department of Health for 17 years.

The compendium is thorough and concise and accomplishes its mission in 85 easy-to-read pages. The text can be equally well utilized by physician and traveler alike. Advice is given in an unpretentious, straightforward style. In particular, chapter 31, section III, titled "Centers for Medical Assistance to Travelers," is an extremely useful reference guide should the sojourner require medical advice or help in a foreign country.

There are some minor downsides to the text. For example, how can the reader be assured that the information in the book is current and accurate, since immunization requirements are regularly updated? The Centers for Disease Control (CDC) updates its recommendations monthly. The last edition (11th) of this text was published in 1982. Despite the fact that the author based his book on World Health Organization (WHO) and CDC information, the dosage recommendations of some of the vaccines (eg, cholera, tetanus, immune serumglobulin) in his text are distinctly different in amount and/or frequency from these guidelines. For children specific dosages are not given; rather the unclear words "proportionately smaller" are used.

Lastly, the "Foreign Travel Health Guide" section should have included a brief recommendation for a pre- and post-travel consultation with one's primary care physician. The dangers inherent in travel, belied by the existence of this text, are sometimes best dealt with by one's personal physician. Such an individual can provide a personalized travel prescription based on the health status of the patient-traveler, and review same upon return. As the author points out in the preface, "some recommendations cannot be standardized"; they need to be individualized and tailored to the specific itinerary. Finally, the chapter "Accidents and Injuries" (chapter 13) should have noted that the major cause of loss to life and limb during travel is not parasites, poisoning, or other infectious maladies, but rather vehicular and pedestrian accidents. Such a statement would allow the reader a realistic perspective with regard to travel risks.

Despite these drawbacks, this reviewer recommends this book. Its utility lies in its simple and easy to understand style. Gray areas can be supplemented by the annual CDC *Health Information for International Travel.* Certainly it is wholeheartedly recommended to the lay sojourner as a practical travel companion.

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SURGICAL LASERS. A CLINICAL GUIDE

Edited by Terry A. Fuller, PHD. 237 pp, illustrated. New York, Macmillan Publishing Co, 1987. $75.00 (hardcover)

The author's stated goal is to provide "a single reference source describing the clinical application of lasers as a surgical tool." The author has been a pioneer in the scientific development of lasers for approximately 15 years. He has published numerous medical journal articles on lasers, given laser courses, and has been an active participant in the American Society for Laser Medicine and Surgery, of which he is a founding member. Dr Fuller is known for basic science research in lasers with a special interest in fiberoptic transmission of various laser wave lengths. The chapter on physiology that precedes the clinical description of lasers in the various specialties uses relatively complicated scientific terms and examples but contains a good description of the various lasers as well as differences in pulse, superpulse, and laser types of delivery systems. Tissue interaction is well described in detail pertinent to surgery. The bibliography is brief and, interestingly enough, lacks reference to Einstein's original theoretical description of laser light generation.

All specialty chapters are accompanied by a very detailed initial outline to direct the reader's attention to the pertinent points of the chapter. The specialty chapters are organized in a "how-to" form mixed with good physiology. Specialty subjects include otolaryngology, pulmonology, gynecology, fertility, dermatology, urology, gastroenterology, podiatry, and neurosurgery. All provide good to excellent technical descriptions of laser wave lengths which are applicable in the specific specialty as well as indications for their use. The chapters on ENT and pulmonology are exceptionally strong in their descriptions of operating room and instrument setup. Many of the chapters have outstanding color illustrations of highly descriptive artists' depictions and very comprehensive bibliographies. One criticism of the chapter on dermatology is that it unfortunately focuses on keloid ablation with the carbon dioxide laser which is illustrated in the initial photograph of the chapter. Unfortunately, this procedure is highly controversial and viewed with great skepticism by experienced laser users, and the success of certain authors has not been duplicated. The neurosurgery chapter is quite brief and only describes basic principles and does not provide any illustrations. There is very little information about equipment setup or patient care, and this single chapter seems to be out of context with the style of the rest of the book. The index is extremely comprehensive.

In summary, this excellent volume will serve as a good introduction to the broad applications of lasers in the various aspects of specialty surgery for the novice or for hospitals and nurse specialists getting involved in lasers. The specialists would also enjoy the thorough review and synthesis of various lasers and their applications in their specialties. The book is especially excellent in terms of the illustrations, containing numerous color photographs and artists' diagrams which illustrate the techniques. A good "how-to" description is provided in all chapters (with the exception of neurosurgery) which would serve as a reference for laser users. The only serious deficit of the book is that it fails to mention any safety considerations which are so vital in the application of lasers both for the physician user and the patient, and similarly there is no suggestion as to the organization of an office or operating room for lasers. Such things as credentialing of laser surgeons, laser safety committees, maintenance, and so on are frequently confusing for medical personnel who are starting to use lasers and are as important as the description of individual specialty usage. This volume is an excellent replacement for the two general textbooks that have been published about lasers in the past.

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JANE BRODY'S NUTRITION BOOK. A LIFETIME GUIDE TO GOOD EATING FOR BETTER HEALTH AND WEIGHT CONTROL

By Jane E. Brody. 552 pp, illustrated. New York, Bantam Books, 1987. $12.95 (softcover)

This is the second edition of a best seller written by the personal health columnist for the *New York Times*. It is intended to be a reference for the health conscious consumer who believes in the adage that "we are what we eat." The text is organized into six parts, each dealing with a large segment of nutritional knowledge.

Part one is entitled "What to Eat" and includes a summary of the findings of the Senate Select Committee on Nutrition and Human Needs. This committee, chaired by Senator McGovern, found that the diet of Americans had changed in an unfavorable fashion since the turn of the century. The American diet was reported to include substantially more protein, saturated fat, cholesterol, and sodium, and substantially less unrefined carbohydrates. The experts who testified suggested that the changes in the American diet were directly related to the dramatic increase in coronary heart disease during this period. Furthermore, more recent evidence has linked such a diet to higher risks of colon and breast cancer. The committee recommended that the dietary goals for Americans should include a dramatic reduction in dietary fat from 42% to 30% of dietary calories. The diet should contain equal proportions of saturated, monounsaturated, and polyunsaturated fats. They further recommended that dietary carbohydrates be increased from 46% to 58% of calories, and that only 10% of calories be from refined sugars. These recommendations have subsequently been adopted by the American Heart Association. The remainder of part one addresses each of the macronutrient classes in turn with an emphasis on the historical changes in the American diet since the industrial revolution.

Part two is entitled "Noncaloric Nutrients." In this part, the author summarizes the controversies surrounding dietary fiber, vitamins, and minerals. The section on calcium and sodium in particular will undoubtedly be read with interest by many, since so much attention has been addressed to these subjects in the lay press as well as in the professional literature.

Part three is entitled "What to Drink" and includes discussions addressing water quality, coffee, tea, milk, and alcohol. Parts four through six include discussions of techniques of weight control and nutrient requirements for pregnancy, lactation, infancy, adolescence, and senescence. Vegetarian diets, food additives, health food, and the labelling practices of the prepared food industry are addressed.

In each section, the topics are presented in a balanced, straightforward fashion. The author is careful to present several sides of a controversy and has done extensive, up-to-date background research.

In summary, this is a well-conceived, well-executed, encyclopedic treatise for the lay reader which presents rather complex nutritional information in a surprisingly readable way. While not extensive enough for the professional, one should be aware of what patients may be reading. Without reservation, I would recommend this book to patients in need of nutritional information.

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Researchers express need for national HIV databases

In order for researchers to track and predict the course of the AIDS epidemic, they need access to comprehensive sources of raw data. A group of authors from Los Alamos National Laboratory (Los Alamos, NM) have proposed "the creation of national HIV databases to coordinate the sharing of raw data" (*Nature* 1988; 333:511–512). In the future, perhaps, these databases could be linked internationally.

The sharing of raw data is necessary for researchers to monitor the prevalence of human immunodeficiency virus (HIV) infection and to develop mathematical models concerning HIV transmissibility and incubation time and trends in the spread of HIV infection. The authors note that there are insufficient raw data in journal articles, government reports, and conference proceedings. The *AIDS Public Information Data Set*, which contains an individual record for each reported case of AIDS, is the largest epidemiologic surveillance database in the US. However, the cases are divided among large metropolitan regions to maintain confidentiality. This dilutes the value of the data and prevents accurate monitoring of the spread of infection.

"In its simplest form, a national HIV database would be an annotated directory of raw data. In its most complete form, it would be a storehouse of raw data on HIV infection and the AIDS epidemic." Regardless of the form of the database, it would have to inform users where to find the needed information and to promote the sharing of raw data among researchers. The authors propose a standard agreement, to be signed by both users and providers of data, that would refer to a specific set of data.

"The establishment of a national HIV database will require an extraordinary level of commitment on the part of the research community." It will be based on a mechanism that facilitates both the sharing and protecting of confidential medical and behavioral data.

New AIDS information service

The main component of this new AIDS Information Service is the AIDS Quick Reference Database from Clinical Reference Systems, Ltd, a source of clinically oriented material on AIDS, including information on patient history and risk status, diagnosis, and therapy. The new service supplements this reference database with AIDS-related medical literature, daily worldwide news concerning AIDS, and AIDS statistics from the Centers for Disease Control. The service also offers predefined literature searches and lists current references and resources for patient use.

American Medical Computing, Ltd, a subsidiary of the American Medical Association (AMA), developed the AIDS Information Service in conjunction with Softsearch, Inc, a distributor of on-line database services. The service is available on-line from AMA/NET.

FDA approves cervical cap

Following FDA approval, the cervical cap returns to the US market as a contraceptive option for women. The approved cap is a flexible, cup-like device made of natural rubber. A physician must fit the cap around the base of the cervix to determine the appropriate size. Once a woman has the prescription, she can insert and remove the device herself.

This reusable cap is applied with a spermicidal cream or jelly and can stay in place for 48 hours, compared to a maximum of 24 hours for the diaphragm. Unlike with a diaphragm, the cervical cap does not require an additional application of spermicide before each act of intercourse. Other benefits include increased comfort, compared to a diaphragm, for some women.

An NIH-sponsored study demonstrated that the cap is about 85% effective in preventing pregnancy—a rate comparable to that of the diaphragm.

During clinical trials with this device, Pap tests of 4% of cap wearers converted from normal to abnormal after three months of use. The manufacturer of the cap, Lamberts (Dalston) Ltd of the UK, will conduct postmarket studies to further evaluate Pap smear conversion rates. FDA labeling requirements state that the cap may be prescribed only for women with normal Pap tests. Women using the cap must be reexamined by a physician after three months of using the device. If a repeat smear is abnormal, they should discontinue using the cap. In addition, the label will state that, like the diaphragm, the cervical cap slightly increases the risk of toxic shock syndrome.

Survey reveals dramatic increase in hospital closures

Executives of nearly 700 hospitals in the US reported that their institutions are vulnerable to failure within the next five years, according to a survey released by the accounting and management consulting firm Touche Ross. Since 1986, 150 hospitals have already closed their doors.

At greatest risk of failure are small, rural, and government-owned hospitals. Of the 1,400 hospitals included in the survey, 63% of those with fewer than 100 beds reported being vulnerable to failure, as did 54% of the government-owned, and 57% of the rural hospitals. The percentage of suburban hospitals facing possible closure increased by 11% since 1986, and hospitals owned by religious organizations are now more vulnerable to failure. On a more positive note, urban hospitals report increased confidence in their ability to survive, as compared to the findings of the 1986 survey.

Hospital executives cited decreasing revenues due to the Medicare prospective payment system as the primary cause for increased vulnerability to failure. Fifty-seven percent of hospitals in the West South Central region of the country, representing Arkansas, Louisiana, Oklahoma, and Texas, say they are at risk of closing, while 53% of hospitals in the midwestern farm states say they may fail. Since the 1986 survey, hospitals in New England have demonstrated the sharpest increase in vulnerability to

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NEWS BRIEFS are compiled and written by Vickie Glaser.

failure, whereas hospitals in the Mid-Atlantic and the Pacific regions are faring better in 1988.

The survey also demonstrated declining hospital occupancy rates and declining quality of care. The latter is believed to be due to physicians' lack of commitment and the nurse shortage. In addition, increasing malpractice premiums are limiting patients' access to care.

Reye syndrome warning mandatory on aspirin products

The regulation requiring a Reye syndrome warning on the labeling information of oral and rectal over-the-counter products that contain aspirin was made permanent by the FDA prior to the June 6, 1988, expiration date. A mandatory revision to the warning will state that Reye syndrome is reported to be associated with aspirin. A study by the Public Health Service Reye Syndrome Task Force has confirmed earlier reports of the association between Reye syndrome and the use of aspirin in children and teenagers with the flu or chicken pox.

New MRI system to feature open-air design

Magnetic resonance imaging (MRI), a relatively new diagnostic tool, may not be appropriate for patients who suffer from claustrophobia and for those who require bulky life support equipment. Researchers at the University of California at San Francisco's Radiologic Imaging Laboratory in conjunction with Diasonics, Inc, have developed an experimental open-air MRI system that is now undergoing clinical trials.

As opposed to the conventional tunnel design, the unit consists of two magnetized plates supported by four columns, one at each corner. The patient, who lies between the plates, can see through all four sides of the scanner. The scanner can fit through doorways and could potentially be transported to critically ill patients.

FDA approves interferon to treat genital warts

Genital warts result from a sexually transmitted viral infection that has responded poorly to available treatments. Patients who suffer from the estimated eight million new and recurrent cases of genital warts annually in the US may benefit from injections of alpha interferon directly into the lesions.

Clinical trials with genetically engineered alpha interferon demonstrated marked improvement of genital warts following injections of the anti-infective agent three times a week for up to three weeks. The FDA reported that 42% of the 192 patients in the trials had complete clearing of all treated warts, 24% experienced a 75–99% reduction in the size of the treated warts, and 18% had a 50–74% reduction in the size of the treated warts.

Adverse reactions included mild flu-like symptoms. Alpha interferon is contraindicated in pregnant women due to a potential increased risk of spontaneous abortion.

New program provides free t-PA to needy patients

Genentech, Inc, has announced a hospital reimbursement program to provide tissue plasminogen activator (t-PA), the company's genetically engineered drug product used in the treatment of heart attacks, free of charge to uninsured, needy patients. The company's goal is to make t-PA available to all patients who need it, regardless of their income and ability to pay. In 1987, Genentech established a similar program for its human growth hormone product.

Tissue plasminogen activator is used to dissolve blood clots in the early stages of a heart attack. Since receiving FDA approval in November of 1987, Genentech's drug is being used in an estimated two thirds of all heart attack patients who undergo thrombolytic therapy.

If a physician appropriately administers t-PA to a patient whose annual income is less than $25,000 and who is not covered by private or government insurance, including Medicaid or Medicare, Genentech will replace the hospital's vials of t-PA.

Promising new drug for treating thrombocytosis

Anagrelide, a member of the imidazo (2, 1-b) quinazolin-2-one series of compounds, blocks platelet aggregation. Small doses of anagrelide can produce thrombocytopenia in humans. Silverstein et al studied the efficacy of anagrelide in treating 20 patients with thrombocytosis (*N Engl J Med* 1988; 318:1292–1294).

Of the 20 patients, 17 had primary thrombocythemia, two had polycythemia vera and thrombocytosis, and one had chronic granulocytic leukemia and thrombocytosis. Initially, each patient received 2 mg of oral anagrelide every six hours. The authors noted that oral doses of 1–1.5 mg of anagrelide every six hours would reduce platelet counts in most patients. After about five days of anagrelide therapy the count would begin to fall, reaching normal levels after about 12 days. Maintenance doses of anagrelide ranged from 0.5 mg twice a day to 1 mg four times a day.

All of the 20 patients responded to treatment except for two of the 17 patients with primary thrombocythemia. Some patients reported nausea, bloating, and minor headache during the induction period. Maintenance level doses relieved these symptoms. Whereas 5 mg of anagrelide every six hours had produced hypertension in normal volunteers, small decreases in the systolic and diastolic blood pressures of patients occurred during the induction period, and all blood pressures returned to normal levels at maintenance doses. In addition, the authors found that the dose of anagrelide needed to lower the platelet count was well below levels of the drug needed to produce an antiaggregating effect on platelets.

"An agent lacking mutagenicity or leukemogenic potential, yet capable of controlling thrombocytosis, has long been needed. . . .Our observations during this study suggest that anagrelide can control the platelet count in patients with thrombocythemia and myeloproliferative disorders, without altering the leukocyte count or hemoglobin level."

How anagrelide produces thrombocytopenia is not known. Its use in the long-term control of platelet counts appears promising, although further studies are needed, including a larger prospective trial.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Bernard Leon Albert, MD, Scarsdale. Died May 14, 1988; age 55. Dr Albert was a 1958 graduate of the George Washington School of Medicine, Washington, DC. He was a Diplomate of the American Board of Internal Medicine. His memberships included the New York Academy of Medicine, the New York State Society of Internal Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Harold Stewart Belcher, MD, New York. Died April 18, 1988; age 95. Dr Belcher was a 1920 graduate of Cornell University Medical College, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Henry Kingsley Blake, MD, Clearwater, Florida. Died October 18, 1987; age 93. Dr Blake was a 1921 graduate of Columbia University College of Physicians and Surgeons, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Morton Lester Davidson, MD, New York. Died April 8, 1988; age 79. Dr Davidson was a 1932 graduate of the New York University School of Medicine. He was a member of the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

J. Louise Despert, MD, Southampton. Died June 14, 1988; age 96. Dr Despert was a 1932 graduate of the New York University School of Medicine. Her memberships included the American Psychiatric Association, the American Academy of Child Psychiatry, the New York County Medical Society, and the Medical Society of the State of New York.

Edmund B. Dombrowski, MD, Brooklyn. Died June 7, 1988; age 70. Dr Dombrowski was a 1943 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Neurological Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Ned Doscher, MD, Southampton. Died June 4, 1988; age 70. Dr Doscher was a 1943 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Jan Ehrenwald, MD, Somers. Died June 15, 1988; age 88. Dr Ehrenwald was a 1925 graduate of the Deutsche Universzita Medizinische Fakulta, Praha, Czechoslovakia. He was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Irving J. Feuer, MD, Larchmont. Died June 8, 1988; age 82. Dr Feuer was a 1931 graduate of the Faculty of Medicine University of Edinburgh, Edinburgh, Scotland. He was a Fellow of the American College of Allergists and a member of the New York County Medical Society and the Medical Society of the State of New York.

Merrill Newton Foote, MD, Delray Beach, Florida. Died June 19, 1988; age 99. Dr Foote was a 1916 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the New York Gastroenterological Society, the American Thyroid Association, the Pan American Medical Association, the American Surgical Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Fausto Aldo Franchini, MD, Utica. Died June 21, 1988; age 73. Dr Franchini was a 1941 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. He was a member of the New York Academy of Medicine, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

George W. Fugitt, MD, Tonawanda. Died May 30, 1988; age 68. Dr Fugitt was a 1945 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology. His memberships included the New York State Surgical Society, the American Urological Association, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Bennett Lewis Gemson, MD, New Hyde Park. Died May 26, 1988; age 68. Dr Gemson was a 1944 graduate of Cornell University Medical College, New York. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the American Public Health Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Samuel Thomas Glasser, MD, North Palm Beach, Florida. Died September 14, 1987; age 86. Dr Glasser was a 1928 graduate of McGill University Faculty of Medicine, Montreal, Canada. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Morton Chester Gluck, MD, New York. Died April 9, 1988; age 57. Dr Gluck was a 1957 graduate of the New York University School of Medicine, New York. He was a Fellow of the American College of Physicians and the American College of Chest Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the American Thoracic Society, the New York County Medical Society, and the Medical Society of the State of New York.

Howard Grossman, MD, Brooklyn. Died June 6, 1988; age 63. Dr Grossman was a 1951 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Diplomate of the American Board of Internal Medicine. His memberships included the American Occupational Medical Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Kenneth Allen Handcock, MD, Slingerlands. Died June 9, 1988; age 60. Dr Handcock was a 1956 graduate of the Medical School, University of Otago, Dunedin, New Zealand. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the American Academy of Child Psychiatry, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Sidney S. Heilweil, MD, Hallandale, Florida. Died May 16, 1988; age 80. Dr Heilweil was a 1932 graduate of the New York Medical College. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

George Carl Hennig, MD, New York. Died May 4, 1988; age 74. Dr Hennig was a 1940 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York Gastroenterological Society, the American Gastro-enterological Association, the New York County Medical Society, and the Medical Society of the State of New York.

Marian M. Konczakowski, MD, Orchard Park. Died June 6, 1988; age 70. Dr Konczakowski was a 1943 graduate of Loyola University Stritch School of Medicine, Maywood, Illinois. She was a member of the New York Academy of Medicine, the Erie County Medical Society, and the Medical Society of the State of New York.

Robert A. Lane, MD, Syosset. Died June 6, 1988; age 64. Dr Lane was a 1951 graduate of the New York Medical College, New York. He was a member of the Academy of Psychosomatic Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Esther Pei-Cheng Lim, MD, Great Neck. Died May 28, 1988; age 67. Dr Lim was a 1946 graduate of the National Central University Faculty of Medicine, Shanghai, Kiangsu, China. She was a Fellow of the American College of Anesthesiologists and a Diplomate of the American Board of Anesthesiology. Her memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Charles Ignatius Maggio, MD, Rochester. Died May 22, 1988; age 98. Dr Maggio was a 1917 graduate of the University of Michigan Homeopathic Medical School, Ann Arbor. His memberships included the New York Academy of Medicine, the American Occupational Medical Association, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

William Peter McKenna, MD, Ellenburg Depot. Died May 23, 1988; age 82. Dr McKenna was a 1934 graduate of the Albany Medical College of Union University, Albany. He was a member of the Medical Society of the County of Clinton and the Medical Society of the State of New York.

Adolph Mechner, MD, New York. Died June 4, 1988; age 91. Dr Mechner was a 1925 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Albert Herbert Meyer, MD, Great Neck. Died June 19, 1988; age 78. Dr Meyer was a 1936 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Sol Nichtern, MD, New York. Died June 6, 1988; age 68. Dr Nichtern was a 1944 graduate of the Chicago Medical School, Chicago. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the American Academy of Child Psychiatry, the American Society of Adolescent Psychiatry, the New York County Medical Society, and the Medical Society of the State of New York.

Virginia Kipke Pierce, MD, New York. Died February 1, 1988; age 75. Dr Pierce was a 1941 graduate of the University of Illinois at Chicago Health Sciences Center, Chicago. She was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Obstetrics and Gynecology. Her memberships included the New York State Surgical Society, the New York Academy of Medicine, the American Medical Women's Association, the Society of Surgical Oncology, the New York County Medical Society, and the Medical Society of the State of New York.

Anthony Paul Pirundini, MD, Brooklyn. Died April 27, 1988; age 60. Dr Pirundini was a 1955 graduate of the New York University School of Medicine, New York. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. He was a member of the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Fellowes Morgan Pruyn, MD, Mount Kisco. Died June 1, 1988; age 79. Dr Pruyn was a 1935 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine. His memberships included the New York Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

William George Rocktaschel, MD, Pittsford. Died May 23, 1988; age 90. Dr Rocktaschel was a 1934 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Fellow of the American Academy of Family Physicians. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Paul S. Romano, MD, Bronx. Died June

10, 1988; age 81. Dr Romano was a 1931 graduate of the New York University School of Medicine, New York. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

John Eugene Santeramo, MD, Rockville Centre. Died June 15, 1988; age 69. Dr Santeramo was a 1944 graduate of the New York Medical College, New York. He was a Fellow of the American College of Anesthesiologists and a Qualified Fellow of the International College of Surgeons. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Nassau County Medical Society, and the Medical Society of the State of New York.

Andrew I. E. Schildhaus, MD, Boca Raton, Florida. Died May 29, 1988; age 73. Dr Schildhaus was a 1939 graduate of the University of Vermont College of Medicine, Burlington. He was a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the American Academy of Orthopaedic Surgeons, the Pan American Medical Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Charles Thomas Scibetta, MD, Eggertsville. Died May 20, 1988; age 74. Dr Scibetta was a 1939 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. Dr Scibetta was a member of the New York Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Sheppard Siegal, MD, New York. Died June 18, 1988; age 79. Dr Siegal was a 1932 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine and the American Board of Allergy and Immunology. He was a member of the American Academy of Allergy, the New York County Medical Society, and the Medical Society of the State of New York.

Douglas George Smiley, MD, Bronx. Died May 31, 1988; age 71. Dr Smiley was a 1940 graduate of the New York University School of Medicine, New York. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the American Society of Abdominal Surgeons, the Bronx County Medical Society, and the Medical Society of the State of New York.

David Steinman, MD, Scarsdale. Died March 9, 1988; age 61. Dr Steinman was a 1956 graduate of the Johns Hopkins University School of Medicine, Baltimore. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

James D. Tyner, MD, Newark. Died April 29, 1988; age 83. Dr Tyner was a 1929 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Fellow of the American Academy of Family Physicians. He was a member of the Medical Society of the County of Wayne and the Medical Society of the State of New York.

Frank Weiss, MD, Bronx. Died June 18, 1988; age 80. Dr Weiss was a 1936 graduate of Rush Medical College, Chicago. He was a Diplomate of the American Board of Internal Medicine. His memberships included the New York Academy of Medicine, the Bronx County Medical Society, and the Medical Society of the State of New York.

Joel L. Ziprin, MD, New York. Died June 12, 1988; age 88. Dr Ziprin was a 1927 graduate of the New York Medical College, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

ERRATUM

Based on information provided by the Kings County Medical Society, the obituary notice for Dr Samuel Lewis Cohen which appeared in the July 1988 issue of the *Journal* contained incorrect biographical information. A corrected notice will appear in a future issue.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456–462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Residents / Fellows

A $500 AWARD will be presented by the Medical Society of the State of New York to the intern, resident or fellow who submits the best paper on a clinical subject for the Society's 1988 Merit H. Cash Competition.

Entrants need not be members of the MSSNY but must be in training at a hospital within the State of New York.

Entries should be "sponsored" by a faculty member, and all papers submitted should be of sufficient quality to be considered for original publication in the *New York State Journal of Medicine.*

The deadline for receipt of entries is December 30, 1988, and the prize for the winning paper will be awarded at the MSSNY's annual meeting in April of 1989.

Address all entries to: Division of Scientific Activities
Medical Society of the State of New York
420 Lakeville Road
P.O. Box 5404
Lake Success, NY 11042

Questions? Contact either the Division of Scientific Activities or MSSNY's Resident Physicians Section at (516) 488-6100.

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Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

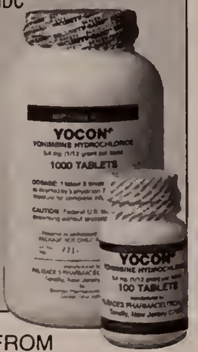
Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85



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(*continued from p 4A*)

Oct 14. **Cosmetology Symposium.** Beth Israel Medical Center, New York. Contact: Beverley R. Baptiste, Medical Education Office, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2849.

Oct 10–14. **Five-Day Emergency Medicine Board Review Course.** 38 Cat I Credits. NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Oct 12–15. **Renal Biopsy in Medical Diseases of the Kidneys.** 24 Cat I Credits. College of Physicians and Surgeons, New York. Contact: Center for Continuing Education, College of Physicians and Surgeons, 630 West 168th St, New York, NY 10032. Tel: (212) 305-3682.

Oct 13–15. **The Psychobiology of Human Eating Disorders.** Sheraton Centre. *Oct 18–20.* **The L. W. Frohlich Award Conference—Under the Volcano: Biomedical Science and the Third World.** The Rockefeller University. *Oct 31–Nov 2.* **Vitamin E: Biochemistry & Health Implications.** Sheraton Centre. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

Oct 13–16. **Fourth Annual International Symposium on Acupuncture and Electro-Therapeutics.** 40 Cat I Credits; accredited by New York State Boards for Medicine and Dentistry for 25 credit hours toward 300-hour Acupuncture Certificate requirement. School of International Affairs, Columbia University. Contact: Y. Omura, MD, ScD. 800 Riverside Drive (8-I), New York, NY 10032. Tel: (212) 781-6262.

Oct 28. **Dr Robert K. Match Distinguished Scholar Program: New Frontiers in Cardiac Imaging.** 8 Cat I Credits. The Grand Hyatt Hotel, New York. Contact: Anne J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 10042. Tel: (718) 470-8650.

Oct 28. **New Biological Vistas on Schizophrenia.** 6 Cat I Credits. New York Academy of Medicine. Contact: Office of Continuing Medical Education, Albert Einstein College of Medi-

(*continued on p 22A*)

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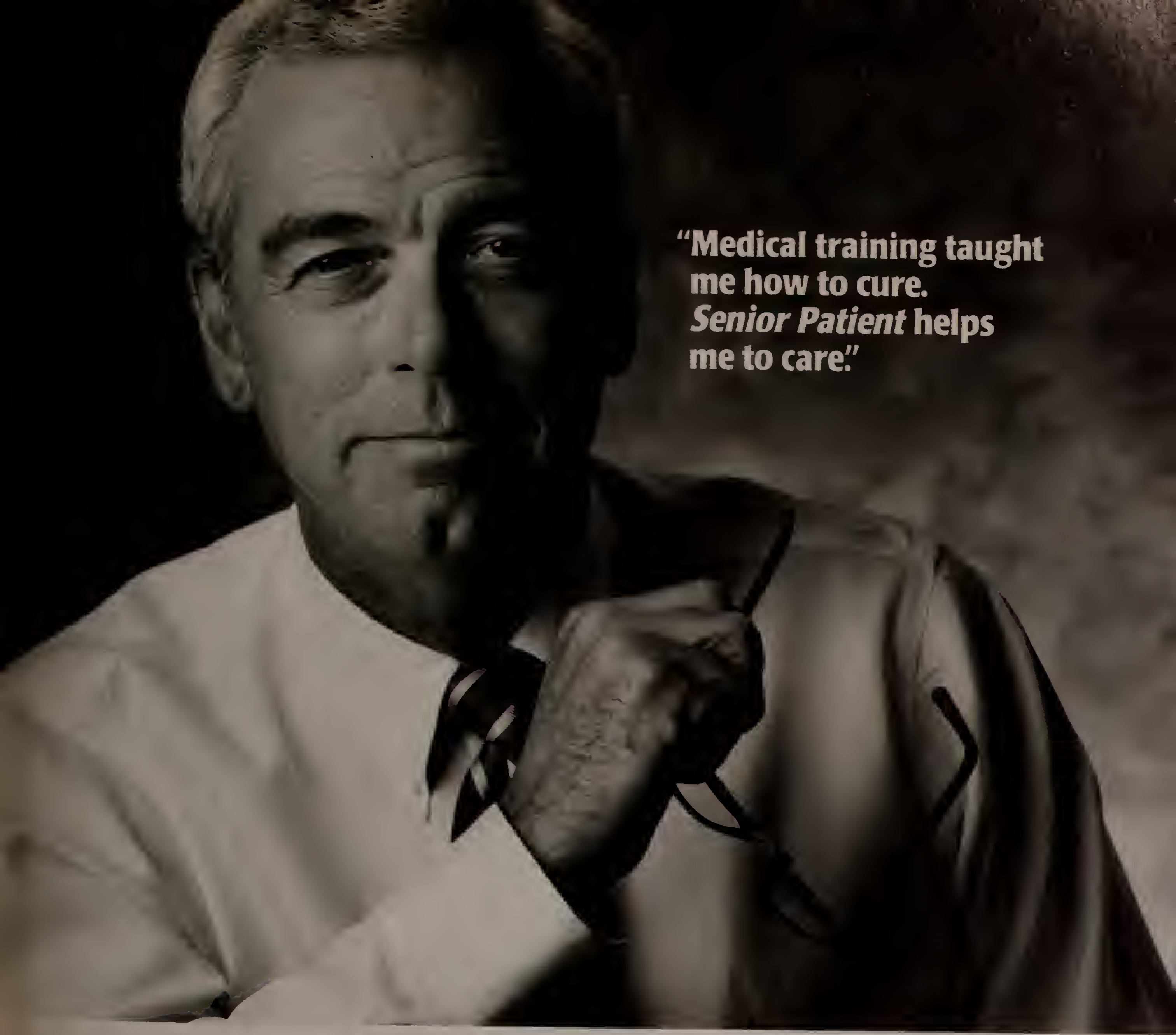
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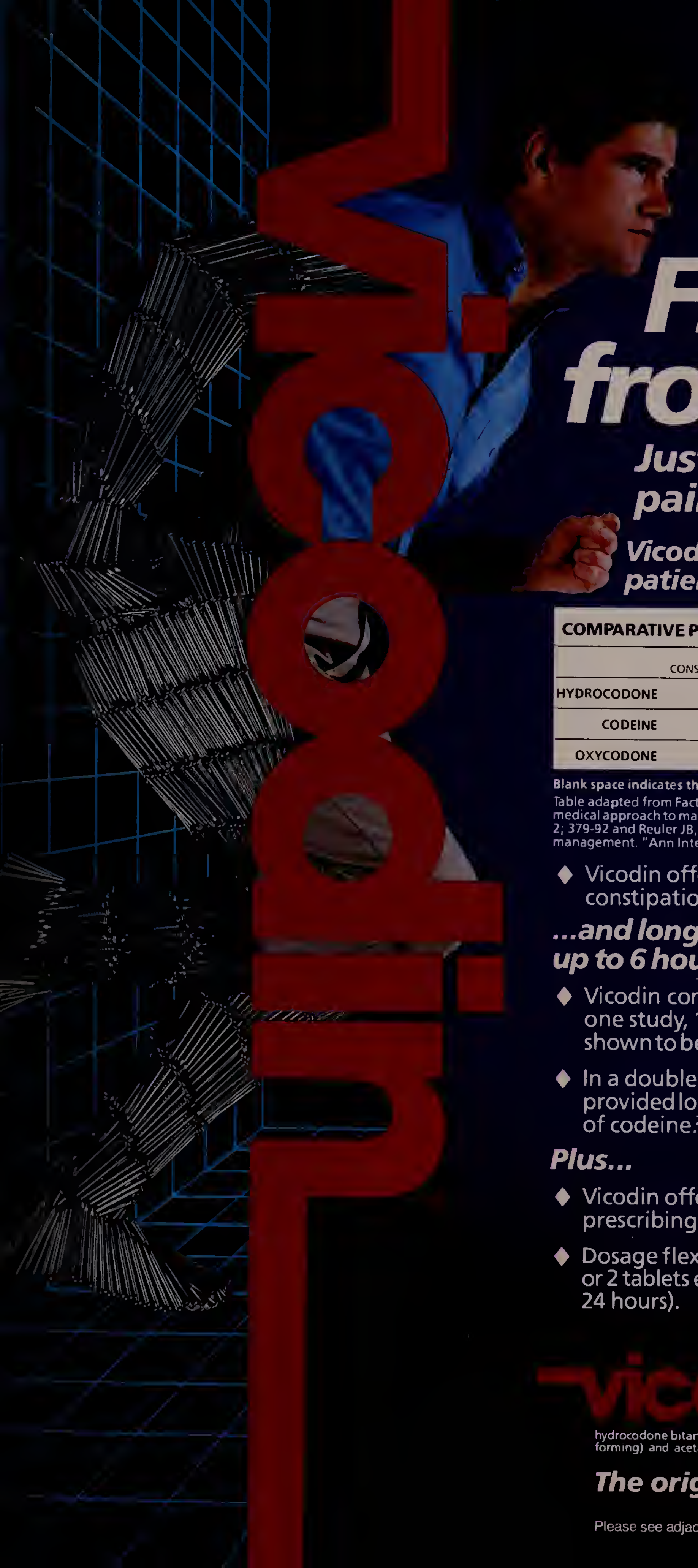

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OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB The medical approach to management of pain caused by cancer "Semin Oncol" 1975, 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome. misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

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1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med,* 141:293-300, 1981.

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CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.
WARNINGS:
Allergic-Type Reaction: VICODIN contains sodium metabisulfite, a sulfite that may cause allergic-type reactions including anaphylactic symptoms and life-threatening or less severe asthmatic episodes in certain susceptible people. The overall prevalence of sulfite sensitivity in the general population is unknown and probably low. Sulfite sensitivity is seen more frequently in asthmatic than non-asthmatic people.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers. Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing. If significant respiratory depression occurs, it may be antagonized by the use of naloxone hydrochloride. (see ADVERSE REACTIONS: Respiratory Depression).
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS:
Special Risk Patients: As with any narcotic analgesic agent, VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture. The usual precautions should be observed and the possibility of respiratory depression should be kept in mind.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
Cough Reflex: Hydrocodone suppresses the cough reflex; as with all narcotics, caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: Patients receiving other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants (including alcohol) concomitantly with VICODIN may exhibit an additive CNS depression. When combined therapy is contemplated, the dose of one or both agents should be reduced.
The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The withdrawal signs include irritability and excessive crying, tremors, hyperactive reflexes, increased respiratory rate, increased stools, sneezing, yawning, vomiting, and fever. The intensity of the syndrome does not always correlate with the duration of material opioid use or dose. There is no consensus on the best method of managing withdrawal. Chlorpromazine 0.7 to 1.0 mg/kg q6h, and paregoric 2 to 4 drops/kg q4h, have been used to treat withdrawal symptoms in infants. The duration of therapy is 4 to 28 days, with the dosage decreased as tolerated.
Labor and Delivery: As with all narcotics, administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from VICODIN, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS:
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients. The antiemetic phenothiazines are useful in suppressing these effects; however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia. Prolonged administration of VICODIN may produce constipation.
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Respiratory Depression: (See WARNINGS.)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. However, tolerance to hydrocodone can develop with continued use and the incidence of untoward effects is dose related.
The usual adult dosage is one or two tablets every four to six hours as needed for pain. The total 24 hour dose should not exceed 8 tablets.

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(continued from p 14A)
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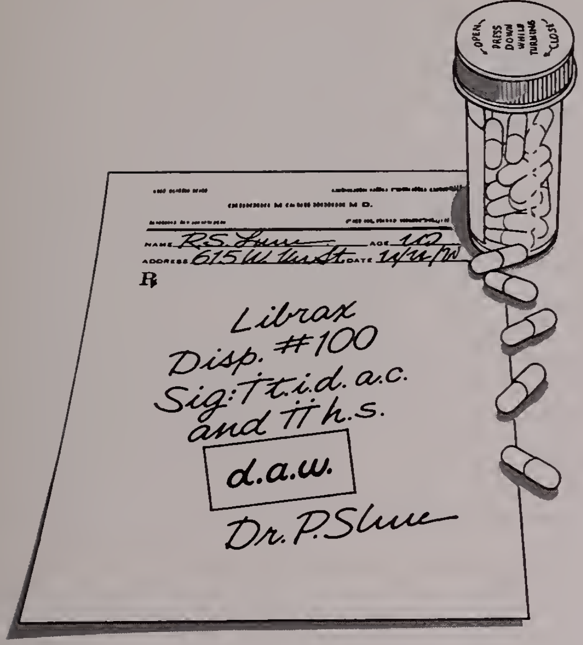

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Antispasmodic

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

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*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of peptic ulcer and IBS.

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For patients with elevated cholesterol

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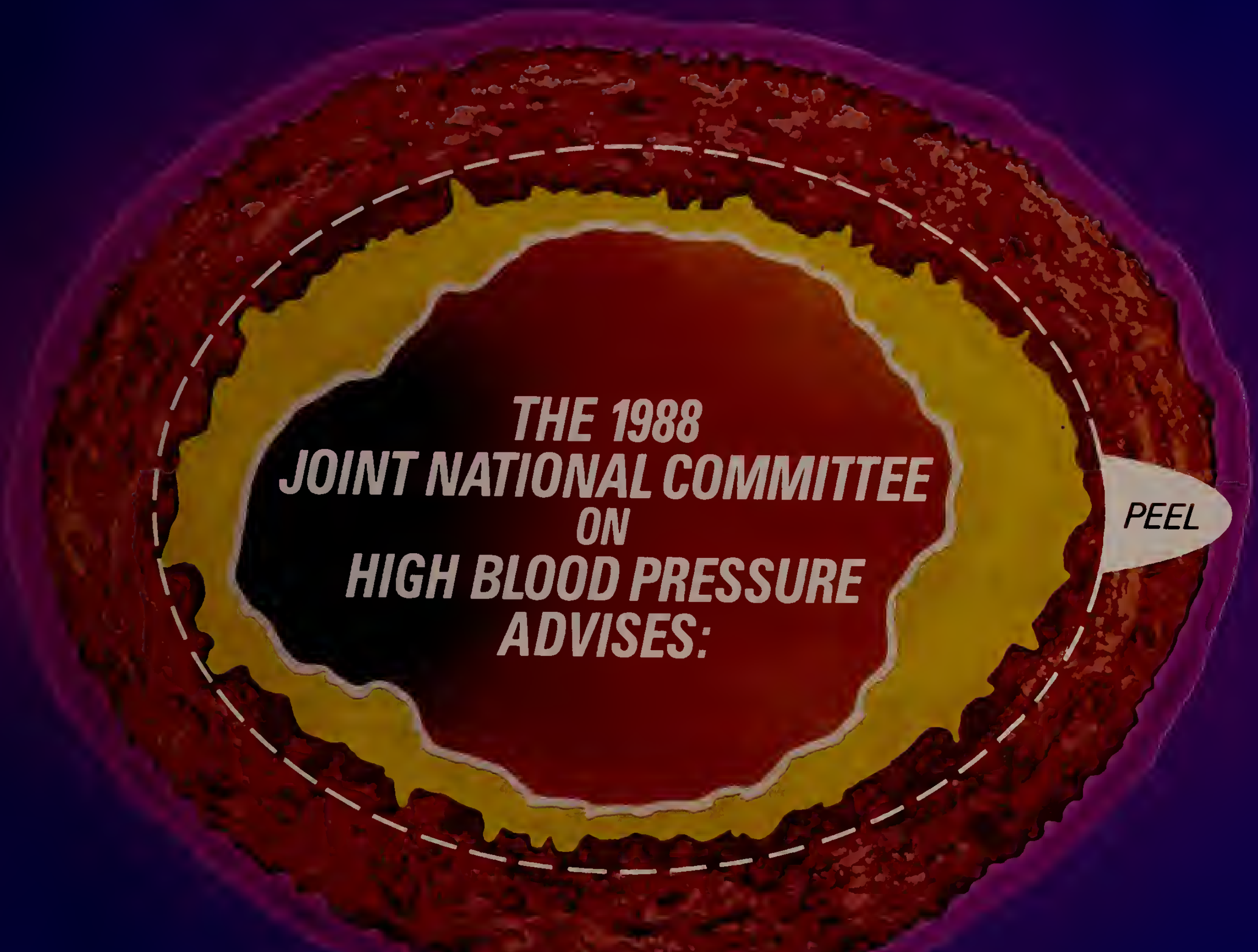

Superior lipids performance in hypertensive patients with elevated cholesterol[1,2]*

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HDL cholesterol	↔ ↑	↔	↔	↔ ↓	↓
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*HYTRIN is not indicated for the treatment of hyperlipidemia.

†*Average wholesale price.*

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(terazosin hydrochloride tablets)

Brief Summary

CLINICAL PHARMACOLOGY: Pharmacodynamics: Clinical studies of terazosin used in once-a-day (majority) and b.i.d regimens with total doses usually in the range of 5-20mg/day, in patients with mild or moderate hypertension. Because terazosin, like all alpha antagonists, can cause large falls in blood pressure after the first dose or first few doses, the initial dose was 1mg in virtually all studies, with subsequent titration to a specified fixed dose or titration to a specified blood pressure end point.

Blood pressure responses were measured at the end of the dosing interval (usually 24 hrs.) and effects were shown to persist throughout the interval, with usual supine responses 5-10mmHg systolic and 3.5-8mmHg diastolic greater than placebo. The responses in the standing position tended to be somewhat larger, although this was not true in all studies. The magnitude of blood pressure responses was similar to prazosin and less than hydrochlorothiazide (in a single study). In measurements 24 hrs. after dosing, heart rate was unchanged.

Limited measurements of peak response (2-3 hrs. after dosing) during chronic terazosin administration indicate that it is more than twice the trough (24 hr.) response, suggesting some attenuation of response at 24 hrs., presumably due to a fall in blood terazosin concentrations at the end of the dose interval. This explanation is not established with certainty and is not consistent with the similarity of blood pressure response to once-a-day and b.i.d. dosing. With the absence of an observed dose-response relationship over a range of 5-20mg, i.e., if blood concentrations fall to the point of providing less than full effect at 24 hrs., a shorter dosing interval or larger dose should lead to increased response. Measure blood pressure (BP) at the end of the dose interval; if response is not satisfactory, patients may be tried on a larger dose or b.i.d. regimen. The latter should be considered if side effects, such as dizziness, palpitations, or orthostatic complaints, are seen within a few hours after dosing.

The greater BP effect associated with peak plasma concentrations (first few hours after dosing) appears somewhat more position-dependent (greater in the erect position) than the effect of terazosin at 24 hrs. In the erect position there is a 6-10 bpm increase in heart rate in the first few hours after dosing. During the first 3 hrs. after dosing 12.5% of patients had a systolic pressure fall of 30mmHg or more from supine to standing, or standing systolic pressure below 90mmHg with a fall of at least 20mmHg, compared to 4% of a placebo group.

INDICATIONS AND USAGE: Indicated for the treatment of hypertension.

CONTRAINDICATIONS: None known.

WARNINGS: Syncope and "First-dose" Effect: Terazosin, like other alpha-adrenergic blocking agents, can cause marked hypotension, especially postural hypotension, and syncope in association with the first dose or first few doses. A similar effect may occur if therapy is interrupted for more than a few doses. Syncope has been reported with other alpha-adrenergic blocking agents in association with rapid dosage increases or introduction of another antihypertensive drug. Syncope may be due to an excessive postural hypotensive effect, although occasionally the syncopal episode has been preceded by severe supraventricular tachycardia with heart rates of 120-160 bpm.

To decrease the likelihood of syncope or excessive hypotension, always initiate treatment with a 1mg dose at bedtime. The 2mg and 5mg tablets are not indicated as initial therapy. Increase dosage slowly, and add additional antihypertensive agents with caution. Caution patients to avoid situations where injury could result if syncope occurs during initiation of therapy.

In early studies, where increasing single doses up to 7.5mg were given at 3 day intervals, tolerance to the first dose phenomenon did not necessarily develop and the "first dose" effect was observed at all doses. Syncopal episodes occurred in 3 of 14 subjects given doses of 2.5, 5, and 7.5mg, which are higher than the recommended initial dose. Severe orthostatic hypotension (BP 50/0mmHg) was seen in two others and dizziness, tachycardia, and lightheadedness occurred in most subjects. These adverse effects all occurred within 90 min. of dosing.

In multiple dose clinical trials involving nearly 2000 patients, syncope was reported in about 1% of patients, in no case severe or prolonged, and was not necessarily associated with early doses.

If syncope occurs, place patient in recumbent position and treat supportively. There is evidence that the orthostatic effect of terazosin is greater, even in chronic use, shortly after dosing.

PRECAUTIONS: ***General Orthostatic Hypotension:*** While syncope is the most severe orthostatic effect of terazosin, other symptoms of lowered BP, such as dizziness, lightheadedness and palpitations, are more common, occurring in 28% of patients in clinical trials. Patients with occupations in which such events represent potential problems should be treated with particular caution.

Information for Patients: Make aware of possibility of syncopal and orthostatic symptoms, especially at initiation of therapy, and to avoid driving or hazardous tasks for 12 hrs. after the first dose, after a dosage increase, and after interruption of therapy when treatment is resumed. Caution to avoid situations where injury could result should syncope occur during initial therapy. Advise to sit or lie down when symptoms of lowered BP occur and to rise carefully from a sitting or lying position. Bothersome dizziness, lightheadedness, or palpitations should be reported to physician.

Tell patients that drowsiness or somnolence can occur, requiring caution in people who must drive or operate heavy machinery.

Laboratory Tests: Small but statistically significant decreases in hematocrit, hemoglobin, WBC, total protein and albumin were observed in clinical trials. The magnitude of decreases did not worsen with time. These findings suggest the possibility of hemodilution.

Drug Interactions: In controlled trials, terazosin was added to diuretics, and several beta-adrenergic blockers; no unexpected interactions were observed. Terazosin has also been used concomitantly without interaction in at least 50 patients on the following: 1) analgesic/anti-inflammatory (acetaminophen, aspirin, codeine, ibuprofen, indomethacin), 2) antibiotics (erythromycin, trimethoprim and sulfamethoxazole); 3) anticholinergic/sympathomimetics (phenylephrine HCl, phenylpropanolamine HCl, pseudoephedrine HCl); 4) antigout (allopurinol); 5) antihistamines (chlorpheniramine), 6) cardiovascular agents (atenolol, hydrochlorothiazide, methyclothiazide, propranolol); 7) corticosteroids, 8) gastrointestinal agents (antacids), 9) hypoglycemics, 10) sedatives and tranquilizers (diazepam).

Carcinogenesis, Mutagenesis, Impairment of Fertility: HYTRIN was devoid of mutagenic potential when evaluated *in vivo* and *in vitro*.

HYTRIN, administered in feed to rats at doses of 8, 40, and 250mg/kg/day for 2 yrs., was associated with a statistically significant increase in benign adrenal medullary tumors of male rats exposed to the 250mg/kg dose. This dose is 695 X max. recommended human dose (20mg/55kg). Female rats were unaffected. HYTRIN was not oncogenic in mice when administered in feed for 2 yrs. at a maximum tolerated dose of 32mg/kg/day.

The absence of mutagenicity in a battery of tests, of tumorigenicity of any cell type in the mouse carcinogenicity assay, of increased total tumor incidence in either species, and of proliferative adrenal lesions in female rats, suggests a male rat species-specific event. Numerous other diverse pharmaceutical and chemical compounds have been associated with these tumors in male rats without supporting evidence for carcinogenicity in man.

Effects on fertility were assessed in a standard fertility/reproductive performance study in which male and female rats were administered oral doses of 8, 30 and 120mg/kg/day. Four of 20 male rats given 30mg/kg and 5 of 19 male rats given 120mg/kg failed to sire a litter. Testicular weights and morphology were unaffected. Vaginal smears at 30 and 120mg/kg/day appeared to contain less sperm than smears from control matings and good correlation was reported between sperm count and subsequent pregnancy.

Oral use for 1 or 2 yrs. elicited a statistically significant increase in testicular atrophy in rats exposed to 40 and 250mg/kg/day, but not in rats exposed to 8mg/kg/day (> 20 X max. recommended human dose). Testicular atrophy was observed in dogs dosed with 300mg/kg/day (> 800 X max. recommended human dose) for 3 months but not after 1 yr. when dosed with 20mg/kg/day. This lesion has also been seen with Minipress®.

Pregnancy: Teratogenic effects: Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women and the safety of terazosin in pregnancy has not been established. HYTRIN is not recommended during pregnancy unless potential benefit justifies potential risk to mother and fetus.

Nonteratogenic effects: In a peri- and post-natal development study in rats, significantly more pups died in the group dosed with 120mg/kg/day (> 300 X max. recommended human dose) than in the control group during the 3-week post-partum period.

Nursing Mothers: It is not known whether terazosin is excreted in breast milk, therefore, exercise caution when administering terazosin to a nursing woman.

Pediatric Use: Safety and effectiveness have not been determined.

ADVERSE REACTIONS: The prevalence of adverse reactions has been ascertained from 14 placebo-controlled studies conducted primarily in the U.S. The studies involved once-a-day administration of terazosin as monotherapy or in combination with other antihypertensive agents, at doses ranging from 1 to 40mg. All adverse events reported during these studies were recorded as adverse reactions. Adverse events where the prevalence rate in the terazosin group was at least 5%, where the prevalence rate for the terazosin group was at least 2% and was greater than the prevalence rate for the placebo group, or where the reaction is of particular interest are summarized below. Only asthenia, blurred vision, dizziness, nasal congestion, nausea, peripheral edema, palpitations and somnolence were significantly ($p<0.05$) more common in patients receiving terazosin than in patients receiving placebo. Other events include **[%TERAZOSIN-%PLACEBO]**: asthenia (11.3%-4.3%), back pain (2.4%-1.2%), blurred vision (1.6%-0%), depression (0.3%-0.2%), dizziness (19.3%-7.5%), dyspnea (3.1%-2.4%), edema (0.9%-0.6%), headache (16.2%-15.8%), impotence (1.2%-1.4%), libido decreased (0.6%-0.2%), nasal congestion (5.9%-3.4%), nausea (4.4%-1.4%), nervousness (2.3%-1.8%), pain-extremities (3.5%-3%), palpitations (4.3%-1.2%), paresthesia (2.9%-1.4%), peripheral edema (5.5%-2.4%), postural hypotension (1.3%-0.4%), sinusitis (2.6%-1.4%), somnolence (5.4%-2.6%), tachycardia (1.9%-1.2%), weight gain (0.5%-0.2%).

Adverse reactions were usually mild or moderate in intensity but sometimes were serious enough to interrupt treatment. Adverse reactions that were most bothersome as judged by being reported as reasons for discontinuation of therapy by at least 0.5% of the terazosin group and being reported more often than in the placebo group **[%TERAZOSIN-%PLACEBO]** are: asthenia (1.6%-0%), blurred vision (0.6%-0%), dizziness (3.1%-0.4%), dyspnea (0.9%-0.6%), headache (1.3%-1%), nasal congestion (0.6%-0%), nausea (0.8%-0%), palpitations (1.4%-0.2%), paresthesia (0.8%-0.2%), peripheral edema (0.6%-0%), postural hypotension (0.5%-0%), somnolence (0.6%-0.2%), syncope (0.5%-0.2%), tachycardia (0.6%-0%).

Additional adverse reactions have been reported, but these are not distinguishable from symptoms that might have occurred in the absence of exposure to terazosin. The following additional adverse reactions were reported by at least 1% of 1987 patients who received terazosin in clinical studies or during marketing experience: abdominal pain, abnormal vision, anxiety, arrhythmia, arthralgia, arthritis, bronchitis, chest pain, cold symptoms, conjunctivitis, constipation, diarrhea, dry mouth, dyspepsia, epistaxis, facial edema, fever, flatulence, flu symptoms, gout, increased cough, insomnia, joint disorder, myalgia, neck pain, pharyngitis, pruritus, rash, rhinitis, shoulder pain, sweating, tinnitus, urinary frequency, urinary tract infection, vasodilation, vomiting.

DOSAGE AND ADMINISTRATION: Dose and dose interval (12 or 24 hrs.) should be adjusted according to BP response.

Initial Dose: 1mg at bedtime. Observe the initial dosing regimen strictly to minimize potential for severe hypotensive effects.

Subsequent Doses: Slowly increase dose to achieve desired BP response. Usual dose range is 1mg to 5mg once a day. Some patients may benefit from doses up to 20mg/day. Doses over 20mg do not appear to provide further BP effect. Doses over 40mg have not been studied. Monitor BP at the end of dosing interval to assure control is maintained. It may be helpful to measure BP 2-3 hrs. after dosing to see if maximum and minimum responses are similar, and to evaluate symptoms which can result from excessive hypotensive response. If response is substantially diminished at 24 hrs., consider an increased dose or b.i.d. regimen. **If administration is discontinued for several days or longer, reinstitute therapy using initial dosing regimen.** In clinical trials, except for the initial dose, the dose was given in the morning.

Use With Other Drugs: Caution should be observed when terazosin is administered concomitantly with other antihypertensive agents (e.g., calcium antagonists) to avoid the possibility of significant hypotension. When adding a diuretic or other antihypertensive agent, dosage reduction and retitration may be necessary.

August, 1988 **Abbott Health Care Products, Inc.** North Chicago, IL 60064 8083873

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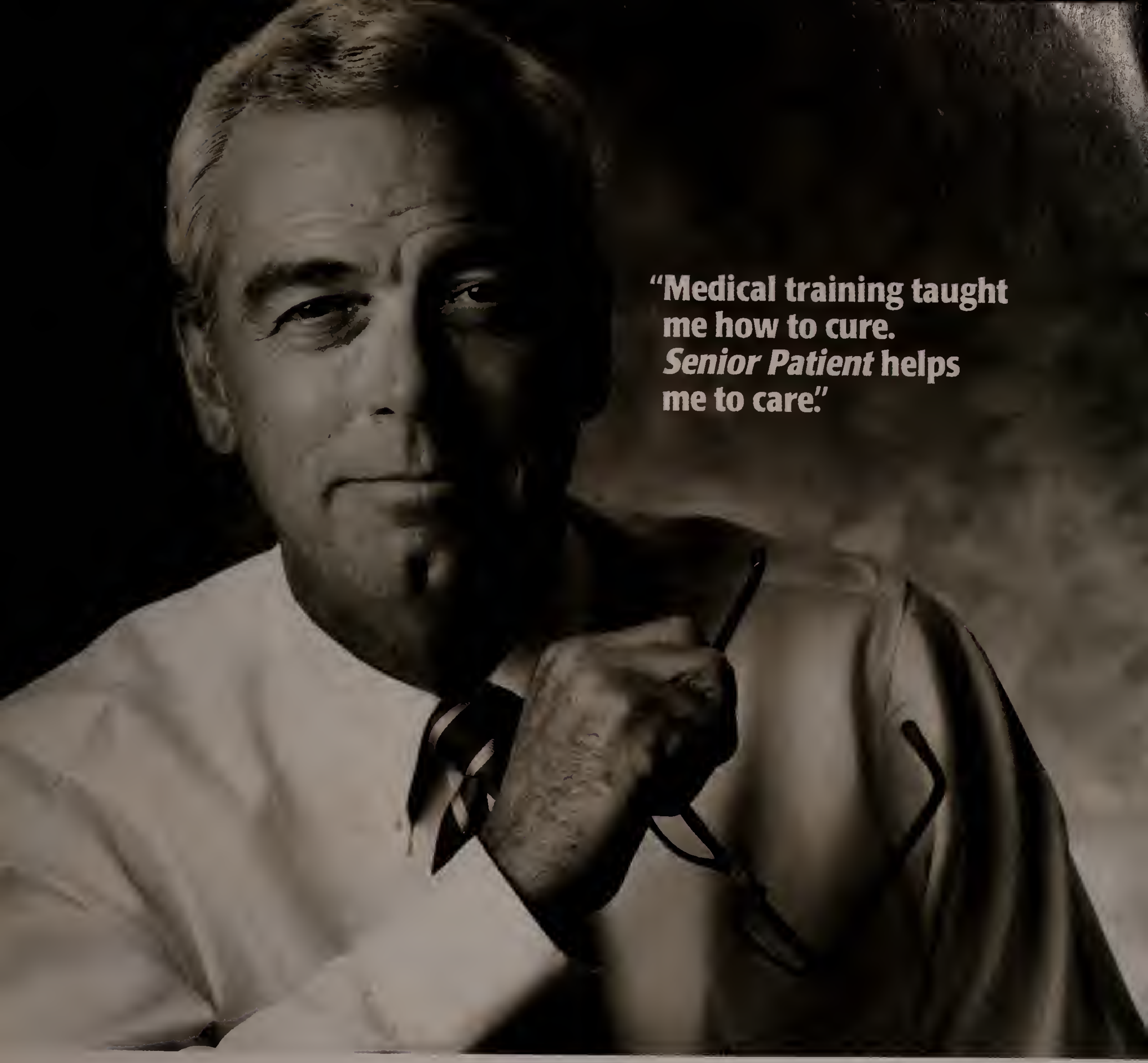
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MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

NOVEMBER 1988

AROUND THE STATE

ALBANY

Nov 3–4. **The Use of the Saphenous Vein In-Situ as an Arterial Bypass.** Desmond Americana & Albany Medical College. Contact: Lynne Wechsler, Office of Continuing Medical Education, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208.

Nov 9–10. **The Second Conference on Geriatric Medicine and Rehabilitation.** Desmond Americana. Contact: Barbara Post, Sunnyview Hospital and Rehabilitation Center, 1270 Belmont Ave, Schenectady, NY 12308. Tel: (518) 382-4595.

COOPERSTOWN

Nov 9. **Focus on Sexually Transmitted Diseases.** 3 Cat I Credits. Bassett Hall Conference Center. Contact: Charlotte Hoag, Medical Education, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326. Tel: (607) 547-3926.

EAST MEADOW

Nov 5. **The Poisoned Patient: Emergency Toxicology.** 6 Cat I Credits. Nassau County Medical Center. Contact: Howard C. Mofenson, MD, Poison Control, Nassau County Medical Center, East Meadow, NY 11554. Tel: (516) 542-3707.

GARDEN CITY

Nov 30. **A Clinical Day in Ophthalmology: Glaucoma Topics.** 7 Cat I Credits. Garden City Hotel. Contact: Anne Boehme, CMP, Associate Director for Continuing Medical Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

(*continued on p 12A*)

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INTERNIST BE/BC: North Shore Internal Medicine, PC is seeking an energetic general internist to enjoy the benefits of a rapidly expanding practice. New office close to hospital. Michigan State Medical School Campus. Send resume to 2420 First Avenue South, Escanaba, MI 49829 (906) 786-1563.

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FAMILY PRACTITIONER needed for established clinic, located in the Black Hills. New hospital and clinic. Contact Dr. Lanny Reimer at (307) 746-3582 or Evonne Ulmer, C.E.O. at Weston County Hospital, 1124 Washington Blvd., Newcastle, WY 82701 (307) 746-4491. Opportunities also available for General Surgeon-Internal Medicine.

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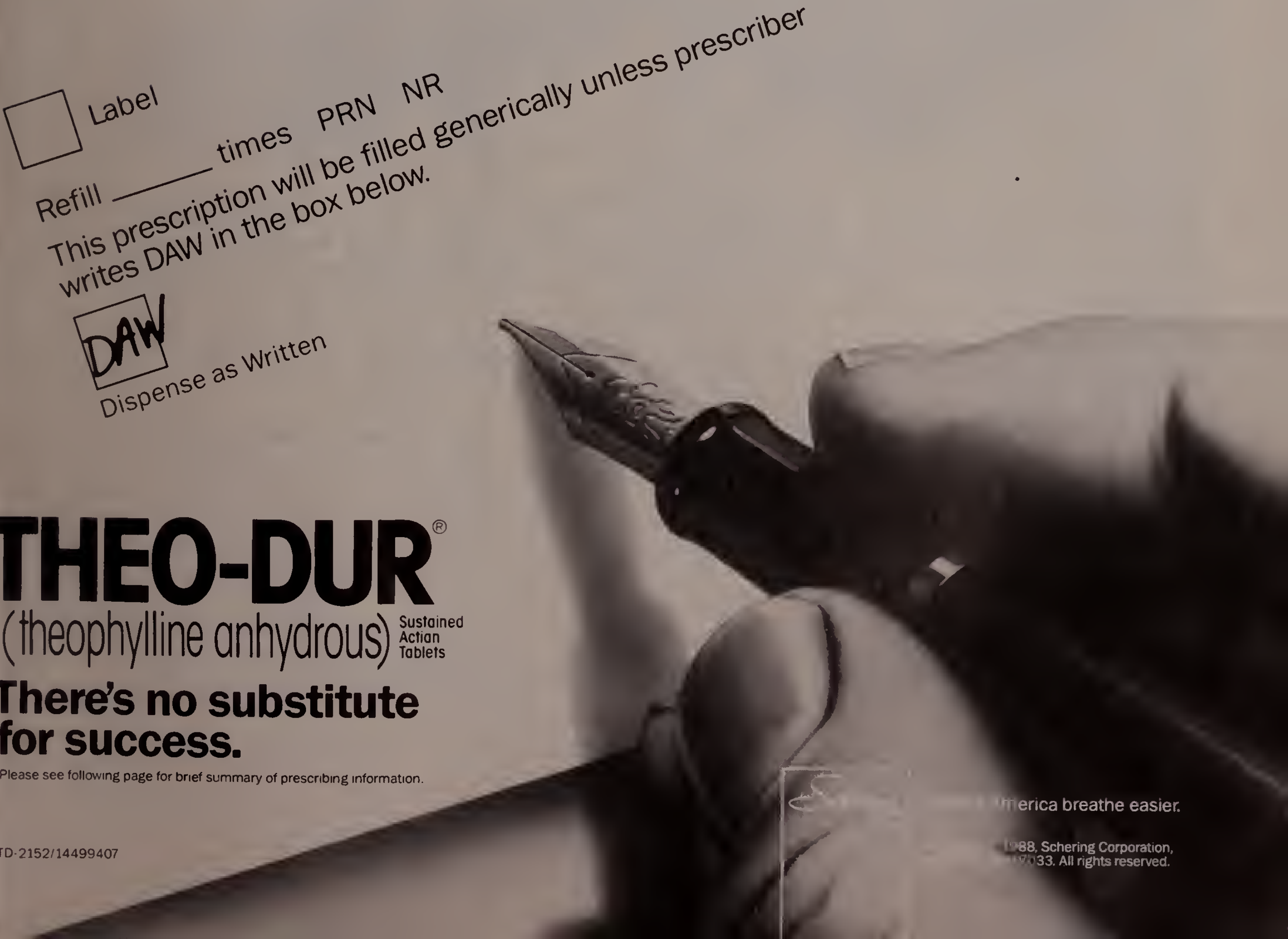
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(theophylline anhydrous) Sustained Action Tablets

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THEO-DUR®

THEOPHYLLINE (Anhydrous)
Sustained Action Tablets

INDICATIONS: THEO-DUR is indicated for relief and/or prevention of symptoms of asthma and for reversible bronchosoasm associated with chronic bronchitis and emphysema

CONTRAINDICATIONS: THED-DUR is contraindicated in individuals who have shown hypersensitivity to theophylline or any of the tablet components

WARNINGS: Status asthmaticus should be considered a medical emergency and is defined as that degree of bronchospasm which is not rapidly responsive to usual doses of conventional bronchodilators. Optimal therapy for such patients frequently requires both *additional medication*, parenterally administered, and close *monitoring*, preferably in an intensive care setting

Although increasing the dose of theophylline may bring about relief, such treatment may be associated with toxicity The likelihood of such toxicity developing increases significantly when the serum theophylline concentration exceeds 20 mcg/ml. Therefore, determination of serum theophylline levels is recommended to assure maximal benefit without excessive risk

Serum levels above 20 mcg/ml are rarely found after appropriate administration of recommended doses. However, in individuals in whom theophylline plasma clearance is reduced *for any reason*, even conventional doses may result in increased serum levels and potential toxicity Reduced theophylline clearance has been documented in the following readily identifiable groups 1) patients with impaired renal or liver function, 2) patients over 55 years of age, particularly males and those with chronic lung disease, 3) those with cardiac failure from any cause, 4) neonates, and 5) those patients taking certain drugs (macrolide antibiotics and cimetidine). Decreased clearance of theophylline may be associated with either influenza immunization or active infection with influenza.

Reduction of dosage and laboratory monitoring is especially appropriate in the above individuals Less serious signs of theophylline toxicity (i e nausea and restlessness) may occur frequently when initiating therapy, but are usually transient, when such signs are persistent during maintenance therapy, they are often associated with serum concentrations above 20 mcg/ml. Unfortunately, however, serious side effects such as ventricular arrhythmias, convulsions or even death may appear as the first sign of toxicity without any previous warning Stated differently, *serious toxicity is not reliably preceded by less severe side effects*

Many patients who require theophylline may exhibit tachycardia due to their underlying disease process so that the cause/effect relationship to elevated serum theophylline concentrations may not be appreciated.

Theophylline products may cause dysrhythmia and/or worsen pre-existing arrhythmias and any significant change in rate and/or rhythm warrants monitoring and further investigation.

The occurrence of arrhythmias and sudden death (with histological evidence of necrosis of the myocardium) has been recorded in laboratory animals (minipigs, rodents and dogs) when theophylline and beta agonists were administered concomitantly, although not when either was administered alone The significance of these findings when applied to human usage is currently unknown

PRECAUTIONS: THED-DUR TABLETS SHOULD NOT BE CHEWED DR CRUSHED

General: Theophylline half-life is shorter in smokers than in non-smokers Therefore, smokers may require larger or more frequent doses Morphine and curare should be used with caution in patients with airway obstruction as they may suppress respiration and stimulate histamine release Alternative drugs should be used when possible Theophylline should not be administered concurrently with other xanthine medications. Use with caution in patients with severe cardiac disease, severe hypoxemia, hypertension, hyperthyroidism, acute myocardial injury, cor pulmonale, congestive heart failure, liver disease, in the elderly (especially males) and in neonates In particular, great caution should be used in giving theophylline to patients with congestive heart failure Frequently, such patients have markedly prolonged theophylline serum levels with theophylline persisting in serum for long periods following discontinuation of the drug. Individuals who are rapid metabolizers of theophylline, such as the young, smokers, and some non-smoking adults, may not be suitable candidates for once-daily dosing These individuals will generally need to be dosed at 12 hour or sometimes B hour intervals Such patients may exhibit symptoms of bronchospasm near the end of a dosing interval, or may have wider peak-to-trough differences than desired

Use theophylline cautiously in patients with history of peptic ulcer Theophylline may occasionally act as a local irritant to the G I tract although gastrointestinal symptoms are more commonly centrally mediated and associated with serum drug concentrations over 20 mcg/ml.

Information for Patients: The physician should reinforce the importance of taking only the prescribed dose and time interval between doses THED-DUR tablets should not be chewed or crushed When dosing THEO-DUR on a once daily (q24h) basis, tablets should be taken whole and not split As with any controlled-release theophylline product, the patient should alert the physician if symptoms occur repeatedly, especially near the end of the dosing interval

ORUG INTERACTIONS: **Orug-Orug**: Toxic synergism with ephedrine has been documented and may occur with some other sympathomimetic bronchodilators In addition, the following drug interactions have been demonstrated

Drug	Effect
Theophylline with lithium carbonate	Increased excretion of lithium carbonate
Theophylline with propranolol	Antagonism of propranolol effect
Theophylline with cimetidine	Increased theophylline blood levels
Theophylline with troleandomycin, erythromycin	Increased theophylline blood levels

Drug-Food: *THEO-DUR 100 mg Sustained Action Tablets* have not been adequately studied to determine whether their bioavailability is altered when given with food Available data suggest that drug administration at the time of food ingestion may influence the absorption characteristics of theophylline controlled-release products resulting in serum values different from those found after administration in the fasting state

A drug-food effect, if any, would likely have its greatest clinical significance when high theophylline serum levels are being maintained and/or when large single doses (greater than 13 mg/kg or 900 mg) of a controlled-release theophylline product are given

THEO-DUR (200, 300, and 450 mg) Sustained Action Tablets The rate and extent of absorption of theophylline from THEO-DUR 200 mg, 300 mg, and 450 mg tablets when administered fasting or immediately after a moderately high fat content breakfast is similar

Orug-Laboratory Test Interactions: When plasma levels of theophylline are measured by spectrophotometric methods, coffee, tea, cola beverages, chocolate, and acetaminophen contribute falsely high values

Carcinogenesis, Mutagenesis, and Impairment of Fertility: Long-term animal studies have not been performed to evaluate the carcinogenic potential, mutagenic potential, or the effect on fertility of xanthine compounds.

Pregnancy: Category C—Animal reproduction studies have not been conducted with theophylline It is not known whether theophylline can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Xanthines should be given to a pregnant woman only if clearly needed.

Nursing Mothers: It has been reported that theophylline distributes readily into breast milk and may cause adverse effects in the infant Caution must be used if prescribing xanthine to a mother who is nursing, taking into account the risk-benefit of this therapy

Pediatric Use: Safety and effectiveness of THEO-DUR administered
1 Every 24 hours in children under 12 years of age, have not been established
2 Every 12 hours in children under 6 years of age, have not been established

ADVERSE REACTIONS: The most consistent adverse reactions are usually due to overdose and are
1 *Gastrointestinal* nausea, vomiting, epigastric pain, hematemesis, diarrhea
2 *Central nervous system* headaches, irritability, restlessness, insomnia, reflex hyperexcitability, muscle twitching, clonic and tonic generalized convulsions
3 *Cardiovascular* palpitation tachycardia, extrasystoles, flushing, hypotension, circulatory failure, ventricular arrhythmias
4 *Respiratory* tachypnea
5 *Renal* albuminuria, increased excretion of renal tubular and red blood cells, potentiation of diuresis
6 *Other* rash hyperglycemia and inappropriate ADH syndrome.

OVEROOSAGE: **Management**: If potential oral overdose is established and seizure has not occurred
A Induce vomiting
B Administer a cathartic (this is particularly important if sustained-release preparations have been taken)
C Administer activated charcoal
If patient is having a seizure
A Establish an airway
B Administer oxygen
C Treat the seizure with intravenous diazepam, 0 1 to 0 3 mg/kg up to 10 mg
D Monitor vital signs, maintain blood pressure and provide adequate hydration

Post Seizure Coma:
A Maintain airway and oxygenation
B If a result of oral medication follow above recommendations to prevent absorption of the drug, but intubation and lavage will have to be performed instead of inducing emesis, and the cathartic and charcoal will need to be introduced via a large bore gastric lavage tube
C Continue to provide full supportive care and adequate hydration while waiting for drug to be metabolized In general the drug [illegible] metabolized sufficiently rapid so as not to warrant consideration of dialysis, however, if serum levels exceed 50 mcg/ml charcoal hemoperfusion may be indicated

CAUTION Federal law prohibits dispensing without prescription For full prescribing information, see package insert

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(*continued from p 6A*)

MANHATTAN

Nov 5. **16th Annual Newer Strategies Series Symposium: Optimizing Outcome in Schizophrenia.** 6 Cat I Credits. Roosevelt Hotel. Contact: Lester E. Shapiro, MD, 43 Andover Rd, Rockville Centre, NY 11570. Tel: (516) 766-3442.

Nov 5. **Inner City Medicine: Challenges for the Practicing Physician.** 7 Cat I Credits. College of Physicians and Surgeons of Columbia University. Contact: Center for Continuing Education, 630 W 168th St, New York, NY 10032. Tel: (212) 305-3682.

Nov 5–6. **26th Annual Bernard H. Eliasberg Symposium. Practical Perioperative Pharmacology.** 11 Cat 1 Credits. Mt Sinai Medical Center. Contact: Joan D. Esbri, Department of Anesthesiology, Box 1010, Mt Sinai Medical Center, One Gustave L. Levy Pl, New York, NY 10029. Tel: (212) 241-6426.

Nov 10–12. **1988 Glover-Lisman Professorship.** Contact: Martha Klapp, Manhattan Eye, Ear & Throat Hospital, 210 E 64th St, New York, NY 10021. Tel: (212) 605-3762.

Nov 11. **New Dimensions in the Management of Facial, Head and Neck Pain.** New York City. Contact: Barry C. Cooper, DDS, Conference Coordinator, New York Eye and Ear Infirmary, 310 E 14th St, New York, NY 10003. Tel: (212) 598-1600.

Nov 12. **The American Thyroid Association Workshop: The Practical Diagnosis and Management of Thyroid Disease in 1988.** 6¾ Cat 1 Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Nov 13. **Insulin Requiring Diabetes 1988.** 4 Cat I Credits. Mt Sinai Medical School. Contact: Helen Levine, Program Director, American Diabetes Association/New York Downstate Affiliate, 505 Eighth Ave, New York, NY 10018. Tel: (212) 947-9707.

Nov 18. **Fourth Annual Symposium on Techniques in Vascular Surgery.** 10 Cat I Credits. Waldorf Astoria Hotel. *Nov 18–19.* **Echo and Cardiac Surgery**

(*continued on p 18A*)

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Indication: Lower respiratory infections, including pneumonia, caused by *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Streptococcus pyogenes* (group A β-hemolytic streptococci).
Contraindication: Known allergy to cephalosporins.
Warnings: CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] and toxic epidermal necrolysis or the above skin manifestations accompanied by arthritis/arthralgia, and frequently, fever), 1.5%; usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%; and, rarely, thrombocytopenia.

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis, elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly). [061088L]

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COMMENTARIES

Crohn ileitis and massive rectal bleeding

Rectal bleeding is fairly common in Crohn disease and occurs more often in colitis than in enteritis.[1] This may simply be due to the fact that there is a much larger area of ulceration in severe colitis than occurs in severe ileitis. Nevertheless, massive hemorrhage in Crohn disease is not common and is an unexpected complication. The incidence of massive bleeding in ileitis is between 1.5% and 12%, with the higher incidences seen in more recent series.[2-4]

In young adults, Crohn disease may be one of the more important causes of massive rectal bleeding. Dent et al[5] reported three cases of massive bleeding in young adults with Crohn enteritis. Mesleh and Lemons[6] reported a case of massive lower gastrointestinal (GI) bleeding in a 26-year-old man. This was the initial presentation of Crohn disease, with a large ulcer in the ileum. Mesleh and Lemons stated that Crohn disease is probably the third most common cause of massive lower GI bleeding in young adults after congenital vascular lesions and Meckel diverticulum. Certainly Meckel diverticulum is a common cause of bleeding in young people but congenital vascular lesions are not very common. Thus Crohn disease should certainly be considered in any young adult who presents with massive GI bleeding and no previous history of a bleeding lesion. In a review of 503 patients with Crohn disease treated at Cornell University Medical Center over a 43-year period, 31% had clinical bleeding but only 1.4% had massive bleeding.[2] Of these seven patients six had known symptoms and one presented with Crohn disease as a first symptom. Yet this series was put together in a retrospective manner and included a time when knowledge about Crohn disease was not well elaborated. A more current review of 60 patients in the late 1970s showed that 13% of patients with Crohn ileitis developed massive bleeding.[3]

The pathology of Crohn disease shows varying degrees of ulceration with hyperemia and dilation of the submucosal vessels. These vascular changes are more evident in the early stages of the disease, while later the disease is characterized by mucosal and submucosal thickening and fibrosis.[2] Thus, the incidence of severe ulceration with bleeding may be higher in patients with earlier stages of the disease. This, in fact, was recently demonstrated at our hospital in a 76-year-old man who had had Crohn ileitis for 20 years but presented with rectal bleeding for the first time. Colonoscopy showed severe ulceration of the cecum for the first time. The patient underwent surgery and the pathologic examination showed chronic ileitis with thickened mucosa and submucosa but not many ulcerations of the ileum. The cecum showed severe ischemic colitis, and the bleeding occurred from ischemic changes and not from Crohn ulcers.

Angiography has been performed on several patients with bleeding from Crohn ileitis.[4,6-8] Superior mesenteric arteriography shows tortuosity and irregularity of vessels that supply the terminal ileum and cecum. Characteristic findings of Crohn disease include diffuse, irregular hypervascularity throughout the bowel wall and mesentery. The "zoning sign" describes a double layer appearance of the bowel wall with a densely stained inner wall reflecting a hypervascular submucosa and a relatively avascular muscular layer reflecting a thickened hyperplastic outer layer. As is often seen in other inflammatory conditions, the venous phase demonstrates early filling of veins as well as dilation and tortuosity of the veins from affected segments.

In the case presented by Smith-Behn et al[9] in this issue of the *Journal*, the bleeding site in the mid-ileum was documented by mesenteric arteriography. This has been helpful in several patients with massive bleeding due to Crohn ileitis. Nevertheless, in the current evaluation of massive rectal bleeding, it is my view that mesenteric arteriography should not play an early or primary role. The first step should include colonoscopy, which is now known to be helpful even in patients with acute rectal bleeding.[10] At the same time the patient can have an emergency gastroscopy. If both gastroscopy and colonoscopy are normal then the small bowel is likely to be the bleeding source. This procedure avoids the time and possible morbidity associated with arteriography. The surgeon will be able to find the diseased bowel segment in most cases without arteriography. In addition, further help may be obtained by the noninvasive isotope red cell scan. Certainly centers that have aggressive angiographers will contest this sentiment but I have found this approach efficacious in a large number of patients with rectal bleeding.

The treatment of massive bleeding from Crohn disease consists of surgical extirpation. As in the case of the bleeding peptic ulcer one cannot expect the medical treatment of Crohn disease to diminish the propensity to bleed during the immediate short term. The medical treatment for inflammatory bowel disease does not influence the likelihood that a clotted vessel or a constricted vessel will bleed or rebleed. The healing period with such treatment is much too slow. In our series of eight patients seven needed surgery to stop bleeding.[3] There are patients who have undergone perfusion of vasoconstrictors through arteriographic catheters, but this has not usually been sufficient.[6,8] In a representative case reported by Renison et al,[11] the injection of vasopressin was initially successful; however, bleeding recurred and the patient needed surgery. Thus, the decision to use vasopressin in these patients would be similar to that in persons with other bleeding lesions. In controlled studies of other bleeding lesions this mode of treatment has never been shown to be effective.

In the patient described by Smith-Behn in this issue of the *Journal*, the mid-ileum was resected and the patient did well, although he needed medical treatment for recurrent disease. This report describes an uncommon presentation for Crohn ileitis. Most often the terminal ileum will be diseased and the typical resection will usually involve the ileum and cecum. The current philosophy of surgery for Crohn disease is to remove only the most diseased area and not to obtain a frozen section of normal tissue at the anastomosis. Thus the surgical resection for bleeding should remove the grossly ulcerated areas and not all microscopically involved ileum.

Crohn disease may present with massive rectal bleeding as the initial manifestation of ileitis. In our series of eight patients massive bleeding was the initial presentation in seven,[3] as it was in all three of the patients of Dent et al.[5] Most often patients who have Crohn colitis will not present with massive bleeding as the initial manifestation. They will have diarrhea and abdominal cramps more often. If the bleeding stabilizes, the evaluation should include colonoscopy, with an attempt at examining the ileum and also a small bowel series. Enteroclysis is a type of small bowel series which may better demonstrate the changes in the ileum but is not routinely necessary.

Crohn disease is a disease on the ascendancy and one can expect to see more patients with massive gastrointestinal bleeding secondary to this problem.[12] The proper organized evaluation of patients presenting with rectal bleeding will facilitate both the diagnosis and treatment. The fact that patients have Crohn disease will not change the success of a standardized approach to massive rectal bleeding.

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The empty lifeboat

In his classic account of the last days of World War II, John Toland[1] describes how thousands of German refugees from Danzig were attempting to get aboard a ship, the *Goya*, that they hoped would take them safely away from the approaching Russian army. During this chaotic situation Werner Juttner, one of the German officers who was on the rapidly overcrowding ship, witnessed a son roughly preventing his aged parents from climbing onto the deck. The son was heard to cry out: "You're of no use any more! You're too old!"

Juttner, to his great horror, was soon to find himself behaving in a similarly callous fashion. A day after leaving port the *Goya* was sunk by Russian torpedoes and he and thousands of others found themselves struggling in the sea. He was able to get to a nearby life raft, but as additional people climbed on the raft it, too, began to sink. Juttner and those already on the raft, to save themselves from drowning, began to beat the newcomers off until no one was left to approach the raft. Of the 7,000 people on the *Goya* when it went down, only 170 survived.

"Lifeboat ethics" is almost a formal subclass of ethical study: the examination of the moral dilemmas that exist when space, food, equipment, personnel, or other life-saving resources are in scarce supply. Today health professionals are being inundated with arguments that they need to make lifeboat-type decisions and to draw similar death-affirming conclusions. Among these arguments are those for the withholding of food and water from certain terminally ill or handicapped patients, for the reduction of the public resources being spent on elderly patients during their last year of life, and for the killing of some patients by overdose for the patient's own good and for the good of the patient's family.

Unfortunately, the people raising these euthanasia arguments have not looked around. If they had, they would have noticed that the American health care lifeboat is not full, it is in fact virtually empty.

The vast resources of the United States are far from the breaking point: Our standard of living is near an all-time high; our medical schools are graduating huge numbers of physicians; our health facilities have thousands of empty beds; and the provision of food, water, and effective painkillers can be readily accomplished in a variety of patient care settings.

The only scarcities we have are apparently those of human compassion and a willingness to meet, at some discomfort to ourselves, the holistic needs of all patients, including the elderly, the handicapped, and the terminally ill.

For example, even if we could somehow discern with complete accuracy the patients who were "in their last year of life," we ought to be looking for ways to use that information to reach out to them in a positive way, not a negative one. By our words, by our individual actions as health professionals, and by our national health policies we ought to make it clear that we are willing to meet their special spiritual, social, mental, and biophysical requirements. We should make it obvious that even though they may not be able to produce a cost-benefit analysis to justify their continued existence, they are nevertheless respected members of the human community.

The needs of the elderly, the handicapped, and the terminally ill should never be considered so intense or expensive that we feel we can justify the withholding of either our compassion or our effort, let alone the killing of human beings in cold blood. As health professionals we must strenuously refuse the temptation to embrace euthanasia as a cheap proxy for our efforts to reduce human suffering, and we must also be willing to refute loudly the argument that killing is a form of healing. Most importantly of all, we must make a personal vow to provide each patient with humane, life-affirming health care services that are untainted by economic or diagnostic prejudice.

There is still plenty of room in the lifeboat.

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Issues related to the medical certification of death

A physician survey

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ABSTRACT. We surveyed Vermont physicians to identify problems associated with medical certification and the pronouncement of death. The response rate was 67%. Thirty-five percent of responding physicians reported having difficulties filling out death certificates for their own patients. Major problems cited included the format of the certification section, identifying the appropriate causal sequence, and completing the certificate within the time period prescribed by law.

Seventy-four percent of the respondents who are not medical examiners completed certificates for patients of other physicians. These certifications were problematic for 64% of respondents, the major issue being the availability of the patient's medical history. Fifty-six percent of respondents stated that someone other than a physician should be allowed to pronounce death in cases in which death is expected. Most favored extending pronouncement rights to physician assistants and registered nurses. These survey results indicate the need for efforts to improve medical certification, primarily in the area of physician training.

(NY State J Med 1988; 88:522–525)

Death certificates serve two purposes: They are legal documents important to the decedent's family, and a source of statistical data from which information about the health status of populations is developed. The utility of mortality statistics for public health surveillance is a function of the completeness of death registration and the accuracy of the data reported on death certificates. In the United States, death registration is virtually complete. The number of unreported deaths is small, the only significant underreporting being geographically sporadic and related to fetal and early infant deaths.[1]

The accuracy of information provided on death certificates has been extensively studied.[2] In general, the accuracy of such items as the decedent's date of death, age, sex, and marital status is high.[1,3] However, numerous studies have demonstrated a lack of agreement between information about the cause of death recorded on death certificates and data in hospital records or autopsy reports.[4–6] In their assessment of numerous cause-of-death studies, Gittelsohn and Royston[2] reported that the quality of this data appears to vary with the specific cause under consideration and the characteristics of the decedent. They also state that "the most striking finding is that so little is known about the quality of medical certification and its effect on diagnostic statistics in general and on national cause-of-death statistics in particular." Despite these findings, cause-specific mortality statistics continue to be a fundamental component of epidemiologic and other studies. Most users of these data tend to assume that the statistics are accurate.[7]

The responsibility for completing the medical certification of death rests with the physician. Although state laws vary, most states require that the physician last in attendance of the deceased pronounce death and complete the medical certification statement (cause-of-death section of the death certificate) within a prescribed time period.[8] Thus, the physician's ability to correctly certify the cause and manner of death is a major determinant of the accuracy and completeness of mortality statistics. As an initial step in a program to improve the quality of mortality data, in 1985 the Vermont Department of Health surveyed physicians about their concerns regarding death certification and pronouncement.

METHODS

In April 1985, surveys were sent to all physicians with permanent Vermont licenses who practiced in Vermont and who had signed at least one death certificate in 1984. Physicians with temporary licenses (primarily first-year hospital residents) were not surveyed. Questionnaire items addressed the pronouncement of death, the appropriate length of time between death and completion of the certificate, and the physician's perception of his or her ability to complete the medical certification. Because of the sensitivity of some of the questions, individual physicians were not asked to identify themselves. This precluded follow-up with nonrespondents. Analysis consisted of simple frequencies and cross tabulations.

Vermont has a statewide medical examiner system consisting of two full-time forensic pathologists and a cadre of fee for service, volunteer physician medical examiners. Medical examiner responses were differentiated from the responses of other physicians.

RESULTS

In 1984, 4,637 deaths occurred in Vermont. These deaths were

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certified by 577 physicians, 405 of whom met the criteria for inclusion in the survey. The 405 physicians certified 3,642 (79%) deaths during 1984. The distribution of the surveyed physicians by type of certifier is presented in Table I. More than two thirds of deaths were certified by the attending physician; 15% by medical examiners. The number of certificates signed by an individual physician ranged from 1 to 118. Two thirds (66%) of physicians signed fewer than ten certificates in a year. At the other end of the spectrum, more than half (51%) of all deaths were certified by 60 physicians.

A total of 272 responses were received, an overall response rate of 67%. Eighty percent of the medical examiners surveyed responded, as did 64% of other physicians. In addition to answering the specific questions, many physicians commented about their concerns and frustrations with various aspects of the death certification process. The extent of these comments and the level of physician interest was surprising. These comments are summarized briefly where appropriate.

Issues Related to Medical Certification. Ninety-six physicians (35%), including 20 medical examiners, indicated that they found it difficult to complete medical certifications for their own patients. Twenty percent of these physicians commented that it was difficult to be precise or to know what information was wanted, with several indicating the need for autopsy findings as a prerequisite to certification. (Comments by 16% indicated that there was not enough space in the medical certification section of the certificate.)

A number of themes dominated physicians' comments related to medical certification. The first was the format of the certification section. Eleven percent of those who had had difficulty completing medical certification found the format confusing or, in the words of one physician, "not suitable for contemporary diagnoses." Some physicians indicated that they would prefer to simply list all conditions present at death rather than to attempt to determine a causal relationship. There was also a group that would prefer to provide no diagnosis, but simply to indicate that the patient died of "natural causes," or that the cause of death was "unknown."

There was some confusion about the use of symptoms as the immediate cause of death. Several physicians indicated that they almost always list cardiopulmonary arrest (or a variation of this) as the immediate cause, although they felt that this was "misleading," "not truly the cause of death," etc. A related issue was an apparent lack of understanding about the relationship between the immediate and the underlying causes of death. For example, one physician commented that the "etiology [is] frequently unclear—cardio/respiratory/brain arrest [is] often the final common denominator, but not truly the cause of death."

Finally, a group of physicians expressed concern about how to certify the death of an elderly patient with multiple medical problems. Examples of comments received included, ". . . in terminally ill elderly who simply stop living—single or specific cause is rarely clear"; "elderly patients who die of simple old age sometimes require somewhat frivolous diagnoses to cover that"; and "many older patients have multiple contributory problems and the cause of death is not the major problem."

Seventy-four percent of respondents who are not medical examiners occasionally complete death certificates for patients they have not attended. Of these, 64% found it difficult to determine accurately and list all diagnoses relating to the causes of death. The availability of the patient's chart and medical history seems to be an important factor, which was cited in the comments written on 16% of the responses. Several physicians also indicated that it was difficult to determine the causes of death particularly in patients from nursing homes whom they did not attend.

TABLE I. Type of Certifier and Number of Certificates Completed by Surveyed Physicians, Vermont, 1984

Type of Certifier	Physicians* No.	Certificates No.	Certificates %
Medical examiner	79	552	15.2
Attending physician	314	2,502	68.7
Staff physician	159	481	13.2
Pathologist	19	107	2.0
Total	405	3,642	100.0

* Does not sum to total because some physicians signed as more than one type of certifier.

TABLE II. Physicians' Estimates of Reasonable Length of Time to Complete the Medical Certification

Hours	Physicians No.	Physicians %
24	112	44.3
48	87	34.4
72	43	17.0
>72	11	4.3
No response	19	—
Total	272	100.0

Issues Related to the Time Between Death and Certification. Vermont law requires that the physician complete the medical certification within 24 hours of death. Almost a quarter (24%) of respondents found this requirement difficult. There was no difference between the responses of medical examiners and other physicians on this issue. The primary concern of those commenting was the logistics of certifying weekend deaths. Another concern related to the availability of autopsy findings within 24 hours. Table II presents the amount of time physicians think would be reasonable for completing the medical certification. More than half (56%) would like to have at least 48 hours to complete the certificate.

Issues Related to Death Pronouncement. Table III presents physicians' views on who should be allowed to pronounce death. Fifty-six percent of respondents said that someone other than a physician should be allowed to pronounce death, while 42% responded that this responsibility should be limited to physicians. Five percent of respondents felt that official pronouncement of death was unnecessary.

Medical examiners were more likely to favor limiting pronouncement to physicians than were other physicians. Fifty-four percent of medical examiners and 39% of other physicians stated that only physicians should pronounce death.

Physicians' comments in favor of allowing others to pronounce death reflected both a level of confidence in the abilities of medically trained personnel to do so and the realities of current practice. For example, the physicians wrote, "I've never been called by a nurse to pronounce and found that the patient was still alive," and "This is effectively what happens at night in the nursing home or hospital."

Physicians who would not delegate the responsibility for pronouncement were concerned with accuracy and the avoidance of legal entanglements. For some, it was also a question of duty. As one respondent wrote, "Death requires a respect to the condition and the deceased requiring the presence of a physician."

Most respondents (81%) said that whoever pronounces death should view the body before doing so. These physicians felt that viewing the body helps to verify the facts. The physician should "identify the patient [and] attempt to avoid overlooking an unexpected cause or contributing factor." Of the respondents who said that pronouncers do not need to view the body, 47% limited their responses to those situations in which death is expected and/or observed by other qualified individuals.

DISCUSSION

The medical section of the death certificate contains two parts. Part I is used to list the conditions that led directly to death, while Part II requests other conditions that contributed to death but were not related to the immediate cause given in Part I. The conditions in Part I are listed sequentially beginning with the most recent, or "immediate" condition, and proceeding in reverse temporal order. The most important condition, listed on the last line

TABLE III. Physicians' Opinions of Who Should Be Permitted to Pronounce Death by Type of Respondent (Medical Examiner vs Others)

	Respondent					
	Medical Examiners		Other Physicians		Total	
Type of Provider	No.	%	No.	%	No.	%
MD/DO	63	100.0	204	97.6	267	98.2
MD/DO only	34	54.0	81	38.8	115	42.2
MD/DO and others	29	46.0	123	58.9	152	55.6
Registered nurse	23	36.5	110	52.6	133	48.9
Physician's assistant	23	36.5	109	52.2	132	48.5
Licensed practical nurse	9	14.3	40	19.1	49	18.0
Emergency medical technician	8	12.7	41	19.6	49	18.0
Funeral director	2	3.2	4	1.9	6	2.2
Law enforcement official	2	3.2	4	1.9	6	2.2
Offical pronouncement unnecessary	2	3.2	12	5.7	14	5.1

of Part I, is the "underlying" cause of death, defined as "the disease or injury which initiated the train of events leading directly to death." [9]

Statistically, the importance of the underlying cause cannot be overstated. Virtually all mortality analyses that utilize a single cause use the underlying cause. All national statistics are tabulated on this item. Consequently, the physician's role in accurately completing the medical certification and correctly identifying the underlying cause is crucial to the development of accurate cause-specific mortality statistics. Unfortunately, most physicians have not been trained in what information is wanted on the death certificate and how to record it.[10] The physician responses presented here indicate that one result of this lack of training is uncertainty about the correct way to fill out the certificate. Topics such as causal sequencing, appropriate diagnostic terms, and the acceptability of "natural causes" as a certification statement are examples of these uncertainties.

Various groups have suggested ways to improve the quality of data reported on the death certificate. The Panel to Evaluate the US Standard (Vital) Certificates and Reports made several specific recommendations related to physician training, such as the need for a multifaceted approach to education that would include medical school courses, hospital training as a prerequisite for admitting privileges, state licensing requirements, and questions on National Board Examinations. The panel further recommended that the US Department of Health and Human Services bring together a group to establish training requirements and to coordinate the development of training materials.[11] The National Autopsy Data Bank Committee of the College of American Pathologists recommended that ". . . all physicians should be required to demonstrate the knowledge of proper completion of a death certificate, especially the medical-certification component, on state and national board examinations." [12]

Of genuine concern is the certification of death for a patient of another physician. This practice, performed by 74% of responding physicians, was cited by one physician as "the biggest problem I have with filling out death certificates. . . ." This physician recommended that "the attending physician should be the one to fill out death certificates. . ." This view, in fact, conforms to a recommendation of the Panel to Evaluate the Standard Certificates. That group modified the Standard Certificate of Death to allow a physician to pronounce death and sign the certificate in the absence of the attending physician without completing the medical certification. The medical certification would be completed by the attending physician at a later time.[11]

The availability of sufficient information to complete the medical certification within the 24-hour time period allowed by Vermont law is another area of concern. To quote one physician, "Although it may be desirable to certify the death as soon as possible for the family's sake, a lot of misinformation results in certain instances because of the 24 hour limit." In this respect, Vermont is more stringent than the national standard, which requires that the certificate be completed within 48 hours,[13] a time frame considered reasonable by 79% of responding physicians. In the interests of improved data quality, it may be appropriate for states that require the medical certification to be completed in less than 48 hours to consider revising this requirement. In fact, the National Autopsy Data Bank Committee has taken this consideration one step further and has recommended a two-part certificate. One part would be filled out immediately to expedite disposal and other legal issues; the second part, containing the medical certification, could be deferred pending clinical or autopsy evaluation.[12]

This survey was undertaken to determine the status of physician understanding of medical certification, so that physician training activities could be appropriately directed. As a result of these findings, several activities have been initiated.

We developed a physician manual on cause of death certification, specific to Vermont, in which the types of concerns expressed by physicians in the survey are addressed. This short booklet was first distributed to all physicians who signed a death certificate in 1985. It is now regularly made available to all newly licensed physicians. We also developed several sample "case sheets" which include a one-page description and a blank death certification section. A discussion of the "correct" certification is given on the back of the sheet. We introduced these materials to physicians through the Health Department's exhibit booth at the annual meeting of the Vermont State

Medical Society. Following the medical society meeting, the exhibit was on display for a month in the library of the University of Vermont College of Medicine.

A program has been launched to introduce physicians to the concepts of cause of death certification through a series of hospital staff meetings. The vital statistics staff will share responsibility for presentations at these sessions with an interested staff physician from each hospital who will provide accurate, scientific responses to medical questions. In addition, a letter has been prepared for physicians who certify deaths detailing the uses of cause of death data, providing some basic statistics, and reminding physicians of the importance of their role in the death registration system. Finally, the relevant Vermont statutes are being reviewed to determine whether they are appropriate to the production of timely, accurate statistics. It may be that the time period for medical certification could be revised.

While these activities are specific to Vermont, they can be generalized to other states. The responsibility for accurate death certification must be shared by the physician and the state vital statistics office. In addition to the activities planned in Vermont, vital statistics offices could consider implementing active query programs to address issues such as inadequate diagnoses and inappropriate sequences. These programs are an excellent opportunity to provide postgradute education.[10] Physician education through medical school courses is another approach that may be beneficial.

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Inhalation marijuana as an antiemetic for cancer chemotherapy

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ABSTRACT. A prospective pilot study of the use of inhalation marijuana as an antiemetic for cancer chemotherapy was conducted. Fifty-six patients who had no improvement with standard antiemetic agents were treated and 78% demonstrated a positive response to marijuana. Younger age and prior marijuana exposure were factors that predicted response to treatment. Toxicity was mild and consisted primarily of sedation and xerostomia. This preliminary trial suggests the usefulness of inhalation marijuana as an antiemetic agent. Because of the lack of a randomized placebo control group, the precise role of this agent is unclear. Further studies should include derivatives of this substance in combination with standard effective drugs to control chemotherapy-induced nausea and vomiting.

(NY State J Med 1988; 88:525–527)

A great deal of clinical information has recently been generated concerning the efficacy of various antiemetic agents for patients treated with cancer chemotherapy.[1-3] Without effective control of nausea and vomiting, patient compliance with potentially curative chemotherapy programs diminishes, compromising not only quality but quantity of life. Effective new chemotherapeutic agents could never be successfully tested in clinical trials if they possessed potent emetic side-effects.

Although a number of agents have recently been found to be active, including metoclopramide,[4,5] haloperidol,[6] dexamethasone,[7] and lorazepam,[8] the need to introduce newer agents continues. Tolerance often develops to single agents and combination antiemetic therapy may be necessary for continued control of symptoms. Also, complete control of nausea and vomiting during anticancer treat-

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ment must take into account not only the physical effects but also the psychological ones. Control of anxiety through behavior modification and relaxation is an effective antiemetic treatment of anticipatory nausea and vomiting.[9]

Natural and synthetic cannabinoids are known to be effective antiemetic agents.[10-12] Delta-9-tetrahydrocannabinol (THC) has been found to be superior to prochlorperazine.[13] Also, patients who are refractory to standard antiemetic agents have significant reduction in nausea and vomiting with oral THC.[14] There is little information on the efficacy of inhalation marijuana aside from anecdotal reports from patients who obtained the drug privately.

As a part of a New York State Department of Health program, North Shore University Hospital conducted a preliminary study of the use of inhalation marijuana as an antiemetic agent for cancer chemotherapy. The purpose of this study was to evaluate the efficacy of inhalation marijuana for patients refractory to standard agents, to identify patient characteristics to predict response, and to evaluate toxicity and patient acceptance of this form of treatment.

METHODS

Patients with histologically confirmed malignancies who were actively receiving chemotherapy were entered into the protocol. Eligibility criteria included: 18 years of age or older, refractoriness to conventional antiemetic agents, and absence of severe cardiac or psychiatric disease. Patients had to agree not to drive or operate heavy machinery or a motor vehicle for at least 12 hours after the last dose of marijuana. Central nervous system depressants including alcohol were prohibited during the administration of marijuana.

Marijuana cigarettes were supplied by the National Institute on Drug Abuse (NIDA) to the New York State Department of Health. All patients were instructed on standard smoking procedures. The patient inhales deeply, holds the inhalation for ten seconds, and then exhales. After waiting 10 to 15 seconds, the cycle is repeated. The total dose is completed within five minutes. A flame-proof holder was available to permit delivery of nearly all of the cigarette appropriate to the patient's dosage. The dose schedule, which was calculated to the nearest one-fourth cigarette, was 5 mg THC/m^2, starting 6–8 hours prior to chemotherapy and every 4–6 hours thereafter, for a total dose of four doses per day on each day of chemotherapy (one cigarette = 10.8 mg THC). In order to prevent cigarettes from drying out and causing harsh smoke, patients were instructed to keep the cigarettes in the refrigerator or humidified.

This was a nonrandomized study where patients served as their own controls. Patients were asked to self-rate their status by completing a patient evaluation form after each therapeutic episode. Nausea was graded on a scale from 1 (none) to 4 (severe), vomiting was graded from 1 (none) to 5 (10+ times), appetite was graded from 1 (none) to 5 (above normal), physical state was graded from 1 (very weak) to 4 (above normal), and mood was graded from 1 (very depressed) to 5 (very happy). Based on the degree of nausea, vomiting, food intake, physical state, and overall mood, patients rated the overall effectiveness of marijuana as none, moderately effective, and very effective.

Physician investigators were approved by the hospital's Patient Qualification Review Board. Physicians utilized the official New York State triplicate prescription form as their research order for medication. Informed consent was obtained from all patients and the procedures followed were approved by an institutional research committee.

RESULTS

Seventy-four patients entered the study and 56 were evaluable. Eighteen patients who had initially agreed to be treated with marijuana later decided not to participate. Eighteen patients rated the marijuana very effective (34%) and 26 patients rated it moderately effective (44%), for an overall response rate of 78% (44/56). Twelve patients (22%) noted no benefit.

TABLE I. Patient Characteristics (Percent)

	Responders (N=44)	Nonresponders (N=12)	*P* Value
Female	64	75	NS*
Mean age (yr)	41†	51‡	
(median)	(40)	(54)	
Breast cancer	36	33	NS
Lymphoma	34	25	NS
Prior radiation therapy	30	8	NS
Prior THC	29	20	NS
Prior marijuana	52	17	0.06
Euphoria (high)	60	36	NS
Smoker	53	38	NS

* NS = not significant.
† Standard deviation = 11.9.
‡ Standard deviation = 15.6.

Characteristics of responding and nonresponding patients are listed in Table I. While no statistically significant differences were noted between responders and nonresponders with regard to sex, type of diagnosis, prior radiation therapy, prior oral THC treatment, incidence of euphoria, or smoking history, it is important to remember that the sample sizes were small, making interpretation of differences difficult. Patients who responded to marijuana cigarettes were more likely to be younger, median age 40 vs 54 for nonresponders, and had prior marijuana exposure, 52% vs 17% (p = 0.06).

The most common diagnoses for this group of patients were breast cancer, lymphoma, lung cancer, colon cancer, ovarian cancer, testicular cancer, sarcoma, acute leukemia, and myeloma. The most common emetic chemotherapeutic agents were cyclophosphamide, doxorubicin, cis-platinum, procarbazine, methotrexate, dacarbazine, and streptozocin, given either singly or in combination. Four of seven patients treated with cis-platinum responded favorably to marijuana cigarettes.

Toxic side effects included sedation in 88%, dry mouth in 77%, dizziness in 39%, and confusion in 13%. Anxiety, headache, and fantasizing were also seen but were less common. There was no toxicity in 13% of patients (Table II).

DISCUSSION

The results of this prospective study suggest that inhalation marijuana is active in controlling nausea and vomiting resulting from chemotherapy. Marijuana benefited patients who were treated with a wide range of chemotherapeutic agents including drugs which have considerable emetogenic potential. A prior report by Chang et al[15] documented effectiveness of oral THC and inhaled marijuana against high-dose methotrexate, which normally has mild gastrointestinal toxicity. While most experience

TABLE II. Percent Toxicity

Sedation	88
Dry mouth	77
Dizziness	39
Confusion	13
Anxiety	11
Headache	11
Fantasizing	11
None	13

indicates that THC is generally ineffective against cisplatinum-induced emesis, benefit was seen in a small number of patients treated in our program with this agent.

Since this was a single arm, nonrandomized, outpatient program, this study lacks a controlled placebo group. Nevertheless, the patients acted as their own controls, having previously failed standard antinausea medications. They evaluated marijuana based on their subjective rating of the severity of nausea, vomiting, appetite and food intake, mood, and physical state after chemotherapy treatment. A placebo-controlled, randomized inpatient study which quantitates all emetic episodes would obviously provide objective and precise information.[16]

Failure to respond to oral THC does not preclude benefit from inhaled marijuana. Twenty-nine percent of patients who failed oral THC responded to the cigarette form. This is not unexpected, since only 5–10% of orally administered THC is absorbed, whereas inhaled marijuana has a five- to tenfold greater bioavailability.[17] Clearly, oral THC is an effective treatment for chemotherapy-induced emesis. Most studies have demonstrated THC to be better than placebo and comparable to prochlorperazine.[18]

The major obstacle related to the oral and inhaled cannabinoids is the route of administration. Patients with anticipatory vomiting do not retain the oral THC. Because of its poor water solubility, parenteral administration of cannabinoids has been difficult. The only cannabinoid available for parenteral use, levonantradol, is currently being investigated and has documented activity comparable to THC.[19] Perhaps intranasal or transdermal forms of THC will be developed and found to be clinically useful.

Patient characteristics were evaluated to identify factors which would predict response to marijuana. There were no significant differences between responders and nonresponders with regard to sex, diagnosis, prior radiation therapy, prior THC ingestion, induced euphoria, and history of cigarette smoking. The only factors that approached significance were young age and prior marijuana intake. Unlike the experience with oral THC, experiencing a euphoric high was not a prerequisite to obtaining the antiemetic effect with marijuana.[20]

The mechanism of the antiemetic action of cannabinoids is unknown. Inhibition of prostaglandin and cyclic adenosine monophosphate has been suggested. Its major action is more likely related to its effect on the brain, as marijuana causes central nervous system depression and impairment of brain function. At the cellular level, cannabinoids interfere with the synthesis of nucleic acids and chromosome proteins.[21]

Some of the problems encountered in this study which could influence interpretation of the results were the low patient accrual and the fact that nearly 25% of patients who initially consented refused to receive treatment. Reasons for patients' refusal to participate included physician and patient bias against smoking, harshness of smoke from the cigarettes, and preference for oral THC capsules. The major objection was related to the social stigma attached to the use of marijuana. Many patients rejected the idea of "smoking pot" at home and exposing their children to the implications of this type of medication. Should this therapy become available in a different vehicle of administration, patient acceptance would significantly improve.

Our results demonstrate that inhalation marijuana is an effective therapy for the treatment of nausea and vomiting due to cancer chemotherapy. A randomized, controlled trial would, however, be necessary to accurately define the exact role of this drug. Toxic effects are well tolerated and the availability of a parenteral form would improve patient utilization of this agent. Future antiemetic protocols should include the active ingredient of marijuana in combination with current effective agents.

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Adult day care—a recent past and a growing future

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The adult day care concept is only a generation old. It was started in the Soviet Union in 1933, appeared in post-war Great Britain in 1943, and finally arrived in the United States in the early 1960s. Yet the adult day care center has been one of the fastest growing programs to assist older Americans who reside in the community.[1]

HISTORICAL PERSPECTIVES

In 1933, Dzhagarov, the chief physician at the Moscow Psychiatric Hospital, was faced with a serious shortage of beds. Increasing the size of the institution, deemed a capitalist solution, was not acceptable. The solution was to maximize the use of the existing space by creating a new program, the day hospital. The plan called for patients to be treated in the hospital during the day in existing ward treatment areas and then to return to their homes at night.[2]

A decade later, two pioneers, Cameron[2] in Montreal and Bierer[3] in London, developed adult day care programs to provide soldiers returning from the second World War battlefields with medical care and psychosocial rehabilitation. Lack of available psychiatrists and nursing staff made timely delivery of needed health services difficult. By assembling patients into therapeutic groups, scarce health resources were maximized.

This early model was well received and was expanded in Great Britain in the 1950s to a medically oriented day program designed to meet the special needs of the physically disabled and the elderly. It became an integral part of the national health system, linking hospital and community services.[4] The first day hospital officially opened in 1958 in Oxford. By 1976, more than 70 day hospitals were in existence in the United Kingdom. The concept was being introduced in many countries throughout the world, including South Africa and Canada.[5-11] The objectives of all programs were to improve utilization of hospital beds, with earlier discharge to the community and prevention or delay of institutionalization. Political and social pressures of the 1960s prepared the way for this new step. With these traditions and hopes the adult day care concept was ready to come to the United States.

ADULT DAY CARE IN THE UNITED STATES

In 1965, the Older Americans Act encouraged the development of programs to provide socialization and recreational services for the elderly.[12] Subsequently, a small number of geriatric day care centers that provided recreational and nutritional services began to appear throughout the nation. Meanwhile, the newly launched programs of Medicare and Medicaid spurred the growth and expansion of hospitals and home care.[13] As a result of the rapid increase in costs, the US government began to promote "efficient care," and not "equitable care," which had been fashionable in the 1960s.[14] Looking for ways to reduce health care costs and to provide new forms of health care delivery, Congress, in 1972, passed Public Law 92-603.[15] This law established the Professional Sources Review Organization (PSRO), a peer review organization to monitor utilization of hospital services. In addition, the law authorized the first demonstration adult day care programs supported by Medicare and Medicaid.

At first, these demonstration day care programs seemed an easy way to provide a new health delivery system, one that had already proved itself in Europe. Unfortunately, the imposed regulations were restrictive and provided little financial incentive. The centers were to provide a comprehensive array of services, including transportation and home care, at no cost to the participant. Furthermore, close monitoring of the centers' cost effectiveness was to start immediately through a research study sponsored by the Department of Health, Education, and Welfare (DHEW).[16]

The day care concept barely survived its first years in the United States. Initiated during difficult political and economic times, the programs had to prove their cost effectiveness during the start-up phase. The first Weissert[17] report (a descriptive analysis of the demonstration day care centers) identified three common objectives to all programs: cost saving, improved outcomes, and improved quality of life. However, such objectives are often inappropriate for the frail elderly who are obviously at great risk of being institutionalized and whose quality of life is often poor and refractory to any improvement. Analyzing both the medical and the social model of adult day care, Weissert concluded, "Ideally a long term care system would provide care at the right level, at the right time, in the right setting, at the most quality achievable with the state of the art and would do it most cost effectively."[18,19]

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The Weissert report's conclusion that adult day care was not cost-effective condemned adult day care in the eyes of the federal government. But while the demonstration projects were still in progress, several state agencies and private organizations became interested in the concept and started sponsoring their own programs. Thus, by 1975, Medicare had totally abandoned the idea of adult day care, but Medicaid had adopted it at the state level.

The first directory of adult day care programs, published in 1977, listed about 200 programs nationwide.[20] A remarkable growth in the number of programs to more than 600 programs was seen by 1980.[21] By 1986, the National Institute of Adult Day Care, a constituent unit of the National Council on Aging, surveyed 1,200 adult day care centers throughout the nation.[22] The annual budget of these programs is estimated to be $150 million dollars. They provide care for some 28 million Americans.[23]

It is important to note that this growth occurred at a time when new federal initiatives were sparse, and human services were being reduced. This movement has been a grassroots response to local communities' needs and resources. It has resulted in a rich tapestry of programs, unique in the flexibility of their designs.[24] It would seem that these programs respond to the needs of frail older Americans and their families.

For many people, the impact of day care can be dramatic. For example, here is the testimony of a woman who survived cancer and who also suffers from Parkinson disease:

> I couldn't even blow my nose when I came here. Now I can write again. I can crochet again. I couldn't do it for two years. It [the center] does you more good than any hospital. You move around and you do things....It's a whole new life opening up for me here.

FUTURE OF ADULT DAY CARE IN THE US

As we look forward to the 1990s, the need for an increased understanding and support of these day programs is vital. Demographers predict that the number of people over 85 years of age (those most likely to need nursing home care), which now totals 2.2 million, will more than double by the year 2000, reaching an estimated 4.9 million.[25]

While people are living longer, their children remain in the work force and are no longer home to provide care. Substitute caregivers are needed. The question then becomes, "Who will care for the old old?" Schmidt[26] discusses the problems that arise from the increasingly frequent living situations of the parent-child pairs, in which children nearing retirement age find themselves caring for parents in their mid-80s. A relatively new term—elder care—is coming into usage to describe the problems of care for an elder relative. Elder care is no longer a concept limited to a few dedicated gerontologists. It is becoming a concern to large corporations who realize its impact on the daily lives of their own employees.

For instance, Traveler's Insurance Company, as a direct result of an extensive employee survey, found that an unexpectedly high percentage of their full-time employees were spending significant amounts of time caring for aging relatives and friends.[27] A new insurance plan was designed to cover the cost of adult day care, recognizing this service as both a cost efficient and viable alternative for the elderly who need long-term care. The coverage is being offered to both active and retired employees of participating companies as well as to the parents of employees.

Another major corporation, Wang Laboratories, Inc, in Lowell, Massachusetts, is also developing a plan to assist its employees who are providing elder care. This company's approach has included a demonstration project to study the feasibility of adult day care for the elderly relatives of employees.[28]

Financial issues remain a key point. Who will pay for day care? Medicaid remains the primary payor, but the coverage varies from state to state. Many elderly earn above Medicaid level, but cannot afford the cost of adult day care. Currently, Medicare does not reimburse costs for day care, although the government is paying for a number of such programs for World War II veterans who are now reaching the age of 65. Representative Leon Panetta of California has put forth a proposal that would amend the Social Security Act, permitting Medicare Part B payment for day care programs that are medically oriented.[29]

At present, adult day care has a bright future. It clearly responds to a growing need in the care of the elderly. It also pools the resources of experienced professionals able to monitor and maximize the quality of life of elderly citizens. This is particularly relevant at a time of great shortages of trained nurses, qualified home attendants, rehabilitation therapists, and geriatricians. With the assistance of gerontological teams in adult day care programs, physicians can effectively treat the complex medical problems of the elderly on an outpatient basis. Families can rely on competent assistance for concrete support and respite. These families are often willing and able to purchase this much needed service.

CONCLUSIONS

Adult day care, it would seem, has achieved wide acceptance. The community and health care system have accepted it, not only as an alternative to institutionalization, but as an independent, free-standing system which delivers care outside hospitals and nursing homes to a group with identified needs. Physicians who are often sought out by the elderly and their families for help and counsel can play a major role in the growth of adult day care. McTernan and Leitzen[30] state that, "...the physician of the future will shift from soloist to conductor, and he/she must recognize the fact that no conductor can perform without a full orchestra." Adult day care can be an important section of that orchestra. This new and powerful resource, which can coordinate the multiple facets of geriatric care, is available to all health practitioners and physicians at a time of increasing complexity in long-term care. Physicians can be involved by discussing adult day care as an option with the families of their elderly patients. They can also support expansion of adult day care by advocating the passage of proposed federal legislation that would pay for adult day care as a Medicare benefit.

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PROGNOSTICS IN MEDICINE

"The human body, constructed by art altogether divine, performs many and wonderful functions." So wrote Gregory one hundred and fifty years ago, and we can easily imagine him first giving expression to this incontrovertible sentiment, perhaps in face of the remarkable fulfilment of some equally remarkable prognosis, or, it may be, when confronted by events for which his judgment had in no wise prepared him.

How much more than he do we know of these wonderful functions? Amid the unparalleled activities of this nineteenth century, physiologists, too, have been busy, inquiring into, experimenting, reasoning, and determining their diverse operations. The anatomist has occupied himself in endeavouring to demonstrate the intricacies and beauties of this construction, this product of art "altogether divine," which yet, like everything in nature, is subject to laws that vary with the influences surrounding it, these too often being of a character unfitting it for the healthy performance of its "wonderful" functions. The pathologist minutely describes those changes that take place in the tissues of the body, or of parts of the body, involved either as cause or effect; and the extent of change forms the measure of danger to life. The sum of knowledge represented by researches of scientists in all these departments, with the added learning and experience peculiar to himself, must form the basis upon which every physician establishes his ability to discern and foretell, in each individual case that challenges his judgment. . . .

We stand almost aghast before the power of that fore-knowledge which enables the medical man not only to decide, in any given case of disease, its probable duration, curability, danger, complications, recurrence, etc., but also to bid the sick more rapidly to recover, and the dying to prepare for his end. The confidence it compels, and the opportunities it gives the physician to prove the value of his calling, the high and noble honesty of his words, cannot be questioned, any more than the propriety of cultivating to its utmost all the ability needful to the judicious exercise of this art of prognosis. The qualifications and attainments essential in this may be said to comprehend those which give superiority in other departments of medical science. There are some, however, more peculiarly requisite. As a foundation, the accurate discrimination of disease is indispensable, and, therefore, the habit of close observation, and the most extensive knowledge of semiology as deduced from pathology, upon which only can a correct diagnosis depend. Yet on many occasions this will prove a very inadequate guide to the prediction of ultimate results. . . .

JOHN CRONYN, MD
(*Trans NY State Med Assoc* 1890; 7:37-49)

The relationship between intrapartum obstetric care and chronic neurodevelopmental handicaps in children

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The medical malpractice crisis has caused physicians, patients, lawyers, and governmental agencies to focus on the timing and etiology of perinatal hypoxic insult and subsequent long term neurologic handicap (eg, cerebral palsy, mental retardation, and nonfebrile seizure disorders). Often, the argument centers on intrapartum events and the diagnosis of intrapartum asphyxia. This limited focus often innocently develops with the parents of children with chronic motor or cognitive handicaps.

Although a delivery is an everyday event in the life of an obstetrician, parturition is an indelible memory in the lives of parents. It is the birth or death of a dream. When something goes awry, parents remember the delivery experience in much more detail than the preceding 279 days. In the search for explanations, prenatal and delivery records are reviewed and, all too often, these "witnesses" are inconsistent, illegible, and incomplete. During this period, professionals, through their desire to answer the parents' questions, enhance the focus on delivery events.

Since 1862, intrapartum hypoxia has been cited as a cause of cerebral palsy.[1] This professional bias remains unchanged. When professionals review obstetric and neonatal charts, evidence of intrapartum asphyxia is sought. The Apgar score and fetal heart rate tracing stand out sharply as "objective" measures of intrapartum management in retrospective review. There is a lack of professional understanding of normal and abnormal fetal neurodevelopment, a lack of understanding of the pathophysiology of perinatal hypoxia, and confusion as to the reliability and validity of fetal heart rate tracings. This inadequate scientific background leads to an inaccurate assessment of the timing and etiology of long term neurologic deficits.

The degree of professional ignorance is difficult to measure; however, a recent study suggests that it is a major problem.[2] A total of 172 health care professionals, of whom 118 (69%) were pediatricians or obstetricians, were asked, "What proportion of infants with five-minute Apgar scores of 0–3 will be free of cerebral palsy and have normal intelligence?" Their estimates for the lack of cerebral palsy and mental retardation were 62.1% and 56.4%, respectively. The actual proportions are 94.9% and 96%, respectively.[2,3]

The purpose of this review is to provide a more realistic assessment of prenatal contributions to perinatal asphyxia and subsequent long term neurologic handicaps. Four principal points will be covered:

- Imprecise definition of "long term handicap" limits the examination. Retrospective studies are most susceptible to this bias.
- A knowledge of fetal neurodevelopment and the pathophysiology of asphyxia argues against intrapartum insult as the cause of long term neurologic handicap in most children.
- Epidemiologic studies suggest that less than 10% of the variance in intelligence quotient scores or in the incidence of cerebral palsy, mental retardation, or nonfebrile childhood seizures is explained by intrapartum events. These studies are supported by examinations of dental enamel, cartilage, and neural tissue of damaged neonates and children. These tissues mark antepartum rather than intrapartum insult.
- Electronic fetal monitoring and neonatal signs poorly predict long term neurologic deficit. This may lead to considerable unnecessary intervention.

DEFINITIONS

Imprecise definitions contribute significantly to the confusion surrounding the timing and etiology of chronic neurodevelopmental deficit. Many different abnormalities have been included in the term "perinatal brain damage." These include hyperactivity, dyslexia, focal language or motor abnormalities, "poor" school performance, as well as cerebral palsy, mental retardation, and nonfebrile seizure disorders. Although the inclusion of soft neurologic signs increases the number of abnormal children, there is little scientific evidence concerning the etiology or timing of such signs. Any "expert" statement relating soft neurologic abnormalities to intrapartum management is speculation.

Cerebral palsy is used as an inclusive term to describe a group of nonprogressive disorders that occur in young children, in which disease of the brain causes impairment of a motor function. More specific classification can change the presumed etiology. Spastic dysplasia is a type of cerebral palsy most often associated with preterm delivery and intraventricular hemorrhage, whereas simple atactic cerebral palsy may have a genetic origin. Unfortu-

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nately, in assessing the type, site, and severity of cerebral palsy, experts agree in only 50% of cases.[4] This confusion is partially explained by the apparent decrease in the incidence of mild or moderate cerebral palsy when the same population is examined at one and seven years of age.[5] The incidence of mild cerebral palsy at seven years falls by 71%, and that of moderate cerebral palsy falls by 39%. The seven-year incidence of severe cerebral palsy falls by only 3%. All cases of monoparesis had resolved by age seven as well as 67% of all cases of spastic diplegia and about one third of the cases of hemiparesis, atonic diplegia, and quadriplegia.

The diagnosis of mental retardation is based on a distribution curve. A test score below 1.96 standard deviations from the mean for a child of that age signifies retardation. Many peripheral factors can affect the IQ score, such as language or cultural differences, motor skills, sex differences, and prior environmental stimulation. Many studies that have examined the relationship between anoxia and intelligence have failed to control for these variables.[6] Furthermore, the clinical opinion as to the etiology of mental retardation has significant error. Chaney and Eyman[7] reviewed 1,025 autopsies of patients with mental retardation. In those in which the etiology was felt to be perinatal (14%), another cause was identified 68% of the time. A perinatal cause was defined as physical trauma or hypoxia related to the birth process.

FETAL NEURODEVELOPMENT AND THE PATHOPHYSIOLOGY OF PERINATAL ASPHYXIA

Gestational age, metabolic activity (rate of oxygen consumption per gram tissue), and rate of perfusion are the most important variables that determine the sensitivity of an organ to asphyxia.[8-12] In the adult, the cerebral cortex and brain stem have high metabolic activity and perfusion. The border areas between arterial trees (watershed zones) are at risk for hypoperfusion from hypotension, increased intracranial pressure, or both. In the adult and fetus, the major watershed areas include the periventricular regions (Fig 1) and the middle parasagittal areas. The early fetal brain (<35 weeks) has different areas of increased metabolic activity and arterial support. A large portion of cortical neurocellular differentiation and innervation occurs in the third trimester.[13,14] These fetal neurodevelopmental changes increase oxygen consumption and arterial support. Figure 1 shows the anatomic changes in a coronal section of the fetal brain at 24, 30, and 36 weeks of gestation. Throughout the third trimester, as in the adult, the brain stem is a vascular and metabolically active area. However, at 24 weeks, the subependymal germinal matrix (Fig 1) is also an active region. The subependymal germinal matrix is a conglomeration of rapidly proliferating cells and an irregular, poorly developed vascular bed. This region is highly susceptible to hypoxia and intracranial injury. On the other hand, the cortex has a relatively simple contour and less vascular support than at 36 weeks (Fig 1). Although hypoxic insult may severely damage the periventricular areas, the cortex is relatively insensitive. By 36 weeks, the cortex has become more metabolically active, with increasing vascular support and increasing sensitivity to hypoxia. The subependymal germinal matrix has virtually disappeared and is much less sensitive to hypoxic insult.

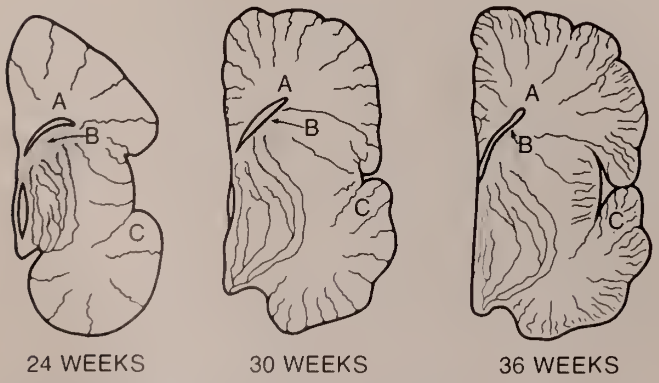

FIGURE 1. Diagrams showing anatomic changes in coronal sections of the fetal brain at 24, 30, and 36 weeks of gestation: A, periventricular watershed region; B, subependymal germinal matrix, and C, cerebral cortex.

Fetal neurosurgery on primate models has significantly furthered our understanding of the changing importance of the cerebral cortex and plasticity of the fetal brain.[13,15] Rhesus monkeys had controlled cerebral cortex resections at mid-gestation and were followed by functional testing and anatomic examination after birth. Thalamocortical innervation of the occipital lobe occurs between 91 to 124 embryonic days. This corresponds to a human gestation of 22 to 30 weeks. Cortico-cortical and callosal fibers innervate the cortex between 124 to 150 embryonic days (30 to 36.5 weeks of human gestation). These innervations correspond to the increase in fissuring and vascular support shown in Figure 1. Removal of the functional associative cortex between 102 and 119 embryonic days (24.5 to 29 weeks of human gestation) caused little change on testing of function after birth. On the other hand, injury later in gestation produced permanent functional changes.[15]

Term Rhesus monkeys were used to determine the physical and functional effects of intrauterine asphyxia. Two different patterns of brain damage have emerged with acute total asphyxia or prolonged partial asphyxia.[8,9] After 10–13 minutes of acute total asphyxia, the brain stem structures, especially the inferior colliculi, are damaged without generalized edema. In the human, this pattern would be seen after total abruption or cord prolapse. Prolonged partial asphyxia is produced by several hours of oxygen contents between 0.4–0.8 volumes percent and a fetal pH <7.00. Damage from prolonged partial asphyxia occurs in multiple organs: central nervous system, heart, lungs, kidneys, liver, and gastrointestinal tract.

In prolonged partial asphyxia, a combination of hypotension caused by hypoxic cardiomyopathy and intracranial hypoperfusion caused by increased intracranial pressure leads to neural damage. The increased intracranial pressure is caused by increased interstitial fluid from hypoxic endothelial damage and extravascular metabolic derangements. The neural damage is variable and can involve the entire cortical surface or may be localized to the parasagittal cortical and periventricular regions. In the human, prolonged partial asphyxia corresponds to the most common situation, in which a previously compromised fetus cannot tolerate the normal hypoxic stresses of labor. The classic fetal cardiographic changes, late decelerations, appear hours prior to acidosis and permanent brain damage.[16,17] Evidence of vagal damage, such as tachycardia, decreased variability, and variables with an "overshoot" pattern (Fig 2) may indicate more permanent damage.[18]

An episode of acute asphyxia severe enough to cause permanent brain damage is followed by a consistent pattern of deficit and recovery whether the victim is a Rhesus monkey,[8] a human neonate,[19,20] a child after a near-drowning event,[21] or an adult after cardiac arrest.[22] Invariably, there is a period of six or more hours during which the neonate is comatose or deeply stuporous. There is little spontaneous or elicited movement. Between 12 and 24 hours, there is an apparent improvement in activity. Often, after 36 hours, this deteriorates to a variable period of obtundation or hypotonia. Early neonatal seizures (30–50%) usually occur 12–36 hours after the insult. However, seizures can occur at any time in the presence of metabolic disorders (hypoglycemia, hypocalcemia), intracranial trauma, or hemorrhage.

Neonatal seizures point to an area of the brain with a lower seizure threshold than the surrounding area. These areas of lower threshold may be acutely injured tissue or scar tissue from prenatal injury. Hypoxia, hypoperfusion, or mild metabolic derangements may initiate seizure activity in scarred areas. Clinically, early neonatal seizure without signs of cortical dysfunction (coma) or other organ system damage is most likely due to injury prior to the onset of labor.

An acute asphyxial episode severe enough to cause permanent brain damage must necessarily produce damage to other organ systems. In fact, hypoxic cardiomyopathy is an essential component in prolonged partial asphyxia. Hypoxia leads to circulatory failure with consequent local venous thrombosis and focal hemorrhagic infarctional damage in the heart, lungs, kidneys, adrenals, brain, and other viscera.[10,11,23] The incidence of multisystem involvement among 59 neonates with Apgar scores ≤5 is shown in Table I.[24]

ROLE OF BRAIN DAMAGE PRIOR TO LABOR

Numerous epidemiologic studies have examined the timing and etiology of perinatal brain damage.[25–30] Epidemiologic studies can be descriptive, retrospective, case-controlled, or prospective cohort design in nature. Descriptive studies arbitrarily define timing and etiology variables and review the medical histories of patients with disease for the presence or absence of the variables. For example, Holm[31] studied a population of 142 children with cerebral palsy in order to assign the timing and etiology of their cerebral palsy. The etiology of 71 (50%) was felt to be prenatal (preconception and first trimester) and 47 (33%) were felt to be perinatal (third trimester) in origin. Seventy percent of cases with a perinatal etiology occurred in children with birthweights less than 2,268 grams. Postnatal etiology occurred in 10%, and 7% had mixed etiologies. Cerebral palsy was considered medically preventable in 6% and possibly preventable in 4% of full term cases with perinatal etiology. However, the major problem in this study, as in all descriptive studies, is the arbitrary assignment of cause and timing and the lack of controls to quantify significance and strength of risk factors.

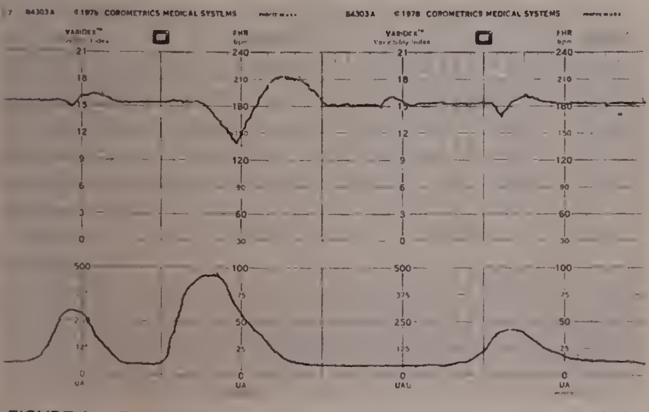

FIGURE 2. Fetal heart rate changes demonstrating parasympathetic injury. This abnormality is indicated by the presence of tachycardia, loss of variability, and an "overshoot" pattern.

TABLE I. Multisystem Involvement of 59 Asphyxiated Newborns (Apgar Scores ≤5)

Organ System Involved	Percent
Pulmonary	39
Cardiovascular	34
Central nervous	24
Gastrointestinal	20
Renal	15
Two or more systems	53

The majority of studies are retrospective, case-controlled studies. Prematurity, third trimester bleeding, growth retardation, multiple gestation, toxemia, and poor reproductive history are consistently identified as prenatal risk factors for long term neurologic handicap. Intrapartum and neonatal risk factors include breech presentation, meconium staining, chorioamnionitis, polyhydramnios, low birthweight, neonatal seizures, hypotonia, low Apgar scores, and congenital abnormalities. Interestingly, an increased risk of malpractice litigation for brain-damaged infants reflects the presence of prenatal factors rather than intrapartum management at term (Table II). In the latter study, Cornblath and Clark[32] reviewed 250 malpractice claims for brain-damaged infants. The incidence of 27 risk factors among the claims was compared to the incidence in the general population. Among the 250 claims, 31%, 42%, and 27% of brain-damage cases were classified as preventable, nonpreventable, and indeterminate, respectively.

Unfortunately, retrospective, case-controlled studies have imprecise and noncomparable definitions of dependent and independent variables, a lack of reliable retrospective data, and bias in the selection of controls. Faulty definitions of the dependent variables, cerebral palsy and mental retardation, have been discussed earlier. In retrospective studies, the independent variables may not be reliable. The most accurate record of prenatal risk factors occurs in the neonate who is identified neonatally, and whose mother received care within the same institution. Unfortunately, a sizable proportion of damaged children cannot be identified in the neonatal period. In prospective studies, a diagnosis of definite neurologic pathology oc-

TABLE II. Relative Risk of Lawsuit Among Obstetric Risk Factors[32]

Risk Factor	Relative Risk*
Meconium	1.5
Mid- or high-forceps	1.6
Cesarean section	2.3
Infertility	2.7
Postdates (≥42 weeks)	2.8
Diabetes	2.9
Birthweight ≥4.5 kg	3.5
Gestational age ≤35 weeks	3.8
Breech presentation	4.2
Apgar score <5 at one minute	4.3
Twins	4.7
Cord prolapse	5.0
Maternal obesity ≥91 kg	6.0
Congenital malformation	10.5
Birthweight <2.26 kg	11.0
Apgar score <5 at five minutes	13.3
Congenital infection	13.6
Neonatal hypotonia	14.1
Neonatal seizure	16.0
Third trimester bleeding	16.2

* $P < 0.05$.

curred in 23% and suspected neurologic pathology in 34% of children with cerebral palsy during their neonatal period.[3,33] Among children with cerebral palsy and birthweights over 2,000 grams, only 20% had evidence of neonatal asphyxia and 14% had evidence of seizures.[3,33] In addition, only 5–18% of term infants with severe neonatal signs will have cerebral palsy at seven years of age (Table III).[3,33] Thus, retrospective studies of children with cerebral palsy may depend on independent variables obtained from five–seven-year-old charts of "normal" patients.

Prospective cohort studies have the significant advantage of standardized definitions and more complete records. These advantages allow an improved statistical analysis. The disadvantages are the required size of the study population when incidence of the disease is low (0.1%). The analysis from large projects may take years, during which time advances in perinatal care may weaken the application of the results.

The National Collaborative Perinatal Project (NCPP) of the National Institutes of Neurological and Communicative Disorders and Strokes is an example of a large prospective cohort study in the United States. In this study, 12 cooperative university hospitals registered approximately 54,000 pregnant women between 1959 and 1966 and followed them and their children until the offspring were seven years old. Detailed descriptions of the sampling technique and study procedures were provided.[34] The sample was at higher risk than the general population because the patients were identified at referral institutions and had an atypical racial distribution: 46% were white, 46% were black, and 8% were from other races.

TABLE III. Neonatal Signs as Predictors of Cerebral Palsy[3,33]

	Prevalence of Cerebral Palsy in Survivors Gestational Ages	
Sign in Neonate	36 weeks or less	37 weeks or greater
Ten-minute Apgar score 0–3	12	13
Multiple apneic episodes	11	5
Neonatal seizures	19	18
Overall impression, definite brain abnormality	20	15

A series of recent articles has reviewed the relationships between prenatal, antepartum, intrapartum, and neonatal risk factors for cerebral palsy, non-febrile seizure disorders, and mental retardation in the NCPP data.[35-41] The NCPP data were examined, using univariate and logistic regression analyses, with cerebral palsy as the dependent variable. The univariate analysis evaluated 400 characteristics of mothers, pregnancies, and deliveries.[36] Significant prepregnancy factors among patients with birthweights over 2,500 included siblings with mental retardation, abnormal menstrual cycles, maternal seizure disorders, and maternal mental retardation. Severe proteinuria, maternal seizures, polyhydramnios, and third-trimester bleeding were significant prenatal factors. Amnionitis, placenta previa, and fetal malpresentation were significant intrapartum factors. The maximum observed incidence of cerebral palsy was less than 3% when a significant risk factor was present.

Subsequently, stepwise logistic regression was performed.[38] Maternal mental retardation, breech presentation (but not delivery), severe proteinuria, maternal seizures during pregnancy, and maternal hypothroidism were the significant risk factors prior to labor. Complications of labor and delivery added little to the model. When the principal risk factors present when labor had begun were considered, patients with the upper fifth percentile of estimated risk, ie, high estimated risk, were seen to have contributed 34% of the cases of cerebral palsy. When intrapartum and neonatal risk factors were added, only 3% more cases of cerebral palsy (total 37%) were identified in the high risk group.

The relationship between pregnancy factors and nonfebrile seizure disorders has been examined in a similar fashion.[35] Late third-trimester and intrapartum complications, such as abruptio placentae, use of midforceps, and meconium staining, were not associated with nonfebrile seizure disorders in children without cerebral palsy. When stratified by low five-minute Apgar scores less than 6, any late third-trimester complication plus a low Apgar score contributed only 2.2% of patients with nonfebrile seizures. Of 259 patients with nonfebrile seizures, 58.1% had Apgar scores of 6 to 10 with one or more complications, and 39% had Apgar scores of 6 to 10 without complications. Using sequential logistic regression, only 2.6% of the variance in non-febrile seizures could be explained by pregnancy, intrapartum, and neonatal risk factors among children without cerebral palsy or other motor disorders. Even when the prediction is confined to patients with the highest risk (upper fifth percentile), only 14.7% of patients with seizures and without cerebral palsy or minor motor disorders are identified. The patients with the highest risk contributed 21% of the total patients with seizures.

The NCPP data also confirm that the variance of IQ scores at seven years is dominated by constitutional and socioeconomic factors.[39,40] Socioeconomic class, race, maternal age, maternal education, and maternal IQ explained 37% of the variance in IQ scores. Prenatal and antenatal factors, including prior perinatal loss, smoking, hypertension, anemia, and alcoholism, were associated with only 2% of the variance.[39] Intrapartum factors, such as arrest of active labor, breech delivery, and postdates, did not explain any of the variance in IQ scores.[39] Signs of perinatal anoxia (meconium, low Apgar scores, apnea, or need for resuscitation) explained less than 1% of WISC intelligence quotient scores and none of the variance in visual motor performance scores (Bender-Gestalt error scores).[40]

Histologic studies support the role of prenatal or antepartum, rather than intrapartum, factors in chronic neurodevelopmental handicaps. Periventricular injury and subsequent cerebral palsy is the central nervous system marker for prenatal insult occuring at less than 35 weeks.[10,42-45] One study[44] examined brain tissue in 31 neonates who had died within the first week of life. The pattern of injury, recovery, and scarring indicated that five of six term infants and ten of 25 preterm infants had suffered significant brain injury prior to the onset of labor. Skeletal structures can also mark injury in the prenatal period. Hypoxia or metabolic insult will leave its mark in the formation of dental enamel and cartilage.

The location of enamel hypoplasia indicates when in the third trimester the injury occurred. The formation of enamel matrix by ameloblasts proceeds in a regular fashion, beginning on the primary central incisor and first primary molar during the third month of gestation. The matrix formation begins at the tips of the tooth cusp and is deposited progressively from the dentinoenamel junction outward and downward until the entire crown is formed. Calcification is essentially complete in the primary central and lateral incisor by nine months. Approximately the lower third to half of the primary canine and the first and second primary molars are calcified by 40 weeks. Injury to the ameloblasts during this process results in abnormal matrix and enamel hypoplasia. The position of the defect indicates the time and duration of the insult. Between 50% and 70% of children with abnormal neurologic examination have enamel hypoplasia.[46,47] When a child's prenatal record indicated a perinatal complication, 53% had positive dental examinations.[46]

Chondroblasts, like ameloblasts, produce cartilage in an orderly fashion without bridging or banding. Hypoxic or metabolic insult causes cessation of growth, bizarre column formation, bridging and/or banding.[48] In a study of 570 stillbirths or neonatal deaths occurring within 48 hours of birth, 60% of children born at 32 weeks or less showed evidence of injury prior to labor. The proportion of term infants was 75%, and over 90% of those delivered at or after 41 weeks had evidence of injury prior to the onset of labor.

RELIABILITY AND VALIDITY OF FETAL HEART RATE MONITORING

Fetal monitoring has two interrelated goals: to distinguish the healthy from the diseased fetus and to predict the progression or development of a hypoxic state within an arbitrary time period. Fetal heart rate monitoring is widely considered a diagnostic and predictive test of uteroplacental insufficiency and hypoxia, and, all too often, the fetal monitor tracing is used to diagnose physician "malpractice." A review of the reliability, validity, and predictive strength of fetal heart rate tracing raises major questions.

Reliability is the measure of the exactness of a test; the variation of test results given the same disease state. Test results may have inter- and intra-observer variation. The variation in reading the same fetal heart rate tracing is considerable. Intra-observer agreement on the reading of nonstress tests is 40–50%.[49] In a review of contraction stress tests, the agreement of four of five obstetric specialists on positive tests was 18% and negative tests was 62%.[50] When 14 intrapartum fetal heart rate tracings were presented to 12 researchers and educators who had contributed substantially to the literature on fetal heart rate pattern interpretation, the average pair-wise agreement was 68%.[51] When fetal scalp sampling results were added, the average agreement on management was 59%.[51]

Validity is the measure of how well the test reflects the true disease state. Fetal heart rate patterns are used to diagnose asphyxia/hypoxia. This information may be correlated with subsequent perinatal mortality and the occurrence of chronic neurodevelopmental handicaps. Efficiency evaluation of the fetal heart rate pattern as a diagnostic test is hampered by the extremely low prevalence of perinatal mortality and long term neurologic deficits (0.5 to 1 out of 1,000 births). Therefore, more frequent but less valid outcomes are used to evaluate fetal heart rate monitoring. These "soft" signs of asphyxia include meconium staining, low Apgar scores, acidosis, operations for fetal distress, intrauterine growth retardation, etc. Fetal heart rate patterns with fetal scalp sampling identify a population of neonates at risk for these outcomes. When an Apgar score of less than 7 at one minute is used as an outcome parameter, fetal heart rate pattern plus fetal scalp sampling is 32% sensitive, 91.6% specific, and has false positive and false negative rates of 43.6% and 19.9%, respectively.[52]

Recent randomized studies have failed to show a superiority of electronic fetal heart rate monitoring over auscultation, using a strict protocol with one-to-one nursing care.[53] None of these randomized, controlled trials have shown a significant difference in perinatal death, low cord pH, low Apgar scores, or neurologic signs other than neonatal seizures. The largest study,[54] which included 12,964 patients, showed a statistical decrease in neonatal seizures in the electronically monitored group (12 vs 27). At one year of age, three children in each group were judged to have major neurologic disabilities. However, the small size of these studies does not eliminate the risk of beta error (that is, a true difference being missed). A recent prospective, but not randomized, trial[55] compared selective electronic fetal monitoring versus universal electronic fetal monitoring in 34,995 patients. This trial did not demonstrate any significant differences in intrapartum fetal death, low Apgar scores, need for assisted ventilation, or neonatal seizures. This study suggests a low risk of failing to detect a true difference in the incidence of neurologic disabilities.

Another way to evaluate the utility of a test is to prospectively record the proportion of "bad" outcomes the test identifies. Sykes and co-workers[56] performed a prospective audit of obstetric diagnosis of fetal distress using the electronic fetal monitor and scalp sampling. All neonates had umbilical artery blood gas and Apgar scores. Neonates with a cord pH less than 7.12, a base deficit greater than 12 mmol/L, and an Apgar score less than 7 at one minute were considered to have "bad" outcomes. Operative delivery for fetal distress was considered as evidence of the intrapartum diagnosis of fetal distress. Among patients with continuous electronic fetal monitoring, only 22% of acidotic neonates had operative deliveries for fetal distress. On the other hand, infants of 86% of mothers who had operative deliveries for fetal distress were without acidosis.

One worrisome source of inaccuracy in fetal heart rate pattern interpretation is the presence of congenital abnormalities in the fetus. Approximately 50% of abnormal fetuses have pathologic fetal heart rate tracings.[57–60] Thirty-five percent of perinatal mortality associated with an obstetric diagnosis of fetal distress was caused by lethal congenital abnormalities.[61] Most of these neonates were delivered by cesarean section.[61]

The relationship between a fetal heart rate pattern and neurologic symptoms has been incompletely studied. Beischer and coworkers[62] studied the long term outcome of 72 patients who had abnormal antepartum cardiotocographs. There were 20 perinatal deaths: six were caused by congenital abnormalities and one occurred in a term infant (3,015 grams). This latter death was associated with staphylococcal septicemia and necrotizing enterocolitis. The remainder of the deaths were associated with preterm delivery. Four of 52 neonates (8%) had major neurologic handicaps; only one was full term. Eight (15%) had minor handicaps; none were full term.

Keegan and coworkers[63] examined heart rate patterns and early neonatal seizures. They found a significant relationship between abnormal heart rate patterns with absent variability and early neonatal seizures; there were seizures in eight of 66 controls, compared with 30 of 66 infants with abnormal tracings. However, only 45% of infants with seizures had long term deficits. In a subgroup with a diagnosis of fetal distress and delivery within one half hour of the diagnosis, all neonates had seizures, and long term disability was present in 45%. Painter and coworkers[64] performed serial neurologic examinations after abnormal heart rate patterns were recorded intrapartum. The sensitivity of late decelerations or severe variable decelerations was about 50% predictive for at least one abnormal neurologic examination in two years. By five years of age, all infants were normal, except for one with congenital abnormalities.

The lack of reliability and validity of fetal heart monitoring brings to focus the following question: What is the potential for unnecessary intervention? The following facts seem relevant to this issue:

- The prevalence of an abnormal tracing with a low scalp pH (less than 7.25) is about 2%.[54,55]
- An abnormal fetal heart rate pattern plus a low scalp pH has a sensitivity of 32% and a specificity of 91.6% in predicting a one-minute Apgar score less than 7.[52]
- A birthweight of over 2,500 grams and a one-minute Apgar score less than 7 predicts an infant mortality of 26 out of 1,000 births and an incidence of cerebral palsy of five out of 1,000 survivors.[3,33] A one-minute Apgar score less than 4 predicts an incidence of mental retardation of 39 out of 1,000 survivors.[40]
- In infants over 2,500 grams, approximately 35%, 10%, and 2% of perinatal mortality, cerebral palsy, and mental retardation, respectively, are caused by asphyxia.[31,39,61]

Table IV depicts the predictive values of fetal heart rate monitoring with scalp sampling. If a Cesarean section is performed for all cases of abnormal tracing with scalp pH samples less than 7.25, then more than 499 cesarean sections would be performed to prevent one intrapartum death from asphyxia. Even more "unnecessary" cesarean sections would be performed to prevent either mental retardation or cerebral palsy. These calculations do not address the issue of reliability[51] or preventability.[63] Both of these factors may further increase the risk.

The role of intrapartum management in the development of long term handicap is elucidated by the examination of documented "bad" obstetrical care to poor outcomes. Since "bad" care cannot be randomized, a case-controlled method is necessary. Niswander and coworkers[65] used the Oxford Cerebral Palsy Data to estimate the contribution of poor obstetric management to adverse outcome. The adverse outcomes were fetal deaths due to asphyxia or trauma, terminal apnea, early neonatal seizures, and cerebral palsy. Suboptimal obstetrical care was designated by independent observers who were blinded to the neonatal outcome. Suboptimal obstetrical response to fetal distress was defined as failure to assess fetal acid-base status or to effect delivery in the presence of prolonged bradycardia (within 40 minutes), severe variables (within 25 minutes), or late decelerations (within 90 minutes).

Table V compares the incidence of adverse outcome in cases with and without adequate response to fetal distress. Attributable risks are estimates of the numbers of each outcome likely to be caused by 1,000 incidences of suboptimal care. Population-attributable risks are estimates of the proportion of cases of each adverse outcome that

TABLE IV. Predictive Value of Electronic Fetal Monitoring with Scalp Sample

Outcome Parameter	Prevalence	Predictive Value of a Positive Test*	Ratio of True Positives to False Positives
One-minute Apgar <7	2%	7.5%	1:13
One-minute Apgar <7 with infant mortality secondary to asphyxia	.05%	0.2%	1:500
One-minute Apgar <7 with cerebral palsy secondary to intrapartum asphyxia	.01%	0.04%	1:2500
One-minute Apgar <7 with mental retardation secondary to intrapartum asphyxia	.02%	0.06%	1:1667

* Positive test equals abnormal fetal heart rate pattern and a scalp pH ≤7.24. Assumption: Sensitivity, 32%; specificity, 92%.

TABLE V. Suboptimal Obstetric Response to Severe Intrapartum Distress

Outcome Parameter	Relative Risk	Incidence of Suboptimal Care	Attributable Risk per 1000 with Suboptimal Care	Population Attributable Risk (%)
Fetal death from asphyxia or trauma	18.0	1/4	4.2	18.9
Terminal apnea	4.1*	5/87	16.8	4.1
Early neonatal seizures	6.6*	3/33	11.4	7.0
Cerebral palsy	1.2	0/34	0.4	0.2

* $P < 0.05$ *vs* controls.

might be attributed to suboptimal obstetrical care. It is clear from this study that more than 98% of babies whose mothers have received suboptimal obstetric response to fetal distress will escape perinatal death, terminal apnea, neonatal seizures, or cerebral palsy. It is also clear that most adverse outcomes cannot be attributed to suboptimal response to fetal distress.

CONCLUSIONS

Despite the current focus of malpractice litigation on "brain damaged" children, the relationship between intrapartum management at term and chronic neurodevelopmental handicaps is weak. A knowledge of fetal neurodevelopment and the pathophysiology of acute asphyxia allows us to explain why 90% of patients with cerebral palsy or mental retardation are delivered with few early neonatal signs. Cerebral palsy without mental retardation is the result of insult prior to 35 weeks. An intrapartum asphyxial insult at term which results in mental retardation and cerebral palsy must also manifest multisystemic disease, including coma, hypotonia, seizures, congestive heart failure, and paralytic ileus, in addition to apnea and/or respiratory distress. Neonatal seizures, hypotonia, and apnea may exist in an otherwise healthy infant who has suffered significant central nervous system injury prior to labor.

The major role of prenatal or antepartum, rather than intrapartum, factors in the causation of chronic neurodevelopmental handicaps is evident from epidemiologic studies and histologic examination of brain tissue, dental enamel, and cartilage. The study of risk factors suggests that less than 5–10% of the variance in IQ scores, or the incidence of nonfebrile seizures and cerebral palsy, can be explained by intrapartum factors. Suboptimal obstetric response to severe intrapartum fetal distress contributes little (less than 7%) to the incidence of cerebral palsy, terminal apnea, or neonatal seizures. Likewise, 99% of babies whose mothers had suboptimal care show no neurodevelopmental handicap.

As a diagnostic test, fetal heart rate monitoring with fetal scalp sampling is extremely crude. This test predicts with some accuracy the soft signs of fetal stress. These include a one-minute Apgar score less than 7, meconium staining, operative delivery, and growth retardation. However, fetal heart rate monitoring with scalp pH sampling poorly predicts intrapartum death, cerebral palsy or mental retardation. The data relating specific heart rate patterns to long term neurologic handicap in normally formed neonates are incomplete. The available data suggest a poor correlation.

There are at least three additional factors that indicate caution in the interpretation and management of fetal heart rate tracings. First, most abnormal tests are false positives. The ratio may be higher than 500 to 1. Secondly, at term, many fetuses with heart rate abnormalities may have developmental or genetic abnormalities. Finally, operative delivery for fetal distress, especially cesarean section, increases many fold the morbidity and mortality of childbirth.

Despite the latter weaknesses and concerns, fetal heart rate monitoring is the best method available to diagnose the fetus who is not tolerating the normal hypoxic stresses of labor. The superiority of electronic fetal monitoring over auscultation, using a strict protocol and one-to-one nursing care, in low risk patients is not clear. The vast majority of fetuses who do not tolerate labor are those who have suffered insults prior to the onset of labor. High risk screening helps to identify these fetuses. Fetal tolerance to labor is determined by the severity of the prior insults. Fetal tolerance to the normal hypoxic stress of labor is measured by fetal blood acidosis, and the heart rate pattern is a screening test for fetal acidosis. In the normally grown term fetus, fetal heart rate abnormalities precede acidosis by at least two hours.[16,17] This grace period may be reduced in growth-retarded or preterm fetuses. Once acidosis develops, the rapidity of neurologic damage and/or sudden fetal death is not clear. Although obstetricians recognize the low predictability of this sign, they are obligated to act to reduce the stress of labor.

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A 73-year-old woman with left lower quadrant pain accompanied by swelling and bluish discoloration of the left lower extremity

CASE PRESENTATION

Lewis Zulick, MD (Surgical Resident): A 73-year-old woman was transferred from another hospital for suspected acute arterial insufficiency of the left lower extremity. Several days prior to her hospital admission, she sustained a bee sting on the left knee. Twenty-four hours prior to her arrival here, severe pain developed in the left lower quadrant of her abdomen and in the left thigh, accompanied by swelling and bluish discoloration of the left lower extremity. Following admission to the other hospital, she was transferred here because of suspected acute arterial insufficiency.

The patient's past medical history was significant for cyclic neutropenia, which had first been diagnosed in 1933, with a consistently low white blood cell count of 3,000–5,000/μL, with neutrophil leukocyte percentages between 15% and 80%. There was no history of frequent or unusual infections. A bone marrow aspiration in 1953 had shown some decrease in neutrophilic granulocytes, but was otherwise unremarkable. The patient was seen clinically for this condition at this hospital during the 1950s and 1960s.

The patient had had a tonsillectomy and adenoidectomy in 1918, an appendectomy in 1933, lysis of adhesions and left ovarian cystectomy in 1935, and drainage of the right maxillary sinus in 1941. A positive purified protein derivative (PPD) skin test and the accompanying chest film suggested tuberculosis in the past, though there was no corroborative clinical history.

On physical examination after transfer to the emergency department, the patient was found to be in marked distress, breathing rapidly, with a flushed, anxious countenance. Her blood pressure was 130/70 mm Hg; pulse rate, 120/min; respiratory rate, 36/min; and oral temperature, 36.5° C. There was no palpable lymphadenopathy. Her lungs were clear on percussion and auscultation; her heart rhythm was regular. The abdomen was nontender and soft without bowel sounds; no masses and no enlarged organs were palpable. There was marked subcutaneous crepitus on the left side of the abdomen up to the costal margins. The left lower extremity was swollen from the thigh to the ankle, especially in the proximal areas where the skin was tense and shiny with bluish mottling. The entire left lower extremity was cool to the touch; the femoral, posterior tibial, and dorsalis pedis pulses were intact bilaterally. There was light but distinct crepitus in the left lower extremity down to the ankle. Sensation to soft touch and superficial pain was absent in the entire left lower extremity in a stocking distribution; the patient could not move the entire left lower extremity.

Shortly after her initial examination, the patient was observed to have bullae containing dark red fluid on the posterior aspect of the left thigh. Gram stain on the aspirated fluid showed gram-positive bacilli without spores.

The hematocrit was 48% (compared to 36% earlier on the same day at the other hospital); the white blood cell count was 5,000/μL, with 3% segmented polymorphonuclear leukocytes, 11% immature band forms, 50% normal lymphocytes, 1% reactive lymphocytes, 4% monocytes, 1% eosinophils, 24% metamyelocytes, and 6% myelocytes. The mean corpuscular volume was 89 femtoliters (normal range, 86–98 fL), the mean corpuscular hemoglobin was 29.5 picograms (normal range, 27–32 pg/cell), and the mean corpuscular hemoglobin concentration was 33% (normal range, 32–36%). The platelet count was 239,000/μl, the prothrombin time (PT) was 14.3 seconds (normal range, 11.2–12.8 sec), and the activated partial thromboplastin time (APTT) was 41.4 seconds (normal range, 25.0–34.0 sec). Six hours after admission, the platelet count fell to 68,000/μL.

Serum electrolyte levels were as follows: sodium, 132 milliosmols per liter; potassium, 5.1 mmol/L; chloride, 99 mmol/L; and bicarbonate content, 10.3 mmol/L. The blood chemistry values were as follows: glucose, 333 mg/dL; urea nitrogen, 23 mg/dL; creatinine, 2.4 mg/dL; creatine phosphokinase, 34,630 U/L (normal range, 0–200 U/L); lactate dehydrogenase, 1,438 U/L (normal range, 60–225 U/L); alanine aminotransferase, 119 U/L (normal range, 0–45 U/L); aspartate aminotransferase, 523 U/L (normal range, 0–40 U/L); and alkaline phosphatase, 90 U/L (normal range, 50–136 U/L). The arterial blood gas values were pH, 7.135; P_{CO_2}, 18.3 mm Hg; and P_{O_2}, 88 mm Hg.

Vittal B. Chanamolu, MD (Attending Radiologist): The initial chest film in the emergency department was unremarkable; thereafter the patient developed pulmonary edema and an endotracheal tube was inserted. The next film taken after the surgical procedure shows a Swan-Ganz catheter in place.

Based on a clinicopathologic conference (CPC) held at Mary Imogene Bassett Hospital, Cooperstown, NY, on December 9, 1987.

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The radiographs of the lower abdomen and the left thigh that were taken on admission (Fig 1) showed considerable collections of subcutaneous gas in the soft tissues of the left thigh, left buttock, and abdomen. This was evidently due to massive infection, most likely by gas-forming organisms. No evidence of osteomyelitis or any other bony abnormalities was seen.

Dr Zulick: The patient received two ampules of sodium bicarbonate intravenously in the emergency department, together with one dose each of gentamicin, ampicillin, penicillin, and clindamycin. She was taken to the operating room three hours after transfer. Her initial blood pressure in the operating room was 110/60 mm Hg, and her pulse was 120/min. However, prior to induction of anesthesia, her systolic blood pressure fell to 70: she was quickly resuscitated to a systolic blood pressure of 90 mm Hg with rapid infusion of fluids. Cardiac arrest occurred on induction of anesthesia, but she was successfully resuscitated and thereafter underwent massive debridement of nonviable tissue in the left lower extremity, left anterolateral abdominal wall, perineum, and left vulva. All tissue on the left thigh and around the left hip girdle appeared to be nonviable; the muscle appeared beefy-red, edematous, and did not contract under the scalpel. Small blood vessels extruded clots.

Extensive fasciotomies were performed in the left leg and foot; the major vessels of the lower extremity appeared to be patent. The musculature of most of the left side of the abdominal wall was debrided. After surgery, the patient was transferred to the intensive care unit; maximum doses of dopamine and epinephrine were continued. Despite maximum pharmacologic and respiratory support, the patient became progressively more acidotic and hypotensive, and died approximately 14 hours after surgery.

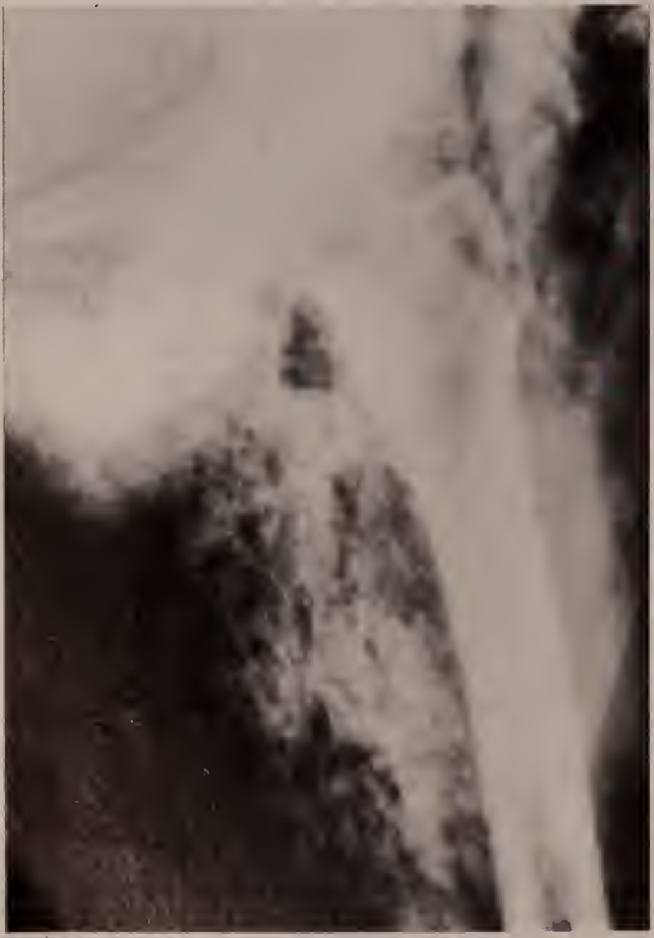

FIGURE 1. Radiograph showing collections of subcutaneous gas in the soft tissues of the left thigh, left buttock, and abdomen.

Wayne Bottner, MD (Attending Hematologist): Were any blood counts done after the operative procedure?

Dr Zulick: A complete blood count done following the operation showed that the hematocrit had fallen to 21%. The white blood cell count after surgery was 12,200/μL; no concurrent differential count is available.

Dr Bottner: Did the patient have a great deal of bleeding during the surgery?

Dr Zulick: Not a great deal, but she underwent extensive debridement, with removal of all of the soft tissue on the thigh, leaving the bone exposed.

Dr Bottner: What was the white blood cell count at the other hospital prior to transfer?

Dr Zulick: We do not have that information.

I would like to make several comments about clostridial myonecrosis, which this patient's clinical course and the Gram stain establish as the diagnosis. Although this clinical picture is the most dramatic form of a clostridial soft-tissue infection, there is, however, a range of such infections of varying severity. Clostridial infection can also cause suppurative infection, tetanus, and enterocolitis.

Clostridia are gram-positive anaerobic, spore-forming bacilli which are widely present in soil and in animals. In humans, such organisms are found in the gastrointestinal tract and in the female genital tract, as well as sometimes on the skin and in the mouth. Most clostridial infections are due to *Clostridium perfringens*, but many other species are known to cause infection. Clostridia elaborate many different forms of toxin, some of which are responsible for the systemic effects seen in some clostridial infections.

Clostridia can be cultured from many wounds; it is reported to be present in 10–30% of civilian wounds and up to 80% of war wounds. But the presence of clostridia by itself does not necessarily imply that a serious clostridial infection exists. Even positive clostridial blood cultures may not necessarily indicate clinical evidence of sepsis. Perhaps it could represent a transient bacteremia. The most common clostridial infection is suppurative and is usually part of a polymicrobial intra-abdominal abscess. Clostridia have been cultured from 25% of intra-abdominal infections.

Clostridial soft-tissue infections have been grouped into several clinical categories. The definition and clinical significance of each category varies with the reviewer,[1,2] but basically the conditions range from relatively benign and clinically unimportant infections to the kind of infection that occurred in the patient under discussion.

A localized soft-tissue or skin infection, indolent and without systemic signs of toxicity, can be present in such areas as amputation stumps, decubitus ulcers, diabetic foot ulcers, and perirectal abscesses. Those infections are usually polymicrobial, and can progress to the form of myonecrosis, which is usually heralded by rapidly advancing soft-tissue crepitus.

Clostridial cellulitis should be distinguished from clostridial myonecrosis; crepitus can be present in both, but in the former there is usually not as much pain as with myonecrosis. A distinguishing feature of the cellulitis is the confinement of the pathologic process to the subcutaneous tissue and to the fascial planes, without myonecrosis. Systemic signs of toxicity, such as hemolysis or renal failure, are always present, according to some clinical authorities; but according to others, these findings can be variable.

Clostridial cellulitis has been associated with silent carcinoma, particularly colon carcinoma. Bowel perforation is suspected as the initiating event, but this is not always found. *Clostridium septicum* is the most common agent with this kind of infection, as opposed to most clostridial infections in which *C perfringens* is the most common. The infection can proceed along fascial planes or be "metastatic," that is, blood-borne.

Clostridial myonecrosis is the most devastating type of clostridial soft-tissue infection. Its incidence in a study of 187,936 civilian and war wounds was 1.76%.[3] In 60% of the cases, the infection is related to trauma; in 40% of the cases it is seen in the postoperative patient. Predisposing factors to its development in trauma include devitalization of muscle, contamination of the wound with soil or clothing, and delayed treatment. Postoperative infection is most often seen in bowel surgery, especially with bowel perforation, in appendiceal rupture, and in perirectal and biliary tract infection with surgery.

Myonecrosis will usually, but not always, exhibit crepitus. Crepitus, in any case, is a very late finding. The average incubation period is 20 to 53 hours, depending on which reference one consults; and there is rapid infection in muscles with necrosis of muscle and thromboses of the small vessels. Toxin elaborated by the organism is responsible for systemic effects such as hemolysis, destruction of platelets, and capillary damage. The most common, earliest important symptom is pain over the area of the wound. Fever is usually not present. The patient usually has tachycardia, but is not hypotensive; the skin over the affected area is usually tense and shiny. The muscle may have a pale, edematous, "cooked" appearance, or it may be beefy-red or frankly necrotic. It does not contract when stimulated. There is a dirty-brown, foul-smelling discharge, and in late stages bullae of the skin develop which contain dark red fluid.

The most common clostridial species in this type of clinical infection is *Clostridium perfringens;* other species found are *Clostridium novyi*, *C septicum*, and *C bifermentans*. On Gram stain, gram-positive rods, usually without spores, are seen in the marked absence of inflammatory cells. The hematocrit is often low, and radiographs may show gas in the tissues.

There also seems to be a range in the clinical severity of myonecrosis. For example, postoperative or posttraumatic infection of the abdominal wall tends to present a relatively more indolent course, with less systemic signs of toxicity. In Altemeier and Fullen's[2] review of 54 cases of posttraumatic clostridial myonecrosis, mortality was 14.8%. On the other hand, in so-called nontraumatic myonecrosis, that is, without known injury which predisposed to the infection, the clinical course is extemely rapid, with marked systemic toxicity, and with a mortality of 50–100%. This type of clostridial infection is quite rare and is often associated with occult malignancies, including colon and pancreatic cancer, lymphoma, and leukemia. In this type of myonecrosis, as in the cellulitis that is related to occult carcinoma, *Clostridium septicum* is the most common organism. The presumed pathogenesis in these cases is an infection with blood-borne organisms in the presence of local tissue factors which allow a nidus of infection to develop.

The review by Kaiser et al[4] of 23 patients with nontraumatic myonecrosis indicates its occurrence in 12 cases of adenocarcinoma of the colon, six cases of leukemia or lymphoproliferative disorders, and one case of idiopathic granulocytopenia, and in three benign disorders. The overall mortality in these cases was 78%; again, *Clostridium septicum* was the most common organism. The common denominator in all these cases appeared to be loss of mucosal integrity of the bowel from an ulcerated tumor or a perforation. Neutropenia was not mentioned as a possible etiologic factor in these cases, although one patient presented with granulocytopenia.

A report by Tikko and others[5] recounted an incident of nontraumatic myonecrosis arising in a patient with lymphoma and neutropenia, who survived after surgical treatment. Kapusta et al[6] reported a case of nontraumatic clostridial myonecrosis in a patient with cyclic neutropenia. Biswold and Harvey[7] in Australia reported an instance of necrotizing clostridial enterocolitis complicating cyclic neutropenia. Another report[8] detailed a case of necrotizing enterocolitis in a patient with chronic neutropenia characterized by cyclic fluctuations.

In the patient presented here, it seems possible that an inoculation via bee sting of clostridia from the patient's skin, combined with neutropenia, gave rise to a fulminant myonecrosis. However, it is also possible that this was a case of so-called nontraumatic infection associated with an occult carcinoma or the neutropenia alone. Whatever the pathogenesis of her infection, the patient's clinical course illustrated the acute surgical emergency this infection can precipitate, and the need for prompt diagnosis and treatment.

Dr Bottner: I think your hypothesis is perfectly plausible. It is impossible, however, without recent prior blood counts, to determine whether this woman was beginning to recover from the nadir of one of her leukopenic cycles, or was leukopenic because of overwhelming infection. The relative paucity of polymorphonuclear leukocytes and predominance of metamyelocytes suggests the former. I would have expected a bit more balance between the more mature and less mature forms in the instance of leukopenia as a result of overwhelming infection.

In patients with cyclic neutropenia there is evidence that marrow stem cell activity is cyclic in nature. I suspect that this woman's marrow was just starting to increase its production of myeloid elements at the time when she was stung by the bee. This increase in production, coupled with the stimulus of the infection, probably led to the extrusion of a large number of less mature forms from the marrow.

It is hard to judge the extent of impairment of this patient's phagocytic system. Her metamyelocytes probably had some phagocytic activity, though not as much as that of the bands or mature polymorphonuclear leukocytes. Metamyelocytes also do not migrate as well as more mature forms to sites of inflammation. If one counts only the polymorphonuclear leukocytes and bands, the absolute granulocyte count was quite low. That does not mean, however, that there were no mature granulocytes at the site of the infection. We know that circulating polymorphonuclear leukocytes are only in transit from bone marrow to sites of active inflammation, and that in neutropenic patients, they will migrate to sites of inflammation at times when the peripheral blood neutrophil count is quite low.

In spite of several early case reports, clostridial infections are not, in my experience, a common cause of sepsis in leukopenic patients, at least not in patients who are receiving chemotherapy for cancer or other conditions. We also know that this woman never previously had trouble with infections, and that individuals who are otherwise felt to be immunocompetent fall victim to clostridial infections. That makes it difficult for me to attribute the entire clinical picture to an underlying leukopenic disorder. It is definitely possible, but there is no way to know for sure.

My only other comment relates to the drop in her hematocrit from 48% to 21% during a 24-hour period. Improved hydration certainly contributed to the drop, but the patient lost the equivalent of six or seven units of blood in that 24-hour period, even though surgical bleeding was not substantial. I would suspect that she had clostridium-induced hemolysis, which might have been evident if pink serum were noticed during the drawing of blood samples, or if hemoglobinuria was observed. Clostridium-induced hemolysis tends to be a very rapidly progressive syndrome and is caused by clostridial toxins which dissolve the red blood cell membrane. I would guess that this played a role here.

Edward Coleman, MD (Chief Surgical Resident): The patient may have had hemoglobinuria; her urine was three-plus positive for blood. A urine blood-dipstick test, which detects hemoglobin or myoglobin, was positive. Serum and urine myoglobin levels were shown to be markedly elevated. Unfortunately, serum and urine hemoglobin levels were not measured.

David Blumenstock, MD (Attending Surgeon): The other aspect of the lowered hematocrit is that the patient probably had massive intravascular coagulation in the areas of clostridium myonecrosis, since there were clots extruding everywhere. When you consider just loss of that clotted blood from the circulation, it might explain the hematocrit drop.

Dr Bottner: The fragmentation from disseminated intravascular coagulation (DIC) could certainly account for some of her hemolysis. And she had some evidence of DIC in her prothrombin and activated partial thromboplastin time values. We do not have any other parameters to help us further with that diagnosis, but hemoglobinuria would be very unusual in hemolysis from DIC alone. I am quite sure that the infection itself played some role in the change of hematocrit.

CLINICAL DIAGNOSES

Clostridial myonecrosis.
Septic shock.
Disseminated intravascular coagulation.
Cyclic neutropenia.

PATHOLOGY PRESENTATION

Harlan D. Alpern, MD (Pathologist-in-Chief): The peripheral blood at the time of transfer demonstrated a low normal white blood cell count of 5,000/μL; the differential count included only 3% segmented neutrophils, 11% band forms, 24% metamyelocytes, and 6% myelocytes. As Dr Bottner indicated, this neutropenia could be due to the infection, cyclic neutropenia, or both. While the thrombocytopenia of 68,000/μL could have been due to the cyclic hematologic production disorder or to a direct effect of the clostridial toxins, the prolonged PT and APTT suggest a consumption coagulopathy due to extensive intravascular clotting in the resected necrotic muscle.

The surgical specimen consisted of the soft tissues of the left thigh and left hip. Histologic examination revealed a profound necrotizing myonecrosis. Myofiber nuclei were absent, and the sarcoplasm was hyalinized with loss of cross-striations (Fig 2). There was focal evidence of inflammation in the fascia, but no inflammation in the muscle tissue itself, which is typical of severe acute clostridium myonecrosis. The process represents a toxic muscular necrosis into which neutrophils are unable to penetrate. The clostridia produce at least 12 immunologically different powerful exotoxins, which are tissue necrosins and hemolysins.

There was bullous detachment of the epidermis at its base from the underlying dermis. The fluid that was obtained from the bullae demonstrated gram-positive rods on smear, and cultures grew *Clostridium septicum.* This soil organism, a normal inhabitant of the human intestine, is a less frequent cause of gas gangrene than is *Clostridium perfringens.* At autopsy, virtually all of the soft tissues of the left leg and the hip had been surgically excised. These tissues were histologically examined and were found to be viable at the surgical margins.

The right ventricle of the heart was markedly dilated, and there were focal areas of pallor in both ventricles. In the posterior septum, there was a scar representing an old

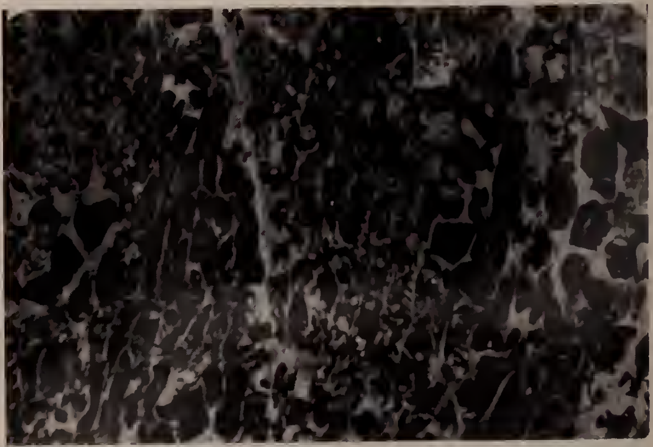

FIGURE 2. Debrided skeletal muscle of left leg, demonstrating necrotizing myonecrosis. Muscle nuclei absent; sarcoplasm hyalinized with loss of cross-striations (hematoxylin-eosin stain; original magnification × 100).

healed myocardial infarction. Examination of the coronary arteries demonstrated severe atherosclerosis, especially of the right coronary artery, with only a pinpoint lumen. The pathology sections demonstrated a marked thickening of the intima, with fibrosis and with focal calcifications. A very recent myocardial infarction was found, involving the left and the right ventricles. Some myofibers had lost their nuclei, and there were cytoplasmic eosinophilia and loss of cross-striations. Neutrophilic infiltration was not seen. These are the changes seen within the period of 12 to 24 hours after an acute myocardial infarction. The patient sustained a cardiac arrest during induction of anesthesia. It is most likely that the myocardial infarction occurred at that time.

The lungs were congested and edematous, secondary to acute heart failure. The bone marrow was cellular, with evidence of a shift toward immaturity in the myeloid forms consistent with the peripheral blood findings. In the stomach there were multiple small ulcers, and a granulation-tissue inflammatory pseudopolyp was present within the cecum. The liver demonstrated marked fatty change. There was atherosclerosis of the abdominal aorta. In the right temporal lobe of the brain, there was a recent infarction, 4 mm in diameter, compatible with an embolic episode. In the dura and attached to the skull was a meningioma, 7 mm in diameter, which contained psammoma bodies.

The patient had cyclic neutropenia, a most uncommon entity, the pathogenesis of which is believed to be periodic failure of pluripotential stem cells in the bone marrow possibly related to normal feedback mechanisms of the granulocytic series. This condition was first reported in 1910 and was recognized as an entity in 1930. It is an autosomal dominant disorder with variable expression. Most frequently, it presents in infancy or childhood, but it may present at any age. It was noted in this patient at least since 1933.

The interval at which the cyclic neutropenia occurs is variable. The periodicity is approximately every 21 days, with ranges from 15 to 35 days. Typically, there is a three- to four-day period of profound neutropenia and fever, which may be associated with stomatitis, pharyngitis, cervical adenitis, dermatitis, rectal and vaginal ulcerations, upper respiratory tract infections, and abdominal pain. The severity of these infections and the symptoms parallels the degree of neutropenia. Patients who escape infection during profound leukopenia almost invariably have a reactive monocytosis.

During the leukopenic phase, neutrophils may disappear completely from the peripheral blood, and there is often a compensatory monocytosis; reticulocytes and platelets usually are also decreased. The bone marrow demonstrates a myeloid aplasia or maturation arrest at the myelocytic phase. Between these leukopenic intervals, the patients are in good health, with normal blood counts; the spleen is often enlarged. In many patients, the disease becomes milder with time and may even disappear. Some mild cases are never clinically recognized.

In terms of therapy, normal plasma has been used in some patients to increase their blood counts; 35–50% of patients may benefit from splenectomy. There are reports of benefit from treatment with normal plasma infusion, testosterone, adrenocorticotropic hormone, and glucocorticoids.

In summary, the postmortem examination demonstrated that the patient died of an acute biventricular myocardial infarction as a result of marked coronary atherosclerosis. The infarction led to marked pulmonary edema and right ventricular dilation. This infarction occurred in a setting of *Clostridium septicum* gangrenous myonecrosis, also referred to as "gas gangrene," involving the left leg and hip. The patient underwent extensive surgical soft-tissue debridement and suffered a cardiac arrest during anesthesia induction for the procedure. The myocardial infarction probably occurred at that time.

The preexisting condition of cyclic neutropenia, dating back to 1933, could have predisposed the patient to the development of the *Clostridium septicum* gangrenous myonecrosis. There is a history of an insect sting on the left knee several days prior to admission, which may have been the source of the clostridium infection, although the intestine could have been the source.

Dr Zulick: Was the lesion in the patient's cecum ulcerated?

Dr Alpern: Yes, it was. The mass was ulcerated granulation tissue. It could have developed secondary to ischemia.

Dr Zulick: That fits in with the series reported in the journal *Cancer*,[4] of patients who had nontraumatic myonecrosis; the common denominator was thought to be the loss of mucosal integrity of the bowel.

Roger MacMillan, MD (Attending Surgeon): I have treated three of these patients in the past, and two have died. One was a 16-year-old boy who had an underlying aplastic anemia. In emphasis of Dr Zulick's point, this patient's initial symptom while at Babies Hospital in New York was the sudden onset of terrific pain in the calves for no apparent reason, associated with phenomenal tenderness. He then went on to develop subcutaneous emphysema, underwent emergency surgery, and the next morning was transferred to Mt Sinai Hospital for treatment in the hyperbaric oxygen chamber, but he ultimately died of an overwhelming infection. Another patient had a below-the-knee amputation—again, sudden onset of exquisite pain and subcutaneous emphysema. Ultimately he recovered. The last case I was involved with, during my residency here, was a patient similar to one with emphysematous cholecystitis, in whom subcutaneous emphysema developed on the lateral aspects of the abdominal wall. The sudden onset of pain was very impressive, and yet the other symptoms were not particularly impressive initially.

Patrick Dietz, MD (Attending Vascular Surgeon): This patient was referred for what was thought to be a vascular problem, either arterial or venous in origin; that question was uncertain. She had had radiographs of both her legs and abdomen at the referring hospital, and they were unremarkable. On arrival, she had a tense, edematous, very painful thigh, so that part of the initial differential diagnosis was the possibility of phlegmasia cerulea dolens (acute venous thrombosis). Her laboratory data on admission reflected dehydration, in that her hematocrit rose from 36% at the referring hospital in the morning, to 48% at this hospital in the afternoon. This was undoubtedly due, at least in part, to fluid loss into edema formation in the soft tissues.

Clinically, there was no palpable gas and no crepitus at the time of the initial examination; shortly thereafter on subsequent examination there was a suggestion of gas, and thereafter, another area where one could feel crepitus. This was recorded by the radiographs which showed a tremendous amount of gas that had collected by that time.

The bee sting was in the area of the left knee, and the initial presentation of all of the patient's pain and swelling was in the more proximal thigh. The possibility of a direct inoculum from the bee sting wound still remains questionable to me. Then again, there may be other sources of the inoculum, as was mentioned.

Dr Blumenstock: As was emphasized in the very beginning, it is not unusual to have patients referred with a vascular problem who do not have a vascular problem. I mentioned here not too long ago a patient with acute meningococcemia who presented on an athletic field with a "thrombosed aorta."

Furthermore, myocardial infarction does indeed explain the pulmonary edema. We are becoming more and more aware of the role of tissue and circulating fibronectin in the etiology of adult-onset respiratory insufficiency, in changing the permeability of the pulmonary capillary membranes, thus leading to the extravasation of fluid into the lungs. Fibronectin is a cold-precipitable glycoprotein, produced by the liver, with a very short half-life of 24 hours. This probably plays a considerable role in the edema. This substance is currently under investigative study.

Michael Gold, MD (Chief of Surgery): Had the patient not had that myocardial infarction, your findings at the margins of the debrided tissue indicate that she might have survived.

Dr Alpern: That is possible. But of course the mortality in this disorder is very high.

Dr Gold: I would like to ask Dr Zulick if in his review of this subject he came on any data that would strongly support the use of a hyperbaric chamber. When I was in New York City, we could refer all such patients to the hyperbaric unit at Mt Sinai Hospital.

Dr Zulick: Every article mentioned the possibility of hyperbaric chamber treatment, and it seemed that there were some cases at least that had dramatic response to that treatment. Some patients actually were not even treated surgically, but only with the hyperbaric oxygen chamber. But this is by no means useful in every case. Another nonsurgical treatment used in the past was clostridial antitoxin. However, it proved ineffective and is no longer used. Prompt antibiotic therapy with penicillin G or clindamycin, on the other hand, appears to be crucial in reducing mortality, when combined with surgical therapy.

John Olson, MD (Attending Surgeon): I thought that the big caveat here is, never replace surgery with the hyperbaric chamber.

Dr Gold: I think that is correct. My one case experience was a young and otherwise healthy woman who died rather precipitously following an elective cholecystectomy. I think the role of the hyperbaric chamber may possibly be in some prevention of the extension of the gangrene. But you are correct that it should never replace aggressive surgical management. I think we have to emphasize the word "aggressive."

Dr Blumenstock: Is the hyperbaric chamber still functioning at Mt Sinai?

Dr Gold: It was as of the time that I left New York City. Are there others in New York State?

Dr Zulick: Yes. There is one multiseat chamber at Mt Sinai Hospital in New York City, and two multiseat chambers in City Island, NY, at the North American Hyperbaric Center. There are also two multiseat chambers at the Millard Fillmore Hospital in Buffalo.

FINAL DIAGNOSES

Atherosclerotic cardiovascular disease. Dilation of right ventricle. Marked atherosclerosis, right coronary artery. Acute myocardial infarction, biventricular. Old, healed myocardial infarct, posterior septum. Congestive heart failure, with pulmonary edema, marked.

Clostridium septicum gangrenous myonecrosis.

Status post extensive soft tissue debridement, left lower leg and left hip area.

Cyclic neutropenia. Ulcerated inflammatory polyp, cecum. Meningioma, dura, inner table of skull.

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Acute massive rectal bleeding as a presenting sign of Crohn disease

JAMES SMITH-BEHN, MD; ARMIN BANEZ, MD; THOMAS BROWN, MD; RENATO SIMON, MD; PAUL LIN, MD

While many patients with Crohn disease have evidence of gastrointestinal bleeding at some point in their disease, the majority of bleeding episodes are minor. Massive bleeding does occur in 5% of patients,[1] and there is evidence that the number of patients with this complication is increasing. We report the case of a patient with massive rectal bleeding as the initial manifestation of Crohn disease of the ileum.

CASE REPORT

A 59-year-old man presented to the Northside Medical Center emergency room complaining of the sudden onset of bloody stools associated with dizziness. He denied nausea, vomiting, abdominal pain, diarrhea, fever, or chills. He was on no medications. The medical history was significant for two episodes of upper gastrointestinal bleeding secondary to Mallory-Weiss tears which required operative intervention. On physical examination, the patient was hypotensive and tachycardic, and had tenderness in the right lower quadrant with a suggestion of a mass. The rest of the examination was unremarkable. Prothrombin and partial thromboplastin times and the platelet count were normal. Sigmoidoscopy to 35 cm showed abundant clots but no source of bleeding. A selective superior mesenteric arteriogram showed active bleeding in the mid-ileal region (Fig 1). The patient was transferred to the intensive care unit and was supported with intravenous fluids and blood. Several hours later he re-bled and it was thought that surgical exploration was warranted. At laparotomy a segment of mid-ileum was significantly thickened and exhibited fat wrapping. Mesenteric adenopathy was also present. A 20-inch segment of diseased mid-ileum was resected, which corresponded to the area of bleeding seen on arteriogram. To confirm removal of the bleeding site, a specimen arteriogram was performed (Fig 2). A primary anastomosis was then carried out. On opening the specimen, the mucosa was found to have a decrease in the number of folds. Multiple areas of mucosal ulcerations containing clots were present. On histologic examination, transmural inflammation as well as lymphoid aggregates were present. The mucosa showed increased numbers of chronic inflammatory cells with focal ulceration, all highly consistent with Crohn disease. However, no definitive sinus fissures or granulomata were seen. The patient made an uneventful recovery and was discharged on the seventh postoperative day. Two years later, he has had no further episodes of gastrointestinal bleeding. Symptomatic relapses of his disease have been treated with prednisone.

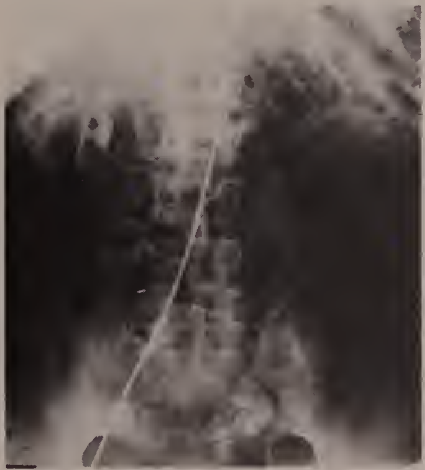

FIGURE 1. Selective superior mesenteric arteriogram demonstrating contrast extravasation from the mid-ileum to the terminal ileum.

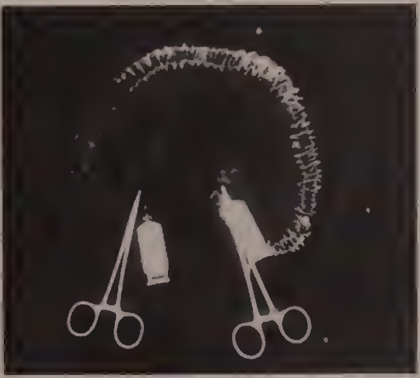

FIGURE 2. Specimen arteriogram showing extravasation of the contrast material into the lumen of the bowel.

DISCUSSION

Some degree of occult gastrointestinal blood loss occurs in most patients with Crohn disease.[2] However, rectal bleeding was formerly thought to be an extremely rare complication. It occurred in only two of Crohn's original 222 patients.[3] However, more recent reports indicate an increase in the number of patients with Crohn disease and massive lower gastrointestinal hemorrhage. In the past ten years, studies show an incidence of between 1.4% and 13%.[4-6] Schwartz[1] claims that massive bleeding occurs in 5% of patients with Crohn disease. In approximately half of the reported cases, the massive bleeding episode was the initial manifestation of Crohn disease.

Angiographically, Crohn disease can be divided into an early and a late stage.[4] The early stage is characterized by dilated tortuous vessels and mucosal hyperemia; the late stage by a decrease in the caliber of the vessels and lack of tapering, suggesting fibrosis. Most episodes of bleeding would be expected to occur in patients in the early stages of their disease. In the series of Rubin et al[5] and Dent et al,[7] the majority of bleeding episodes occurred in patients in the initial stages of their disease. On the average, patients who bled massively were also younger than other patients with Crohn disease (18.6 years vs 27

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years of age). However, a similar series presented by Mesleh and Lemons[8] included patients at both ends of the disease spectrum, and they concluded that there is no relationship between the duration of Crohn disease and the incidence of massive bleeding. The reports of Homan et al[4] and McGarrity et al[9] also suggest that there is no relationship between age, duration of the disease, and the risk of bleeding.

The bleeding usually originates from eroded vessels at the base of an ulcer. Asakura et al[10] have shown abrupt interruptions in the vasa recta of the small bowel submucosa in patients with Crohn disease who have bled, indicating that the bleeding originated from those vessels. The site of bleeding corresponds to the area of intestine affected by Crohn disease. Sixty-five percent of patients bled from the small bowel, 12% from the colon, and 23% from an undetermined site (small bowel or colon).[4]

Conservative treatment is usually not warranted, as most patients will have persistent or recurrent massive bleeding.[5,11] There is one report in the literature on the use of vasopressin infusion to stop bleeding, however, it represents an isolated case.[12]

The length of bowel resected must be individualized according to the degree of disease present. Ideally, all affected bowel should be removed, as failure to do so may result in re-bleeding.[4] In patients with extensive disease, the risk of re-bleeding must be weighed against the possibility of creating a short bowel syndrome due to extensive resection of small bowel. In these patients, it is acceptable to resect only the bleeding area. A specimen arteriogram will help to document removal of the bleeding site.

To our knowledge the patient described here is the first in whom the bleeding site in the small bowel was angiographically documented both pre- and postoperatively. Initially the patient was managed conservatively. However, when this failed, operative intervention was required. The arteriogram of the resected specimen documented removal of the bleeding site.

Massive gastrointestinal bleeding is occurring with increased frequency in patients with Crohn disease. While massive bleeding occurs mainly in young patients, patients at all stages of disease and at any age may have episodes of massive bleeding. Conservative therapy is usually unsuccessful. All affected bowel should be removed when feasible. Finally, a specimen arteriogram can help document removal of the bleeding site when only a partial resection of affected bowel is performed.

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See also pp 519–520.

Extramammary Paget disease of the inguinoscrotal area

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CONSTANTINE P. KARAKOUSIS, MD, PHD

Extramammary Paget disease is a rare clinical entity that is observed more frequently in areas where apocrine glands are present in high density, such as the anogenital region. It has also been reported in the axilla,[1] eyelid,[2,3] knee,[4] and in modified apocrine glands such as the ceruminous glands of the ear canal.[5]

Paget disease of the scrotal area must be differentiated from other dermatologic conditions arising in this region, including erythroplasia of Queyrat, Bowen disease, and malignant melanoma. Histologically, it is characterized by the presence of cells similar to those seen in mammary Paget disease, but its histogenesis and biological behavior are considered significantly different. In mammary Paget disease, there is direct extension into the epidermis of the underlying ductal carcinoma of the breast.[6] It is controversial whether extramammary Paget disease originates in the pluripotential cells of the epidermis or represents an extension from an underlying adenocarcinoma.

There are reports of extramammary Paget disease associated with other malignancies. We present three cases of scrotal extramammary Paget disease. Two were associated with secondary malignancies. The first patient later developed two malignant melanomas and also two squamous cell carcinomas. In the first two patients, extramammary Paget disease was initially mistakenly diagnosed as malignant melanoma.

CASE REPORTS

Case 1. A 55-year-old man noted a small lesion on the skin of his left scrotum in April 1973. A year later, during a left ingui-

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nal hernia repair, the lesion was completely excised. The biopsy was thought to be malignant melanoma, Clark's level II. Subsequently, the patient underwent wide excision of the site and removal of suspicious left groin lymph nodes, but the nodes were negative for tumor. He was then seen at Roswell Park Memorial Institute in September 1974 and had a metastatic workup, which included liver scan, chest radiograph, intravenous pyelogram, bone marrow examination, upper gastrointestinal series, and lymphangiogram. Results of these were all normal. On physical examination, the patient had multiple dysplastic nevi scattered on the trunk. A biopsy of another scrotal lesion in October 1974 showed dermatofibroma.

In May 1978, the patient developed a small rash around the reconstructed area of the scrotum and around the shaft of the penis, which on biopsy proved to be Paget disease. There was no evidence of malignant melanoma. On December 1978, he underwent wide excision of these areas and split thickness skin graft from the left thigh. Histologic examination showed nests and cords of uniform cells with granular cytoplasm containing mucin; the cytoplasm stained positive with muci-carmine and periodic acid-Schiff reagent. The margins were free and there was no underlying adenocarcinoma (Fig 1). The slides of 1973 from a lesion in the same area were reviewed and were now interpreted as Paget disease.

Two malignant melanomas subsequently developed; one on the abdominal wall (Clark's level IV, excised in May 1979) and the other on the patient's left upper arm (Clark's level II, excised in November 1981) (Figs 2, 3). These tumors were pigmented. The pigment was identified as melanin with special stain.

Additionally, two squamous cell carcinomas developed on the floor of the mouth, one in January 1981 and the other in October 1982. The second was invasive and required wide excision and suprahyoid lymph node dissection. At present, the patient is doing well nine years after excision of the extramammary Paget disease and five years after excision of the squamous cell carcinoma.

Case 2. A 73-year-old schizophrenic man was admitted to Roswell Park Memorial Institute on May 7, 1985, with a diagnosis of malignant melanoma of the skin of the pubis. He had approximately a three-month history of an ulcerated lesion involving the skin of the right groin and pubis for approximately three months. A biopsy performed at another institution in April 1975 had been reported as malignant melanoma.

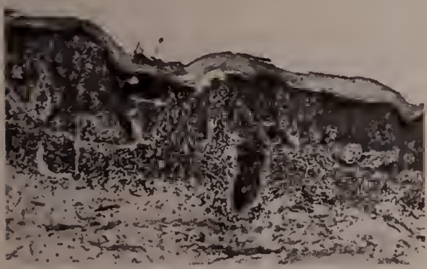

FIGURE 1. Skin showing Paget's disease. Arrows show Paget's cells.

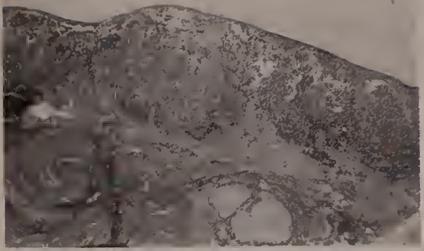

FIGURE 2. Malignant melanoma with foci of invasion of papillary dermis (level II) (hematoxylin-eosin stain, original magnification × 80).

On physical examination, a 9 × 9-cm superficial erosive lesion involving the skin of the right scrotal and inguinal areas was found. A review of the 1975 slides in the institute demonstrated that this was extramammary Paget disease. Repeat biopsy confirmed the diagnosis. A metastatic workup for a secondary malignancy was negative. The patient underwent wide excision of the skin of the scrotum and right groin on May 20, 1975. Pathologic examination demonstrated numerous Paget cells in the epidermis, which showed vacuolated cytoplasm positive with mucicarmine and periodic acid-Schiff reagent. With immunohistochemical stains (Fig 4), these cells were also positive for carcinoembryonic antigen (CEA) (Hybritech) and negative for prostate specific antigen and prostatic acid phosphatase (RPMI). Some Paget cells were seen involving the hair follicles, but there was no infiltration of the dermis, no underlying carcinoma, and the margins were free. The patient was subsequently transferred to a nursing home, where he died of metastatic adenocarcinoma of the prostate seven years later.

Case 3. A 72-year-old man was first seen in this institute in June 1982 because of a 15 × 1-cm eczematous lesion on the right side of the scrotum. This patient had a long history of Paget disease of the scrotal area, the original diagnosis having been made in 1973. Since then he had received a variety of treatments with creams and electrocoagulation and had multiple recurrences. He underwent local excision of the scrotal lesion which showed extramammary Paget disease (Fig 5). A few months later he had a wide excision, but the Pagetoid cells were still extending to one margin. In May 1984, recurrent disease was demonstrated on biopsy, and the patient underwent wide excision of the skin of the right groin and scrotum. Pathologic examination demonstrated Paget disease without underlying carcinoma or invasion of the dermis, and the margins were free. The patient is doing well three years after his last excision.

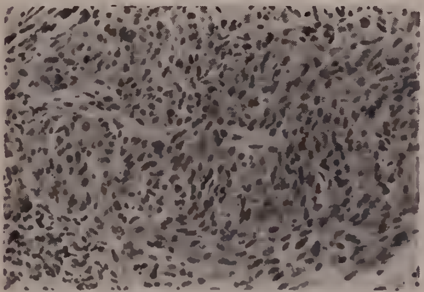

FIGURE 3. Higher magnification of the invasive area demonstrating considerable cellular atypia and mitotic activity justifies the diagnosis of melanoma (hematoxylin-eosin stain, original magnification × 480).

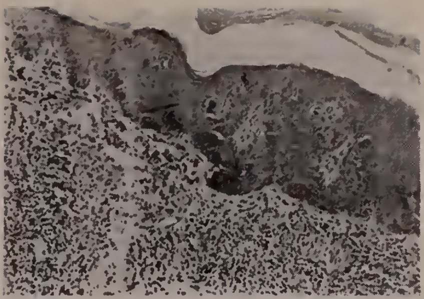

FIGURE 4. Carcinoembryonic antigen positivity in the intraepidermal neoplastic cells (immunohistochemical stain).

DISCUSSION

Extramammary Paget disease is a relatively uncommon disease that occurs predominantly in the older age group. The perianal and genital areas, especially the vulva, seem to account for most cases[7,8]; however, it has been reported in the tongue[9] and esophagus.[10] Clinically, extramammary Paget disease is characterized by a progressive erythematous eczematoid skin lesion associated with pruritus. The diagnosis is made by biopsy. Histologically, the lesion is characterized by intraepidermal nests of round cells with clear to pale cytoplasm, large nuclei, prominent nucleoli, and increased nuclear/cytoplasmic ratios. The cytoplasm of these cells stains positive with periodic acid-Schiff reagent (PAS) and mucicarmine and alcian blue, which is helpful in differentiating them from malignant melanoma and Bowen disease.

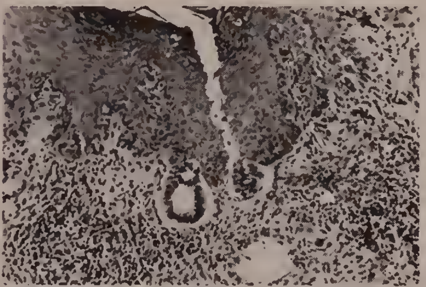

FIGURE 5. A higher magnification showing actual gland formation of Paget disease.

Cases 1 and 2 illustrate the fact that extramammary Paget disease may be confused histologically with malignant melanoma. The original diagnosis in these patients was malignant melanoma, but a careful review of the slides and subsequent excision demonstrated that the lesions were extramammary Paget disease instead. Immunohistochemistry utilizing CEA is helpful in differentiating Paget disease from melanoma and Bowen disease.[11]

A controversial issue in regard to this entity is the origin of the Paget cells. There are two opposing theories. One postulates that these cells originate in an adjacent adenocarcinoma of the sweat glands and then migrate to the epidermis. This seems to be a good explanation for mammary Paget disease where there is underlying intraductal carcinoma. The other theory postulates that these cells originate from the pluripotential cells in the epidermis as a response to a presumptive carcinogenic stimulus. This theory seems more applicable to extramammary Paget disease, where the presence of an underlying carcinoma is relatively rare. The three patients reported here did not show underlying sweat gland adenocarcinoma. The involvement of squamous epithelium of the hair follicles, such as was present in case 2, does not represent an underlying neoplasm.[12] This type of involvement could be compared with squamous cell carcinoma in situ of the uterine cervix in which not only surface epithelium but also endocervical glands are involved in the process.

The treatment of extramammary Paget disease is clearly surgical. For localized disease wide excision with free margins and, in some cases where the margins are positive, reexcision until the margins are free, should be the treatment of choice. When there is invasion or underlying adenocarcinoma, the treatment should be directed to the malignancy.

Radiotherapy has been of little benefit in the treatment of this disease regardless of the stage,[7] but 5-fluorouracil used topically has been of some benefit, especially in patients who need local palliation.[13,14]

Local recurrence is frequent after seemingly adequate surgical excision. This may be explained in part by the fact that the histologic involvement is far greater than that of the grossly visible lesion[15] or by the fact that the lesions tend to be multicentric. The margins of the specimens in two of our patients were free, and neither had local recurrence; but the third patient had positive margins and hence had recurrence.

The presence of associated malignancies in patients with extramammary Paget disease is well recognized. Koss et al[8] reported it to be as high as 50%, and in the review of seven series by Lee et al[16] it was 32.4%. Two of our patients presented associated malignancies; patient 2 died as a consequence of metastatic carcinoma of the prostate and patient 1 had multiple malignancies. Other dermatologic conditions are also frequently associated with separate and unrelated cancer. Acanthosis nigricans, erythema gyratum repens, and exfoliative dermatitis are good examples.[17] Therefore, the necessity of a search for secondary malignancies in patients with extramammary Paget disease and close follow-up is emphasized.

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Brain abscess: A complication of cocaine inhalation

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The epidemic of recreational cocaine abuse has led to a host of adverse health consequences. Frontal sinusitis secondary to nasal inhalation of cocaine is well recognized,[1] as is the relationship of frontal sinusitis and brain abscess.[2] This report describes a patient whose clinical and pathologic findings relate cocaine "snorting" to the sequential development of frontal sinusitis and a lethal brain abscess.

CASE REPORT

A 31-year-old man was found by paramedics in a stuporous condition on a subway platform. When evaluated in the emergency department at New York Infirmary-Beekman Downtown Hospital, he was disoriented and unable to respond to questions. A medical history obtained from family members revealed that the patient had complained of a "band-like" headache during the preceding two weeks. The additional medical history was remarkable only for recreational cocaine abuse ("snorting").

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During his two-hour stay in the emergency department he gradually regained his orientation; however, he was slow to answer questions.

Physical examination initially revealed a man in no acute physical distress. His temperature was 101°F (38°C). The pupils were equal and reactive, and the optic disc margins were sharp. There was no neck stiffness. Examination of the heart and lungs was unremarkable. A neurologic examination was significant for a right facial palsy. There were no other motor or sensory deficits. Deep tendon reflexes were equal and intact. He had an extensor plantar reflex on the left side. The patient's mental state was unchanged following intravenous naloxone, and he was admitted to the inpatient service.

The white blood cell count was 12,400 with six immature polymorphonuclear leukocytes. Blood cultures were negative. Chest films and other routine laboratory studies were normal. Skull radiographs were negative for fractures.

On the first hospital day the patient continued to complain of frontal headaches and his temperature stayed at 101°F. The neurologic examination revealed a disappearance of the facial palsy. An initial computed tomographic (CT) scan of the head was negative. Lumbar puncture revealed clear cerebrospinal fluid with 274 red blood cells and 24 white blood cells per microliter (60% polymorphonuclear leukocytes, 40% lymphocytes), a protein level of 106 mg/dL, and a glucose level of 66 mg/dL. Bacterial cultures and stains of the cerebrospinal fluid were negative.

On the second hospital day the patient continued to be febrile with intense frontal headaches, and the neurologic examination was unchanged. An electroencephalogram performed on this day showed only a normal awake pattern. On the third hospital day he complained of worsening headaches and continued to be febrile. That night he had a grand mal seizure and was subsequently maintained on 300 mg of dilantin per day. He remained free of seizures for the rest of his hospital stay.

The next day (the fourth hospital day), a repeat lumbar puncture was performed. The cerebrospinal fluid remained clear with 2,560 red blood cells and 189 white blood cells per microliter (70% polymorphonuclear leukocytes, 28% lymphocytes), a protein level of 37 mg/dL, and a glucose level of 78 mg/dL. Treatment was started with intravenous penicillin, 4.8 million units every six hours, and acyclovir, 400 mg, every eight hours. The patient remained febrile to 102°F. Other studies of his cerebrospinal fluid, including bacterial counterimmunoelectrophoresis, stains, and cultures remained negative. He now complained of frontal sinus pain with tenderness to palpation. Intravenous penicillin was changed to intravenous ampicillin, 2 g every four hours.

On the fifth hospital day the patient continued to have a fever, his headaches lessened, and he remained oriented. Therapy with intravenous chloramphenicol, 1 g every six hours, was begun. Sinus radiographs taken on this day revealed opacification of both frontal sinuses and the left maxillary sinus. On the sixth hospital day the patient refused repeat lumbar puncture. His temperature rose to 102°F; chloramphenicol was discontinued and cefotaxime, 2 g every four hours, was begun. All cultures, of both blood and cerebrospinal fluid, remained negative. On the seventh and eighth hospital days the patient became more lethargic, stopped eating, and again refused a repeat lumbar puncture. He remained febrile to 101°F.

On the ninth hospital day, a third lumbar puncture revealed clear cerebrospinal fluid with 2,100 red blood cells and 250 white blood cells per microliter (95% polymorphonuclear leukocytes, 5% lymphocytes), a protein level of 53 mg/dL; and a glucose level of 81 mg/dL. Later during the night, the patient was found unconscious in bed. His pupils were fixed and dilated. Endotracheal intubation and ventilatory support became necessary. A repeat CT scan of the brain revealed a lucency in the left frontal lobe of the brain, with a shift of the right falx cerebri. A repeat electroencephalogram showed no electrical activity. The patient died on the 13th hospital day.

An autopsy revealed acute left frontal sinusitis (Fig 1), together with an acute abscess of the left frontal lobe of the brain (Fig 2), and diffuse cerebral edema. Other findings included bilateral pulmonary congestion and edema, pleural effusions, atelectasis, and bronchopneumonia.

DISCUSSION

The centuries-old recreational use of cocaine has involved the chewing of the *Erythroxin coca* leaves by the Amara Indians in the Andes, as well as intravenous injection and nasal inhalation by individuals from all segments of modern society. Some 22 million Americans have "tried" cocaine. It is estimated that four million people use it at least once per month.[1]

The hydrochloride moiety of the molecule renders it water soluble but topically irritating, with an acidic pH of 4.0.[1] Inhalation of pure, or more often adulterated, cocaine, may cause mucoperichondrial ischemia that can lead to necrosis and infection (sinusitis) with adjacent osteitis. Nasal and paranasal sinus symptoms, when otherwise unexplained, should arouse suspicion of cocaine inhalation.

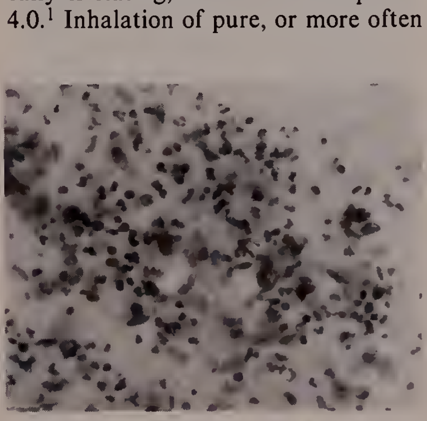

FIGURE 1. Smear of frontal sinus showing purulent inflammation (hematoxylin-eosin stain; original magnification × 450.)

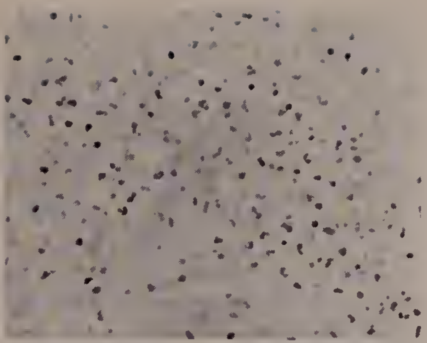

FIGURE 2. Section of brain showing abscess formation (hematoxylin-eosin stain; original magnification × 450.)

Brain abscess can be a sequel of ear and sinus infections,[2] especially when the frontal sinuses are involved.[3] The spread of infection from the sinuses to the brain substance may occur directly through areas of osteitis, or as a septic thrombophlebitis along the valveless venous plexi which connect the intracranial veins with those of the sinus mucosa.[4] Typically, a cerebral infection is associated with local necrosis and abscess formation.

The high death rate due to brain abscess is the result of a combination of factors: failure to locate the abscess, a virulent infecting organism, an immunocompromised host, multiple abscess formation, ineffective antibiotic or other medical therapy, and inadequate surgical treatment.[5]

In the patient presented here, the negative initial CT scan of the brain led to an underestimation of the seriousness of the lesion. The rapidly expanding cerebral abscess with surrounding edema caused fatal compression of the midbrain and brainstem. Given the ease of availability and widespread abuse of cocaine, sinus pathology and intracranial symptoms should alert physicians to the possibility of brain abscess.

Acknowledgment. The author thanks Dr Edward Weiner for the preparation of the pathology illustrations.

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Endobronchial lipoma

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Endobronchial lipoma behaves like many other benign endobronchial tumors, with symptoms that are due either to recurrent infection behind the obstructing lesion or to hemoptysis. The principal value of this report is that it reminds physicians of the existence of benign endobronchial lesions, and that when physicians detect these lesions early enough, they can prevent much morbidity. One can achieve early diagnosis by bronchoscopic biopsy and complete cure by bronchoscopic resection. Delayed diagnosis can lead to destructive distal lung disease necessitating an extensive surgical procedure, as occurred in the patient described here.

CASE REPORT

A 45-year-old man presented with the chief complaint of recurrent pneumonia for five years. He was well developed and well oriented. He had had two recent episodes of hemoptysis and a 20-pound weight loss in the previous five months. He denied shortness of breath and night sweats. He had smoked a pack of cigarettes per day for 30 years. The physical examination was unremarkable except for diminished air entry over the right lower lobe and basal rales. Routine hematologic and chemistry studies were within normal limits. Pulmonary function tests revealed mild airway obstruction. Chest roentgenograms showed persistent pneumonic infiltrates (Fig 1) in the hilar area and atelectasis of the right middle lobe (Fig 2). A computed axial tomographic (CAT) scan confirmed right middle lobe atelectasis. Flexible bronchoscopy revealed a pearly white endobronchial lesion in the right middle lobe. The biopsy was reported as submucosal fibrosis (Fig 3). An open thoracotomy disclosed a mass at the right perihilar region and atelectasis of the right middle lobe. The right middle lobe was firmly adherent to the right upper lobe, right lower lobe, and pericardium by inflammatory reactions. All three right lobes of the lung were damaged and firmly adherent to one another; hence, pneumonectomy was performed.

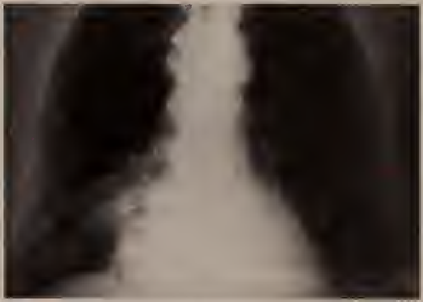
FIGURE 1. Chest roentgenogram, anteroposterior view, showing pneumonic infiltrate by the hilum of the right middle lobe.

On pathologic examination the right lung weighed 470 g. The right middle lobe showed an obstructing, endobronchial, polypoid mass 2 cm in diameter in the main bronchus. There were bronchiectasis, abscesses, and organizing pneumonia. The endobronchial mass had a white peripheral zone and a large pale yellowish central area. On light microscopy, the central area consisted of mature fat cells enclosed by a dense fibrous capsule (Fig 4). The mucosa directly lined the fibrous capsule. The right upper lobe and right lower lobe of the lung showed patchy, organized pneumonia and bronchiectasis with dense fibroadhesive pleuritis. The diagnosis was polypoid endobronchial lipoma. The patient recovered and was discharged and followed in the chest clinic.

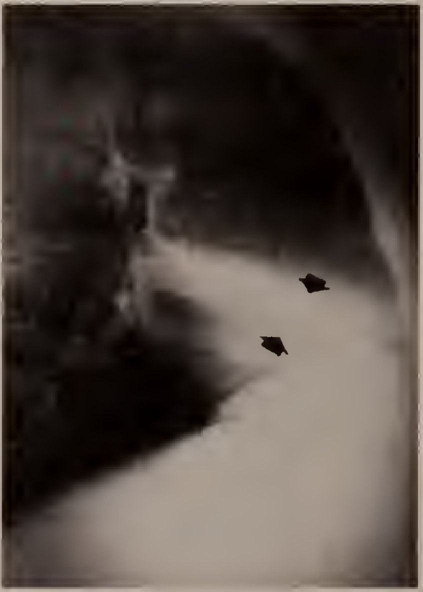
FIGURE 2. Chest roentgenogram, lateral view, showing pneumonic infiltrate and atelectasis (arrows) of the right middle lobe.

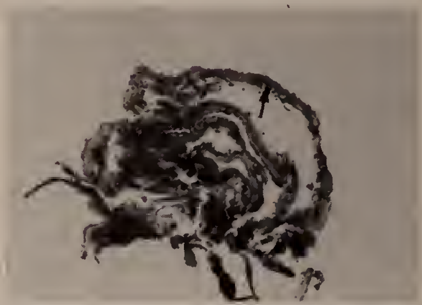
FIGURE 3. Flexible bronchoscopic biopsy showing bronchial mucosa, basement membrane and lipoma's fibrous capsule (arrow) (hematoxylin-eosin stain; original magnification × 40).

DISCUSSION

Lipomas of the bronchus are extremely rare.[1] Approximately 54 cases

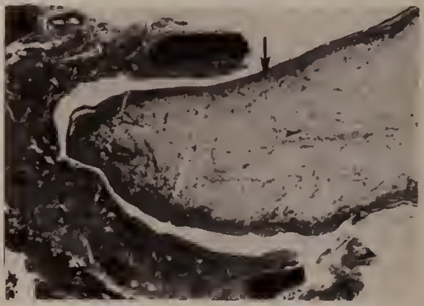
FIGURE 4. Endobronchial lipoma, polypoid, obstructing the bronchial lumen. It consists mostly of mature fat cells surrounded by fibrous capsule (arrow) and lining mucosa (hematoxylin-eosin stain; original magnification × 10).

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have been reported in the English literature. Hurt,[2] in 1984, reviewed 128 benign tumors of the bronchus reported in the literature and listed 74 carcinoids, 38 hamartomas, 7 chondromas, 3 lipomas, and other types.

Endobronchial lipomas originate from the adipose tissue normally present beneath the submucosa, and at times behind the cartilage of the bronchus.[3] They are mostly pedunculated, occasionally sessile, and rarely dumbbell-shaped.[4] Lipomas have a gray-yellow color which results from the fibrous capsule. The central fat rarely develops cartilage or bone resembling hamartomas.[5] Lipomas can produce bronchial obstruction of varying degree. In neglected cases, recurring pneumonias, atelectasis, irreversible bronchiectases, and parenchymal damage usually develop in the distal lung.[1,4,6]

Endobronchial lipomas are often encountered in adults of middle age, and there is a striking preponderance in men. The symptoms are those associated with progressive bronchial obstruction.[7] The duration of symptoms before diagnosis ranges from a few months to several years.[6,8] The most common symptoms are cough, recurrent fever, hemoptysis, dyspnea on exertion, and wheeze. Cough is observed in 85% of reported cases and wheeze in 23%. The cough is usually productive of sputum. Fever occurs from repeated episodes of pneumonic infection. Hemoptysis evolves late in the disease and, in the great majority of patients, directly relates to the destructive distal lung disease rather than to the lipoma. Wheeze develops when the obstruction of the bronchus is partial; it disappears once the obstruction becomes complete. From the symptoms, the differential diagnosis can include bronchial asthma, tuberculosis, bronchial adenoma, and bronchogenic carcinoma.[8] Worthy of note is the significant weight loss that is observed in 8% of patients with endobronchial lipoma. This is a common phenomenon in tuberculosis and malignancy. The 20-pound weight loss in the patient reported here could be attributed to recurrent chest infections.

The diagnosis can often be suspected or established by bronchoscopic biopsy. Most endobronchial lipomas are in the main bronchus.[8] Sometimes, as in the patient described here, biopsy by flexible bronchoscope is too superficial, including only the mucosa and peripheral capsule of the lipoma (Fig 3).[9,10] This does not establish the diagnosis of lipoma, much less preclude deep submucosal tumors such as adenomas or carcinoma. In such cases, the diagnosis can only be established by bronchotomy or lobectomy. Radiologic findings of endobronchial lipomas are nonspecific. Fat tissue cannot be delineated in spite of the bordering air in the lung. Atelectasis, recurring pneumonias, and eventually bronchiectasis often develop and conceal obstructing lesions of the bronchus.[8] Mendelsohn et al[11] reported the first endobronchial lipoma diagnosed by CAT scan.

The treatment of endobronchial lipoma consists of bronchoscopic excision (20%), bronchotomy (3%), lobectomy (57%), and pneumonectomy (23%).[1,2,10,12] In each case, the size and character of the lesion and the presence of irreversible peripheral lung changes must be assessed. Bronchoscopic excision usually succeeds in small, pedunculated early lesions. An awareness of an obstructing benign lesion can improve the cure rate by endoscopic excision. Undiagnosed broad-based lesions may necessitate bronchotomy and frozen section. Spinelli et al[9] resected a sessile lesion three times by bronchoscopic procedure at weekly intervals. Smirniotopoulos et al[12] reported an endobronchial lipoma that was removed by neodymium-YAG laser through a fiberoptic bronchoscope. When the lung distal to the endobronchial tumor is damaged (bronchiectasis or persistent atelectasis) due to obstruction, segmentectomy, lobectomy, or pneumonectomy may become necessary.[6]

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Cancer of the male breast

TO THE EDITOR: I respectfully take issue with some of the statements made with regard to cancer of the male breast in the commentary by Dr Kenneth Ouriel in the June 1988 issue of the *Journal.*[1]

It is quite true that no lobules are formed in the normal male breast or seen in most cases of gynecomastia studied microscopically. However, lobule formation may occasionally be seen in estrogen-induced gynecomastia of men treated for prostatic cancer.[2] Additionally, Dr Ouriel states that male breast cancer differs from that in the female histopathologically by the absence of the lobular variety. In fact, several well-documented cases of infiltrating lobular breast carcinoma have been described in men.[3-7]

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In reply. In regard to Dr Schwartz's comments on "Carcinoma of the male breast,"[1] I appreciate his thoughts and insight into the very occasional occurrence of lobular breast carcinoma. Nonetheless, the fact remains that lobular carcinoma is exceedingly uncommon in the male breast, as evidenced by the fact that only several cases have appeared in the world literature over a 25-year period.

In the study of complex disease processes, one must review the available data and condense a broad fund of knowledge into several meaningful, learnable caviats. In male breast carcinoma, one caviat remains: lobular carcinoma is exceedingly uncommon and occurs with an incidence which is orders of magnitude below that in the female breast.

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Amelioration of Graves disease during pregnancy

TO THE EDITOR: Pregnancy has been known to cause fluctuations in the course of hyperthyroidism. We report here the case of a 17-year-old girl with longstanding Graves disease who had marked fluctuations in her disease during two successive pregnancies. Although she was on maintenance methimazole therapy (30-80 mg/day) with suboptimal control (thyroxine levels in the range of 15.0 to 20.0 μg/dL), reduction and cessation of methimazole therapy was required after three to four months of pregnancy. During the patient's first pregnancy, at about ten weeks of gestation the dose of methimazole was reduced in anticipation of falling serum thyroxine (T_4) levels. She was lost to follow-up until 34 weeks of gestation, when the T_4 level was 13.4 μg/dL (normal for pregnancy, 15-17 μg/dL); thyroid stimulating hormone (TSH), 6 uU/mL (normal, 0.0-6.0 uU/mL); and free T_4, 0.9 ng/dL (normal, 0.8-2.6 ng/dL). At this point methimazole was discontinued (Fig 1) until symptoms of thyrotoxicosis returned three months postpartum.

Prior to the second pregnancy, the patient's T_4 level was in the range of 15-20 μg/dL (normal nonpregnant, 4.5-12.0 μg/dL) and triiodothyronine radioimmunoassay (T_3 RIA) was 230-250 ng/dL (normal, 90-220 ng/dL). TSI assay was not available. With the second pregnancy, levels of T_4, free T_4, and T_3 RIA were normal by the third to fourth month of gestation, at which point methimazole was tapered and then discontinued. T_4 levels were in the 8.0 μg/dL range although free T_4 and TSH levels were normal. Both pregnancies went to term and yielded infant girls with normal T_4 and TSH levels.

The antithyroid antibody titer (ATAB) was 1:25 during the second pregnancy, rose to a peak of 1:64 four months after delivery, and further decreased to 1:25 (normal, < 1:10). The antimicrosomal antibody (AMAB) titer remained at 1:6,400 until four months postpartum, when it fell to 1:400, only to rise again to 1:100,000 when thyrotoxicosis returned (normal AMAB, <1:100). Thyrotropin-binding inhibiting immunoglobulin (TBII) was 12-17% immediately postpartum but dropped to 0% despite development of thyrotoxicosis (normal, < 10%). The level of thyroxine-binding globulin (TBG) was 2.6 μg/dL prior to pregnancy (normal, 1.4-3 μg/dL) and during the last trimester was 3.9 μg/dL (normal, 5.6-10.2 μg/dL).

In summary, this patient had clearly

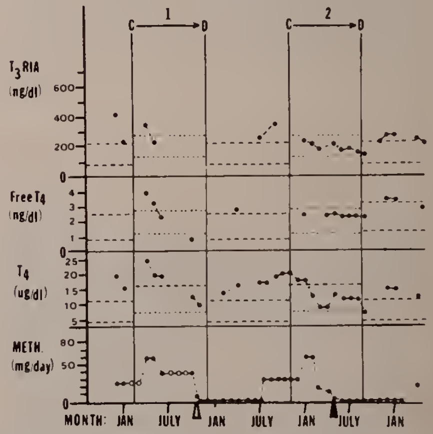

FIGURE 1. Levothyroxine (T_4), triiodothyronine radioimmunoassay (T_3 RIA), and methimazole (METH) therapy are shown as closed circles. Open circles indicate periods of time when the patient did not return for follow-up. C–D indicates the gestational periods (conception–delivery). The triangles indicate the dates methimazole was discontinued.

documented changes in thyroid hormone levels; normalization of T_4, free T_4, T_3 radioimmunoassay; and amelioration of thyrotoxicosis during two successive pregnancies. No antithyroid medication was required until after delivery, when mild thyrotoxicosis returned. Despite the lowering and discontinuation of antithyroid medication, the T_4 level fell to less than 10 ng/dL, accompanied by low free T_4 in the first pregnancy (0.8 ng/dL). T_3 radioimmunoassay levels were lower during both pregnancies but remained relatively normal as is often observed in mild hypothyroxinemia. After delivery, both T_4 and free T_4 rose to hyperthyroid levels. The antibody titers measured did not help explain the course of thyroid dysfunction in this patient, although fluctuating levels of ATAB and AMAB have been related to changes in thyroid function in pregnant women with autoimmune thyroiditis.[1] Following both the TBII and TSI has been found to be useful in following the course of Graves disease. However, at least one report has demonstrated a poor correlation between TBII levels and childhood Graves disease.[2] The doses of thyroid medication are often reduced in anticipation of decreasing requirements during pregnancy because maintenance doses of antithyroid drugs may cause maternal hypothyroidism and possible subsequent fetal hypothyroidism. This concern is supported by a recent report which showed higher free T_4 levels in the cord blood of infants born to thyrotoxic women who discontinued antithyroid medication as compared to lower free T_4 levels in infants born to women who continued taking medication.[3]

In conclusion, we report remissions in the course of Graves disease during two successive pregnancies in an adolescent. This case emphasizes the need for careful monitoring of thyroid functions in pregnancies complicated by thyrotoxicosis. Surveillance of TSI, TBII, and other immunoglobulins may be of help in anticipating fluctuations in the course of Graves disease in pregnant women.

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Preemployment drug testing of housestaff physicians

TO THE EDITOR: The increased use of drugs in the workplace and the availability of improved testing techniques is contributing to a proliferation of preemployment drug testing programs.[1] This letter describes the results of a preemployment drug testing program on housestaff physicians at the Presbyterian Hospital at the Columbia-Presbyterian Medical Center in New York City.

Previous reports have noted that physicians are 30 to 100 times more likely to become addicted than are members of the general population.[2] Although reports of drug use among physicians are not new, no one really knows how many physicians have drug-related problems. A recent analysis of the published literature concluded that the overall prevalence of drug problems among physicians may not be different from that found among the general population.[3]

Housestaff physicians, however, are a specific subgroup of physicians. They are generally younger, usually ranging in age from 24 to 36 years. Housestaff physicians face unique stresses in relation to their training programs, career identities, and developing family relationships. Depression and psychological distress during the training years are commonly referred to as "house officer stress syndrome."[4] One study of housestaff physicians reported that 12.7% had scores on a self-administered Michigan Alcoholism Screening Test that were either suspicious or presumptive of alcoholism.[5] While no reliable data are available on the overall prevalence of drug use among housestaff physicians, their age and the stresses encountered during housestaff training may contribute to making them vulnerable to drug use.

The Presbyterian Hospital is a 1,291 bed, urban, tertiary care institution employing approximately 650 housestaff physicians. Guided by the New York State Health Code, which prohibits any person from being employed in a health care setting "who is habituated or addicted to depressants, stimulants, or other substances that may alter behavior," a housestaff preemployment drug testing program was initiated. All new housestaff, as part of their routine preemployment examination, are evaluated for the unprescribed use of depressants, stimulants, narcotics, hallucinogens, or other illicit drugs that may affect behavior.

All 164 prospective housestaff physicians were notified in advance in writing about the drug testing program. Housestaff orientation includes a detailed explanation of hospital drug testing policy and available counseling and treatment services, and provides an opportunity for discussing problems of drug use and chemical dependency among professional staff.

All housestaff are required to have completed a standard preemployment history and physical examination before their arrival. During orientation week, laboratory specimens are obtained in the Employee Health Service under the supervision of the occupational health nursing staff. Urine samples for drug testing are placed in a tamper-proof, sealed container with an accompanying chain of custody form.

All samples are analyzed in the hospital laboratory by radioimmunoassay for barbiturates, opiates, methadone, amphetamines, benzodiazepines, cocaine, alcohol, phencyclidine, and tetrahydrocannabinol (THC). Positive specimens are sent to an outside laboratory for confirmation by gas chromatography/mass spectrometry. A test for THC is not considered positive unless there is more than 75 ng/mL present. Positive test results, not supported by legitimate prescription, result in the individual's not being hired. All test results remain confidential.

A total of 102 male and 62 female housestaff were tested. All (100%) tested negative for drugs.

The institution of preemployment housestaff drug testing appeared to be neither objectionable nor burdensome. Because prospective housestaff receive advance notification, routine preemployment drug testing is not an accurate indicator of recent drug use. However, drug testing does provide a clear message that drug use by housestaff is not condoned. The inclusion of drug testing provides an opportunity to discuss issues of substance abuse and to inform housestaff of available treatment resources.

A drug testing program is only as

good as the policies, procedures, and personnel governing its use. It is essential that proper procedures be followed for specimen collection, handling, transport, and analysis, and that policies be fair and nondiscriminatory and protect the confidentiality and dignity of the individual.

The results of this study indicate that preemployment housestaff drug testing, if carefully planned and implemented, may be a useful adjunct to a broader approach to managing drug use among physicians.

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ERRATUM

We regret that the name of Dr Allison B. Landolt was misspelled in the article which he coauthored in the July 1988 issue, "The anencephalic fetus and newborn as organ donors."

FROM THE LIBRARY

CREDULIM AND SKEPTICISM IN MODERN MEDICINE

. . . This perfunctory administration of drugs is one of the crying evils of the medical practice of the present day. The physician is to frequently imbued with the idea that it is not only his privilege, but his professional duty to hand over to the patient a written prescription. In the minds of the laity the common belief prevails that disease can only be cured by drugs, and this belief is generally encouraged and often unfortunately shared by many of our profession. Indeed, it is the undue importance which the physician usually attaches to his Latin prescription which leads patients to believe that his advice is perhaps worth listening to, but seldom worth following.

Most physicians of experience will readily agree that the majority of patients who consult them for chronic ailments are far more in need of judicious advice regarding their habits of life than they are in need of medicine. And yet how few of us ever make the advice more prominent than the medicine! Theoretically the advice and the prescription may supplement each other and both do good. Practically the written prescription is apt to do positive harm in many cases by diverting the patient's mind from the advice which is so essential for him to follow, and centring it upon the pharmacopoeial remedy. It is this unfortunate custom on the part of physicians of writing a prescription for each and every patient that fosters the popular belief that disease can only be cured by drugs, and does far more than anything else to increase the business of the patent medicine venders. It is this routine administration of drugs that is gradually shaking the faith of the public in the efficacy of orthodox medical treatment—lessening confidence in true medical science, and leading many people of intelligence to investigate the alleged virtues of Christian science, mental healing, electric appliances, and movement-cures, or forcing them to the conviction that they will do better under the care of the athletic trainer than in the hands of the average drug-prescribing doctor. . . .

GEORGE HENRY FOX, MD
(*Trans Med Soc State NY* 1895; pp 39-50)

Reprinted from the June 1988 issue of Epidemiology Notes (*Vol. 3, No. 5*), *published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

Long Island Breast Cancer Study

As many as 2,000 breast cancer deaths could be prevented on Long Island in the next 20 years with widespread use of physician exams and mammographic screening.

This potential impact of prevention screening is highlighted in the first report of the Long Island Breast Cancer Study, a cooperative effort of the Nassau and Suffolk County Health Departments, the New York State Department of Health and the State University of New York at Stony Brook. It represents the first population-based study of breast cancer in New York State.

The study was prompted by an analysis of mortality data indicating that the breast cancer rate in Nassau County is 13% higher than the New York State rate and 12% higher than the Suffolk County rate.

The study establishes a breast cancer risk profile for women in Nassau and Suffolk, and explores the differences in risk factors between the two counties. In addition, the study provides a basis for breast cancer detection programs on Long Island.

To establish a risk profile, 1,420 women with breast cancer were interviewed plus 1,420 women without the disease, matched by age and county. Participants were interviewed by telephone about social, occupational, nutritional, environmental and health histories. In addition, 12,177 pathology slides (from 755 cases) were reviewed for diagnostic accuracy.

A comparison between Nassau and Suffolk controls (ie, women without cancer) showed that Nassau women tended to have a higher frequency of long-term or lifelong residence in the county than their Suffolk counterparts. The Nassau population also had a higher frequency of known breast cancer risk factors: they are more highly educated, more often Jewish, have an earlier menarche (before age 12), a later menopause (after age 49), a later age at first birth and a higher frequency of benign breast disease. Family history of breast cancer was about the same in both control groups.

Breast cancer screening practices were also studied for the Long Island population. The data show between 50% and 75% of women visited a physician one or more times a year in the previous year. A large percentage of women had also learned how to examine their breasts for physical changes, but only one-third practiced it monthly. About 40% of women ages 50 to 70 had at least one mammogram in their lifetime, but only 10% in Nassau and 5% in Suffolk received the annual mammogram recommended by both the American Cancer Society and the National Cancer Institute for women over age 50.

These statistics are comparable to levels in statewide surveys. But the National Cancer Institute has recommended a goal of 80% participation in annual clinical examination and mammography by the year 2000. At that level of participation, a total of 4,180 deaths could be prevented in New York State.

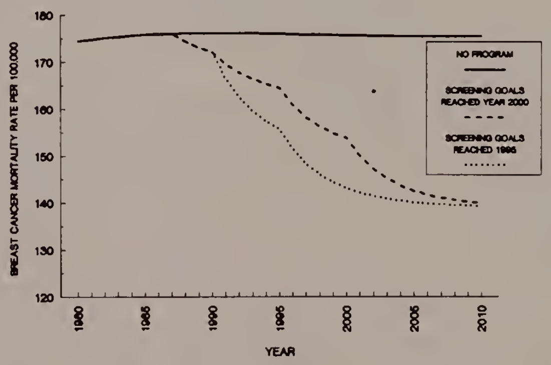
Early detection provides the best opportunity for survival. A study of Nassau and Suffolk counties indicates that most areas can meet the demand for optimal mammography services.

Seventy-five percent of the breast cancers included in this study were first detected by patients themselves, 56% were detected accidentally (eg, while bathing), and 19% were detected during breast self-exam. Fifteen percent were detected by a physician during a routine exam, and only 8% were detected by mammography.

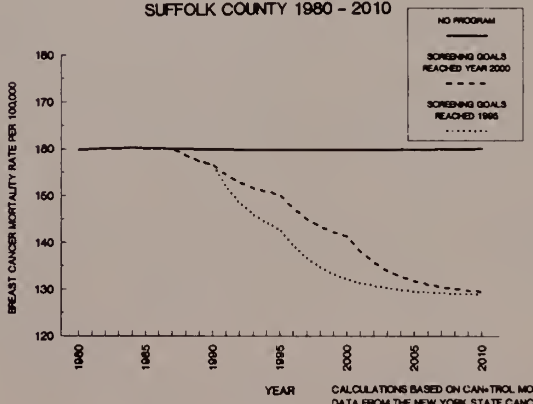
Breast cancer remains the leading form of cancer among New York women with more than 10,000 new cases and 3,700 deaths reported each year. In New York State, breast cancer is responsible for over 14,000 hospital admissions, 163,000 hospital days and 247,000 years of life lost each year.

Because breast cancer continues to be a major cause of illness and death among women, the New York State legislature allocated $1 million in 1987 to the State Health Department for support of breast cancer detection and counseling services. The goal is to facilitate the earlier detection and treatment of breast cancer through informa-

PROJECTED REDUCTIONS IN BREAST CANCER MORTALITY
DUE TO MAMMOGRAPHY AND CLINICAL EXAMINATION:
NASSAU COUNTY 1980 - 2010



SUFFOLK COUNTY 1980 - 2010



CALCULATIONS BASED ON CAN•TROL MODEL AND
DATA FROM THE NEW YORK STATE CANCER REGISTRY

tion, education, referral and the provision of services, with a special emphasis on minority populations.

Nassau County has submitted an application to further support these goals. In addition, the Nassau County Health Department currently provides programs on teaching breast self-examination (in conjunction with Nassau County Medical Center), and sponsoring mammographic exams free of charge at 30 locations through the mobile unit of Women's Outreach Network.

In Suffolk County, a full scale mobile screening program for all women over 36 is in operation, that includes education on early detection and breast self-exam, physical breast examinations and mammograms. The county subsidizes costs for those patients without private health insurance or other sources of payment.

Other State Health Department initiatives include: a program to assure that mammograms are of good diagnostic quality, the development of screening guidelines for physicians and additional research activities.

Future reports on the Long Island Breast Cancer study will focus on lifestyle factors (diet, smoking, alcohol consumption), general environment (length of residence, drinking water supply) and medical care factors (use of menopausal and other hormones). The next report is expected within the year.

For more information about the Long Island Breast Cancer Study or for copies of the report, contact Philip C. Nasca, PhD, Director, Bureau of Cancer Epidemiology, New York State Department of Health, at (518) 474-7950.

All-terrain vehicles

First marketed in 1970 for off-road use by farmers and hunters, all-terrain vehicles (ATVs) have experienced increased popularity in this country. Approximately 2.5 million Americans own ATVs.[1]

Consumer Product Safety Commission (CPSC) statistics and sales figures of the major manufacturers—Honda (60% of sales), Yamaha, Suzuki and Kawasaki—show a ten-fold increase in the number of ATVs in use between 1980 and 1986. Consumers are purchasing approximately 2,000 of these small three- and four-wheeled motorized cycles per day at a price of $200 to $3,500 each. However, the soaring popularity has been accompanied by a corresponding increase in deaths and injuries related to ATVs.[2]

CPSC injury statistics estimate the number of ATV-related injuries treated in hospital emergency rooms rose ten-fold from 8,600 in 1982 to 86,400 in 1986.[1] Almost half of the injuries were incurred by young people under age 16. The most recently analyzed CPSC data of June 1987 tallied 789 fatalities nationwide (1982–1987), 44% of which were under the age of 16 (345 deaths) and 20% were under the age of 12 (55 deaths). The CPSC task force studying ATVs advised that children under 12 lack the physical and cognitive abilities to ride ATVs and that young people 16 and under should not operate adult-sized ATVs. As reported by the task force, 30% of all ATV fatalities were alcohol-related and 14% of the operators involved in reported injuries had consumed alcohol prior to riding ATVs.[3] The CPSC estimates that in 1986 alone, ATV-related deaths and injuries were responsible for costs in excess of 1.5 billion dollars.[4]

The CPSC has reported 55 fatalities in New York State since 1982. Of these, more than one-third (20) were under the age of 16. Over three-fourths (78%) of the deaths involved the three-wheeled model (43 victims). However, the number of deaths involving four-wheelers is on the rise. Twenty-nine percent of the fatal crashes in 1986–1987 involved the four-wheeled ATV, up from 19% in 1985 and 5% in prior years.[5,2]

The three-wheeled model of ATV comprised most of the market through 1984. In 1982, the four-wheeled ATV represented 11% of the sales, rising to 28% in 1984, 61% in 1985 and more than 82% for the first half of 1986. In 1987, only two manufacturers were still producing the three-wheeled model.[2]

The average useable life of an ATV is seven years and during that time there is a one-in-three chance of its operator being involved in an incident which results in injury. CPSC data in 1986 reported 11% of the persons treated for ATV-related injuries in hospital emergency rooms were hospitalized, indicating the severity of the injuries.[2]

The design of ATVs has three major flaws. First, the lack of mechanical shock absorbers creates problems in maneuvering through bumps and ruts. The early ATVs relied on large soft tires to absorb the impact from rough terrain. Some newer models are fully suspended, but approximately two-thirds of ATVs sold are not.

Secondly, the solid rear axle in three-wheelers often forces the outside wheel off the ground during turns. The driver must lean to the opposite side to keep balance. The maneuvering requires skill often not possessed by untrained drivers.[2]

The third flaw is that ATVs have a high center-of-gravity that, coupled with the three-wheeled design, creates a very unstable vehicle. The CPSC data indicate that ATVs experience a high roll-over rate that can cause serious injury or death.[2]

The Health Department advises that parents consider the safety issues before deciding to purchase an ATV. Health care providers and educators can increase public awareness of the following CPSC safety recommendations.[1]

- Beginning riders need training from certified instructors. (The Department of Motor Vehicles and the Special Vehicle Institute of American are two organizations that provide information about instructor certification.)
- Practice driving skills on a level area before riding on rough or unfamiliar terrain.
- Maintain adult supervision.
- Use only child-sized ATVs for children between ages 12–16.
- Wear a protective helmet, gloves, boots and long pants.
- Do not ride double.
- Never use alcohol or drugs.
- Read the owner's manual and follow instructions for use and maintenance.
- Avoid public highways.

- Drive at reasonable speeds.
- Use good judgment.

The CPSC estimates that the use of helmets could prevent 25% of all deaths from head trauma in ATV operators. The American Academy of Pediatrics and the Medical Society of New York State further recommend that children under age 14 be restricted from operating ATVs.[4]

New York State legislation regulating the use of all-terrain vehicles took effect in 1987.[6] The law requires young people ages 10 to 16 to be trained in safe operating techniques for use of ATVs. According to the Department of Motor Vehicles (DMV), 69 training sites have been established, predominantly in 4-H Cooperative Education Centers. The Board of Cooperative Educational Services, Erie County Community College and several ATV dealers also offer sites for training. The course curriculum was developed by the Special Vehicle Institute of America, a not-for-profit organization founded by major manufacturers. The course is approximately four hours in length and is limited to classroom instruction only. The costs of demonstration ATVs and insurance prohibit participatory classes. In 1987, the New York State Department of Motor Vehicles reportedly trained 4,500 people in the 10 to 16 age group.[7]

The law requires all owners to register their ATV for a fee of $10. One-half of the fee is targeted for trail development, with the remaining $5 to supplement law enforcement. In 1987, the DMV registered 30,000 vehicles in New York State, one-fourth of the estimated 120,000 ATVs currently in use in the state.[7]

In December 1986, the CPSC recommended to the US Department of Justice a recall of all three-wheeled ATVs and all four-wheelers for use by children under 16 years of age and enforcement of a manufacturer's notification and education program. One year later, a lawsuit was filed in US District Court to declare ATVs as imminently hazardous consumer products. In response, the Department of Justice announced a preliminary consent decree halting the sale of all three-wheeled ATVs and requiring the industry to launch a rigorous public awareness campaign to inform consumers of the risks related to ATVs. The decree also requires training of new ATV buyers and of those who purchased one in the last year, a notification of potential hazards mailed to past purchasers, warnings displayed in dealer showrooms and a restriction on marketing adult-size ATVs to children under 16. [8,1]

For more information about the prevention of ATV-related injuries and deaths, contact the Injury Control Program at (518) 473-1143.

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Ferrets as pets

The European ferret (*Mustela putoris furo*) has become increasingly popular as a household pet in New York State. This wild animal is a descendant of the European polecat (*Mustela putoris*). During 1987, over 5,000 permits were issued by the New York State Department of Environmental Conservation (DEC) for the purchase of ferrets. This probably constitutes an underestimate of the number of ferrets actually purchased since many ferrets are purchased without a required permit, and a single permit allows any number of ferrets to be kept.

From 1984 to 1987, the number of ferrets submitted to the New York State Department of Health rabies laboratory (WCL&R) for analysis increased from 13 to 57. Of the total, 90% were submitted due to a biting incident. Reports of ferret bites, which in some cases have been mutilating, continue to increase. California has accumulated data[1] on more than 33 attacks on infants and young children, some so severe that plastic surgery was necessary. In New York State, numerous biting incidents have occurred. In one instance a ferret attacked a baby sitting on the kitchen floor. Before the mother could rescue the child, 12 to 15 bites had been inflicted on the baby's arms and face. It is not known why ferrets have a tendency to bite young children; but it has been suggested that babies emit an odor resembling baby rabbits, or that they carry the residue smell of spilled milk. Whatever the reason, it is not uncommon for household ferrets to crawl into a baby's crib and attack the child.

There is no licensed rabies vaccine for use in ferrets, nor in any other wild animal. Although the likelihood of ferrets contracting rabies is slight, the possibility exists. At least 11 cases of ferret rabies have been reported since 1980.[2] Ferrets which are caged or never leave the house are unlikely to become infected, however in some of the reported cases the ferrets had temporarily escaped, were exposed and later diagnosed with rabies. In New York State, laboratory reports indicate that as many as 30% of the animals submitted are strays with no known owner.

Since ferrets are considered to be wild animals in New York State, a biting ferret would have to be destroyed and submitted to the rabies laboratory for testing. The 10-day confinement period permitted for dogs and cats is not permitted for ferrets since the incubation period for rabies in that animal is unknown.

The New York State Department of Health recommends that households with children under six years of age do not have a ferret in the house. Even those ferrets which are caged may escape when adults are not present and attack young children. In order to assure that prospective ferret owners are aware of the safety hazards, the department has prepared a special ferret warning statement which the DEC has included in its permit application form.

Biting incidents should be reported to your local health department or to Dr John Debbie, Bureau of Communicable Disease Control, at (518) 474-3186.

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CANCER OF THE HEAD AND NECK

By Stephan Ariyan, MD. 827 pp, illustrated. St Louis, Mo, CV Mosby Co, 1987. $125.00 (hardcover)

Dr Ariyan, in conjunction with other authors, has written a textbook on cancer of the head and neck which is meant to serve as a resource for clinicians interested in the management of patients so afflicted. Dr Ariyan is a well-known authority in the field of cancer treatment and reconstruction, and has had experience in all aspects of head and neck cancer therapy. Other contributors include an international group of individuals from allied medical and surgical disciplines. Dr Ariyan has organized his book in unique fashion in that he separates malignancies into different anatomic sites, which allows the surgical as well as radiotherapeutic treatment options of these lesions to be discussed in the same chapter.

The initial section of the book deals with general principles and biological aspects of malignancies. Included is a historical perspective of cancer treatment as well as a discussion of trends in cancer of the head and neck. Next, a review of the radiobiology of therapeutic radiation is presented, followed by a chapter on clinical application of therapeutic radiation. Two chapters on chemotherapy dealing with the pharmacobiology of chemotherapy as well as clinical applications are next and serve as a concise description of current chemotherapeutic agents and their use in head and neck cancers. A chapter on dental care of the cancer patient includes a discussion of maxillofacial prosthodontics as well as other aspects of dental care following radiation therapy. Finally, chapters on physical diagnosis and staging of head and neck cancer, and a discussion of premalignant and malignant lesions of the oral mucosa conclude the introductory section.

Part Two concerns clinical management. Cancer of the oral cavity is discussed relative to the principles of surgical management and radiotherapy. A chapter on reconstruction of the oropharyngeal area includes a good explanation of the various myocutaneous flaps used in head and neck reconstruction. Following are three section reviews of cancer of the nasopharynx and carcinoma of the paranasal sinuses. Relevant to the foregoing is a chapter regarding cranial orbital resection and reconstruction. Cancer of the larynx and hypopharynx are reviewed and complemented by a discussion of pharyngoesophageal reconstruction. The techniques of flap reconstruction, gastric pull-up, and microvascular bowel transfer are well covered. Microvascular surgery as it applies to the head and neck is covered separately and is reasonably complete.

A good discussion of neck dissection and the pathophysiology of cervical lymph node metastases is presented as well as lengthy descriptions of classical and functional neck dissections. The text concludes with chapters on salivary gland tumors, carcinoma of the thyroid, parathyroid tumors, and melanoma.

The book is generally well written, and adequately illustrated. There are numerous photographs, photomicrographs, surgical drawings, and reproductions of scans in the book. Although the book is not intended to be an atlas of surgical techniques, there are areas in which considerable exposure is given to technical issues, which helps clarify some of the discussions in the text.

I believe this book compares favorably with other single-volume publications in the field and achieves its goal as a reasonably up to date, nonexhaustive text on the diagnosis and treatment of the various cancers of the head and neck.

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PRIMARY CARE OF CANCER. RECOMMENDATIONS FOR SCREENING, DIAGNOSIS AND MANAGEMENT

By Edward A. Mortimer, Jr, MD. 195 pp. Cleveland, Oh, Case Western Reserve University School of Medicine, 1987. $14.00 (paperback)

Cancer is a difficult office diagnosis for the primary physician to make. One reason is that it is infrequent. Slightly more than one in 1,000 patients will have a diagnosis of a new cancer upon presenting themselves to a physician in the office. Also, primary care physicians tend to be at greater ease with diseases they can diagnose and treat themselves in their offices. However, everyone agrees that because of its potentially lethal nature, the outcome for cancer is generally better if it can be diagnosed early and treated promptly. Herein lies the dilemma: How can one be on the alert and always thinking of a disease which is newly diagnosed about 12 to 15 times a year in the average physician's office?

It is because of these considerations that this book is valuable, although the title appears to belie its small size of 195 pages. The book is "designed to provide practical, clinical information for the primary care physician." Despite my misgivings upon picking the book up, I was pleasantly surprised. With such a panel of distinguished cancer specialists as contributors, I expected it to be obtuse and impractical. However, it provides practical clinical information for the primary care physician. Kudos to the editorial committee for keeping it basic enough to be understandable, yet clinical enough to be useful.

An important and dutiful warning is inserted that the types of cancer included are the 30 most common types reported in the Northeast Ohio area (which may vary slightly from rates in other areas of the country). The reader is reminded that the recommendations must be considered as general guidelines and not as standards of care; and I agree that both of these things are important to keep in mind.

Mention is made in the introduction that no similar publication designed to assist primary care physicians in the prevention and control of cancer in their patients is available. I think this is probably true. However, several publications are available that have been helpful to me. Like this one, they are supported at least in part by the American Cancer Society, making them widely available and inexpensive. For the sake of completeness they deserve mention. Information about any of these is available at a local office of the society. They include *Clinical Oncology: A Multidisciplinary Approach; The Cancer Manual* (Massachusetts Division); and the booklet that has proved very helpful in my own practice—*Follow-up of Cancer, A Handbook for Physicians* (Connecticut Division). Then, of course, there is the American Cancer

Society's *Cancer Book*, edited by Dr Arthur Holleb. All of these have some information which is included in *Primary Care of Cancer*, but none of them are as complete or direct as the latter.

Now for some comments about the contents of the manual. It would have been helpful to include an explanation of the estrogen receptor concept, for this is relatively new and not well understood by many practicing primary care physicians. Under the section on head and neck cancers, I appreciated being reminded again of the importance of deferring a request for a biopsy of cervical adenopathy before conducting a careful physical and endoscopic examination of the oral cavity and larynx. We tend to think (correctly) in terms of biopsy of unknown masses, but here is an important exception.

Another good reminder was the note that almost half (40%) of patients with renal cell carcinoma do not show abnormalities on microscopic examination of the urine. The book noted also that the number of childhood cancers is small and treatment is both evolving and demanding so that, whenever possible, initial workup in a tertiary care center should be considered for all children.

Lastly, a comment about the final chapter, "Home Care for the Terminally Ill Patient." It is certainly an appropriate topic, but seems almost out of place as the only chapter not addressed to a specific cancer site. When this manual is revised and updated, I would like to suggest that consideration be given to adding a chapter on pain control. This is a very important and often underdiscussed area of cancer treatment. I believe primary care physicians are not comfortable or adept in this vital area of care of their cancer patients. This is an excellent small volume and would be a worthwhile addition to a primary care library.

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CEREBROSPINAL FLUID AND THE BRAIN EDEMAS

By Thomas Herrick Milhorat, MD. 168 pp, illustrated. New York, Neuroscience Society of New York, 1987. $45.00 (hardcover)

This concise monograph with 58 illustrations and extensive but not exhaustive references is a delightful book to read. It also reflects the razor-sharp mind of a neurosurgeon who utilizes the little spare time he has to its maximum effectiveness. Neurosurgeons have to dwell in realities and have little time for armchair hypotheses; this book does the same. While there are other and bigger books on either cerebrospinal fluid or cerebral edema alone, this one combines a discussion of both, a unique and useful idea. Another appealing feature is the inclusion of various types of illustrations varying from beautiful hand drawings, electron micrographs, and computed tomographic and magnetic resonance imaging scans to quantitative autoradiographs employing double-marker techniques and radioisotopic cisternographs. All these are used to illustrate precisely each major point. There are a few deficiencies, of course, such as the lack of histologic illustrations depicting the various acute and chronic changes associated with cerebral edema, but these are minor. The author's flow of thought is easy to follow, and the extensive footnotes contain important ancillary information.

After a brief and interesting introduction to the historical aspects of cerebrospinal fluid circulation and cerebral edema, four major chapters follow, one each on the anatomy and physiology of cerebrospinal fluid formation and circulation, the classification of brain swelling and edema, and one special chapter on interstitial edemas. Subjects such as pseudotumor cerebri and the cerebral edema associated with intracranial tumors are covered, enhancing the clinical usefulness of this book.

Of particular interest is the author's approach to the classification of cerebral edema. He favors the separation of cerebral swelling from cerebral edema, originally classified by Igor Klatzo (1967) into cytotoxic and vasogenic types. Under cerebral swelling he includes increased vascular volume and cellular swelling as the main entities. True interstitial cerebral edema or fluid accumulation in the extracellular compartment is subdivided into vasogenic, osmotic, compressive, and hydrocephalic types. There is much sense in this modification, especially when there is clarification later regarding a variety of clinical disorders that may employ more than one mechanism in causing elevated intracranial pressure. While neurosurgery residents should benefit most from this book, neurologists, neuropathologists, and those medical personnel who are entrusted with the daily monitoring of intracranial pressure in hospital patients will get an overall view of the subject of cerebral edema.

The brevity of expression is a measure of the author's respect for the time of others in the busy world of science and medicine. Dr Milhorat is well known among neurobiologists, neuropathologists, and neurosurgeons for his work on cerebrospinal fluid circulation. This book is a nice legacy to science from a dedicated and hard-working academician.

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CONTROVERSIES AND CLINICAL VARIANTS OF MIGRAINE

Edited by Joel R. Saper, MD. 127 pp. Elmsford, NY, Pergamon Books, Inc, 1987. $29.50 (hardcover)

This book is a collection of papers from some of the leading headache experts in the world. The list of authors contributing to this small book is remarkable, and the quality of the discussions provided is consistently good. The occasion for this distinguished gathering was the June 1983 meeting of the American Association for the Study of Headache. This is a collection of papers presented at that conference, a conference which focused on controversies in the field of migraine research, diagnosis, and treatment.

For the practicing neurologist who every day faces the diagnostic and therapeutic dilemmas posed by patients with migraine headaches, this little book is delightful and enlightening. It is delightful because the contributors are articulate and authoritative. It is enlightening because they are able to settle none of the controversies that are discussed. For most neurologists, the diagnosis and management of migraine is often frustrating and occasionally treacherous. Patients with symptoms of classic migraine, complete with premonitory visual disturbances, unilateral headache, vomiting, photophobia, and irritability, occasionally have structural brain lesions, such as vascular malformations or low grade gliomas. Any patient with complicated migraine, in which focal neurologic deficits persist beyond the onset of the headache, may develop cerebral infarction during a migraine attack, an attack which is identical to dozens of prior episodes that caused no structural damage to the brain. Every neurologist concerned that inadequate familiarity with the most recent thinking on migraine limits his or her ability to deal optimally with such headaches will take comfort in the perplexity of the most authoritative neurologists in the field.

The controversies raised are discussed to varying extents, but none is resolved. The pathogenesis of migraine is more uncertain now than it was 20 years ago. Reactive vasoconstriction followed by excessive vasodilation neatly explained many of the phenomena observed in migraine decades ago. However, more discerning investigative techniques and equipment have revealed that these vascular events, when they occur at all, do not explain the most important components of migraine episodes, including the auras and the headaches. Vascular changes are out of step with symptom evolution. The treatment of migraine and its variants is not systematically discussed in any of the papers, but this is partly because there was no consensus among the participants on how to treat migraine. Some strongly advocate beta blocking drugs and ergotamines; others dismiss all current efforts at treatment as inadequate. The shortest paper in the collection is on the etiology of migraine, and this is the most provocative. In it, Dr Lance of Australia proposes a complex model for the evolution of migraine which involves many elements of the central and autonomic nervous systems. This closing chapter is a glimpse of where the future probably lies in migraine research.

The major shortcomings of this book are its limited scope, late publication, and poor illustrations. For the neurologist interested in recent ideas on migraine, it is provocative and valuable. For other physicians, it is highly technical and circumscribed. The book discusses controversies in the area of migraine study and little else. Regrettably, the publication and release of this 1983 conference was apparently delayed until 1987. Its brevity and simple format provide no clue as to what required more than three years for production. One of the attractions of this type of conference is that it provides physicians who did not participate with an idea of what was discussed. That this discussion occurred five years ago makes it less newsworthy. The other disappointing feature of the book is that it has only one illustration. Several of the presentations read as if there were accompanying slides, but figures and diagrams are completely absent until the last chapter.

This is overall an enjoyable, but brief, monograph. Every chapter has something to interest a neurologist or headache specialist. It should not be misconstrued as a reference text for the internist or family physician. The real value of the book lies in its consideration of neurological arguments, arguments with which most non-neurologists will simply not be familiar or concerned.

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INSECT ALLERGY. ALLERGIC AND TOXIC REACTIONS TO INSECTS AND OTHER ARTHROPODS

Second Edition. By Claude Albee Frazier, MD. 463 pp, illustrated. St Louis, Mo, Warren H. Green Inc, 1987. $42.50 (hardcover)

This is the second edition of a book initially published in 1969 which addresses a wide range of reactions to insects. The contents include discussions of classical allergic reactions to stinging insects, biting insects, inhalation of insect debris, and entities such as body lice and scabies. The extensive entomologic data with descriptions of insect habitat and life cycles is basically unchanged from the initial publication.

The major concerns in the field of insect allergy are anaphylactic reactions to the sting of insects such as bees, wasps, hornets, and yellow jackets, which are frequent and can be fatal. Since the availability of purified venoms for diagnosis and treatment in 1979, there has been an enormous advance in the understanding of the problems of stinging insect allergy. Areas under study in which important information has accrued include the natural history of the disease process, appropriate diagnosis and therapy for patients at risk, mechanisms of immunity, cross-reactions between various venoms, and knowledge of the important constituents of insect venoms. In addition, sensitivity to the fire ant, a nonwinged Hymenoptera which is an increasing problem in southeastern United States, has received extensive analysis in recent years. Unfortunately, none of these advances are critically detailed in Dr Frazier's book.

The most recent entry in the bibliography of the Hymenoptera section is dated 1979. This important chapter was apparently prepared at that time and never updated. The author states that venom extracts had just been released for use at the time the chapter was written. Whole insect body extracts, now recognized as impotent and no longer marketed for diagnosis and therapy, are still described in this chapter as an effective modality. Other concerns, such as the indications for immunotherapy and the important allergens in venom, are inaccurately addressed. It is difficult to understand how a supposedly current textbook on insect allergy can be published and marketed when so clearly out of date.

The sections on reactions due to biting insects and allergy due to inhalation of insect debris contain no substantive changes from the first edition. Critical analysis of the biting insect problem is lacking. There are only a few biting insects which have been documented as being responsible for causing generalized allergic reactions with an IgE-mediated pathogenesis. Such reactions would be amenable to appropriate immunotherapy and are not clearly distinguished from the problems of primarily local reactions related to other types of biting insects, such as fleas and mosquitoes. Once again, the bibliography in this section stops in 1980.

The insect habitats are well described. The illustrations vary greatly in quality and would be improved considerably with the use of color plates. Anecdotal case reports, generally serving little purpose, are found throughout the text.

This book may provide some information for individuals interested in insect entomology. Unfortunately, it is entirely inadequate for a current discussion of allergic reactions to insect bites and stings.

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GERIATRIC MEDICINE ANNUAL 1987

Edited by Richard J. Ham, MD. 284 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $39.95 (hardcover)

This book is the second volume of an annual series. As with the previous volume, the authors address important geriatric topics appropriate for this rapidly developing area of medicine. The topics included in the 1987 *Annual* are very timely and in the opinion of the reviewer more valuable to the practicing clinician than the first volume (1986).

Osteoporosis, home care, alcoholism, and sleep disorders are a few of the selections offered to the reader. Other areas included are exercise prescriptions, malnutrition, pressure sores, oral health, and anemia of the older adult. The chapters on dementia and Alzheimer disease deserve special mention.

Dalziel presents an extremely well organized and concise chapter on approaches to the demented patient. He accurately distinguishes delirium from dementing conditions and gives helpful suggestions for differentiating between these two clinical states. There are clear, concise tables of data included in the chapter that will be helpful to clinicians reviewing this topic. The role of depression and diseases in causing the patient to appear demented is also clearly outlined.

Following Dalziel's chapter is a section entitled "Alzheimer's and Family" written by the editor, Richard Ham, MD. This chapter is one of the most interesting and thought provoking that this reviewer has read to date on this topic. The material is organized and oriented to the primary care clinician who not only diagnoses this problem but also longitudinally cares for the person and the family. Ham gives an excellent review of the current literature, a means of assessing the severity of the process, and suggestions on how to treat and educate the family. This chapter and Dalziel's constitute a very complete discussion on dementia and a valuable reference for any gerontology course.

As with the 1986 *Annual*, the clarity and style of each chapter is excellent. The unevenness in the amount of detail and depth of discussion of topics noted in the previous volume has been corrected. There is an obvious effort to make this book as multidisciplinary as possible so as to appeal to diverse readers. The book is a valuable review for those clinicians who have a knowledge of adult medicine. The references listed after each chapter allow for more in-depth review for the reader and a "Suggested Further Readings" section is also an added feature.

Because of the rapidly growing volume of geriatric/gerontological literature, it is not possible to buy a single reference text adequately covering this area. Research and development of new conceptual models of medical care also make an expensive reference text out of date by the time it is published. For these reasons, periodic review texts such as the *Geriatric Medicine Annual* are a more logical means of remaining current in this area. This reviewer continues to feel that this series is an excellent resource and hopes that it will be an ongoing series.

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ENJOYING LIFE WITH EMPHYSEMA

Second Edition. By Thomas L. Petty, MD, and Louise M. Nett, RN. 178 pp, illustrated. Philadelphia, Pa, Lea & Febiger, 1987. $14.95 (paperback)

Enjoying Life with Emphysema is a carefully written, brief book for patients with emphysema and chronic bronchitis. The authors are well qualified to write this second edition, as over the years they have treated large numbers of patients who have these disorders. Petty and Nett have had well-publicized interests in the development of new methods of care for respiratory patients, including pulmonary rehabilitation techniques and home oxygen therapy.

The book has 178 pages and includes 52 figures, as well as a fairly sophisticated glossary of terms for the reader. In addition, there are suggested references and reading lists for patients and their families as well as addresses where pamphlets for laypersons can be obtained. Furthermore, instructive questions commonly asked by patients are sensitively answered throughout the book, making it a practical text.

This soft-covered book is divided into 15 sections, initiated by descriptions of the lungs and exactly what emphysema and chronic bronchitis are. The authors then discuss topics such as oxygen, rehabilitation, work, and recreation. Problems that emphysema patients have that are associated with travel are particularly well covered. The section on sexuality is handled in a sensitive and informative manner.

What makes this book successful is that the authors, through their clinical experience, have captured the essence of the emphysema problem and have effectively expressed it in terms the layman can understand. Throughout the book they have conveyed important information about the nature of emphysema, and how patients are evaluated and treated. Through the use of good photographs, patients are seen receiving continuous oxygen therapy while carrying out daily activities, even swimming in a pool. In my opinion, therefore, the book makes believers of patients afflicted with this disease. "If the patient in Petty and Nett's book can do it, so can I!" That is the message that continuously comes through—"Remember to Live!"

I believe this book is straightforward, quick reading, and a must for all clinicians who manage patients with chronic obstructive lung disease. It certainly should be prescribed for all patients who are diagnosed with emphysema, as well as their family members.

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BOOKS RECEIVED

AIDS

The AIDS Bureaucracy. Why Society Failed to Meet the AIDS Crisis and How We Might Improve Our Response. By Sandra Panem. 194 pp, illustrated. Cambridge, Mass, Harvard University Press, 1988. $22.50 (clothcover)

AIDS: The Facts. By John Langone. 247 pp. Waltham, Mass, Little, Brown & Co, 1988. $9.95 (paperback)

AIDS: A Guide for the Primary Physician. Edited by King K. Holmes, MD, and Arno G. Motulsky, MD. 66 pp, illustrated. Seattle, Wash, University of Washington Press, 1988. $15.00 (paperback)

CARDIOLOGY

Cardiology Handbook for Health Professionals. By M. Wayne Cooper, MD. 186 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1988. $22.50 (paperback)

DERMATOLOGY

Colour Atlas of Dermatology. By Prof. L. K. Bhutani. 209 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, Publishers, 1988. $42.50 (hardcover)

GASTROENTEROLOGY

Diseases of the Colon, Rectum, and Anal Canal. Edited by Joseph B. Kirsner, MD, and Roy G. Shorter, MD. 724 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $115.00 (hardcover)

GERIATRICS

Research and the Ageing Population. By Ciba Foundation Symposium 134. 264 pp, illustrated. New York, John Wiley & Sons, Inc, 1988. $49.95 (hardcover)

Caring for the Disabled Elderly. By Alice M. Rivlin and Joshua M. Wiener. 318 pp. Washington, DC, The Brookings Institution, 1988. $29.95 (clothcover)

HEALTH CARE

Health Services Administration: Education and Practice. By Karen M. Lorentzen, RN, EdD, and Linda Roemer, PhD. 118 pp, St Louis, Mo, Warren H. Green, Inc, 1988. $19.50 (softcover)

INTERNAL MEDICINE

Diagnosis and Management of Renal Disease and Hypertension. By Anil K. Mandal, MD, and J. Charles Jennette, MD. 540 pp, illustrated. Philadelphia, Lea & Febiger, 1988. $67.50 (hardcover)

Lipoproteins and Atherosclerosis. (Atherosclerosis Reviews, Volume 17). Edited by Robert I. Levy, MD, Anatoli N. Klimov, MD, Vladimir N. Smirov, MD, and Herman A. Tyroler, MD. 335 pp, illustrated. New York, Raven Press, 1988. $95.00 (hardcover)

Hypercholesterolemia: Clinical and Therapeutic Implications. (Atherosclerosis Reviews, Volume 18). Edited by Joseph Stokes III, MD, and Mario Mancini, MD. 218 pp, illustrated. New York, Raven Press, 1988. $65.00 (hardcover)

MISCELLANEOUS

Doctors. The Biography of Medicine. By Dr Sherwin B. Nuland. 519 pp. New York, Alfred A. Knopf, Publisher, 1988. $24.95 (hardcover)

What Your Doctor Didn't Learn in Medical School. . .and What You Can Do About It! By Stuart M. Berger, MD. 336 pp, illustrated. New York, William Morrow and Company, Inc, 1988. $18.95 (clothcover)

Planning Ahead for Pregnancy: Health, Fitness, and Fertility. 270 pp, illustrated. New York, Penguin Books, 1987. $7.95 (paperback)

House Calls. The Memoirs of a Country Doctor. By Marvin Brown, MD. 214 pp. Buffalo, NY, Prometheus Books, 1988. $19.95 (clothcover)

The Complete Guide to Living with High Blood Pressure. Revised Edition. By Michael K. Rees, MD. 286 pp. New York, Prentice Hall Press, 1988. $10.95 (softcover)

Within Our Reach. Breaking the Cycle of Disadvantage. By Lisbeth B. Schorr. 398 pp, New York, Doubleday, 1988. $19.95 (clothcover)

Medical Abbreviations: 5500 Conveniences at the Expense of Communications and Safety. Fourth Edition. By Neil M. Davis. 139 pp. Huntington Valley, Pa, Neil M. Davis Associates, 1988. $7.95 (paperback)

Worse Than the Disease. Pitfalls of Medical Progress. By Diana B. Dutton. 528 pp. New York, Cambridge University Press, 1988. $29.95 (hardcover)

Remember Me. A Collection of Poems. By Albert D. Anderson, MD. 39 pp. New York, Albert D. Anderson, MD, 1987.

Funny Bones. Rib-tickling Comics from Medical Economics. Edited by James D. Hendricks. 288 pp. Oradell, NJ, Medical Economics Books, 1988. $19.95 (paperback)

The Light Beyond. By Raymond A. Moody, Jr, MD. 176 pp. New York, Bantam Books, 1988. (hardcover)

Loving an Alcoholic. Help and Hope for Co-Dependents. By Jack Mumey. 224 pp. New York, Bantam Books, 1988. $4.50 (paperback)

OBSTETRICS AND GYNECOLOGY

Management of Common Problems in Obstetrics & Gynecology. Second Edition. Edited by Daniel R. Mishell, Jr, MD, and Paul F. Brenner, MD. 699 pp, illustrated. Oradell, NJ, Medical Economics Books, 1988. $39.95 (softcover)

Novak's Textbook of Gynecology. Eleventh Edition. By Howard W. Jones III, MD, Anne Colston Wentz, MD, and Lonnie S. Burnett, MD. 919 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $66.50 (hardcover)

Gynecologic Surgery. Edited by Luis E. Sanz, MD. 450 pp, illustrated. Oradell, NJ, Medical Economics Books, 1988. $57.95 (hardcover)

OCCUPATIONAL MEDICINE

Occupational Medicine: Worker Fitness and Risk Evaluations. Edited by Jay S. Himmelstein, MD, and Glenn S. Pransky, MD. 200 pp, illustrated. Philadelphia, Hanley & Belfus, Inc, 1988. $29.00 (hardcover)

ORTHOPEDIC SURGERY

Clinical Imperatives in Surgery and Orthopedics. Edited by G. F. Dommisse and B. G. P. Lindeque. 450 pp, illustrated. Pretoria, South Africa, Haum Educational Publishers, 1988. $67.00 (hardcover)

PATHOLOGY

Progress in Surgical Pathology. Volume VIII. Edited by Cecilia M. Fenoglio-Preiser, MD, Marianne Wolff, MD, and Franco Rilke, MD. 284 pp, illustrated. New York, Macmillan Publishing Co, 1988. $72.00 (hardcover)

PEDIATRICS

Child Sexual Abuse: A Handbook for Health Care and Legal Professionals. By Diane H. Schetky, MD, and Arthur H. Green, MD. 264 pp. New York, Brunner/Mazel Publishers, 1988. $27.50 (hardcover)

PHYSIOLOGY

Electromyography: Neuromuscular Transmission Studies. By Shin J. Oh, MD. 304 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $49.95 (hardcover)

PSYCHIATRY

Beginning Child Psychiatry. By Paul L. Adams, MD, and Ivan Fras, MD. 620 pp. New York, Brunner/Mazel Publishers, 1988. $50.00 (hardcover)

Practical Clinical Psychopharmacology. Third Edition. By William S. Appleton, MD. 199 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $26.95 (spiral bound)

Developing Ericksonian Therapy: State of the Art. Edited by Jeffrey K. Zeig, PHD, and Stephen R. Lankton, MSW. 556 pp. New York, Brunner/Mazel Publishers, 1988. $50.00 (hardcover)

Diagnostic Issues in Anorexia Nervosa and Bulimia Nervosa. Edited by David M. Garner, PHD, and Paul E. Garfinkel, MD. 228 pp, illustrated. New York, Brunner/Mazel Publishers, 1988. $25.00 (hardcover)

Cognitive-Behavioral Therapy with Families. Edited by Norman Epstein, PHD, Stephen E. Schlesinger, PHD, and Windy Dryden, PHD. 392 pp. New York, Brunner/Mazel Publishers, 1988. $37.50 (hardcover)

Ericksonian Monographs: Number 4. Research Comparisons and Medical Applications of Ericksonian Techniques. Edited by Stephen R. Lankton, MSW, and Jeffrey K. Zeig, PHD. 118 pp. New York, Brunner/Mazel Publishers, 1988. $25.00 (hardcover)

SEXUALLY TRANSMITTED DISEASES

Colour Atlas of Sexually Transmitted Diseases. By Prof. L. K. Bhutani. 160 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, Publishers, 1988. $42.50 (hardcover)

SPORTS MEDICINE

Exercise and Sport Sciences Reviews. Edited by Kent Pandolf, PHD. 551 pp, illustrated. New York, Macmillan Publishing Co, 1988. $48.00 (hardcover)

SURGERY

Atlas of CO_2 Laser Surgical Techniques. Edited by Raymond J. Lanzafame, MD, and J. Raymond Hinshaw, MD. 297 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, Publishers, 1988. $145.00 (hardcover)

Hernia Repair Without Disability. Second Edition. By Irving L. Lichtenstein, MD. 252 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, 1987. $85.00 (hardcover)

Otolaryngology: Head & Neck Surgery: Principles & Concepts. Edited by Jerry Templer, MD, and William E. Davis, MD. 190 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, 1987. $35.00 (hardcover)

Atlas of Surgical Operations. Sixth Edition. By Robert M. Zollinger, Jr, MD, and Robert M. Zollinger, MD. 473 pp, illustrated. New York, Macmillan Publishing Company, 1988. $120.00 (hardcover)

TROPICAL MEDICINE

Manson's Tropical Diseases. Nineteenth Edition. By P. E. C. Manson-Bahr, MD, and D. R. Bell, MB. 1,557 pp, illustrated. Philadelphia, WB Saunders, 1988. $125.00 (hardcover)

FROM THE LIBRARY

THE MEDICAL PROFESSION AS A PUBLIC TRUST

. . . In the present state of medical science in this country, and the subdivision of its departments for special investigation, we see the activity of united efforts combined in organisations and journals specially devoted to such interests; and hence we find, particularly in the great medical centers, a large increase during the past few years of literature devoted to a definite field of labour. Without calling in question the ultimate utility and effect of this multiplication upon the future of the profession, we may rest assured that the fundamental truths of our science will ever remain, and that we may cheerfully welcome every honest labourer into the fields of our science, already "ripe for the harvest."

Our medical journalists have done a great and distinctive service, especially to the younger members of the profession, by inviting and encouraging the frequent expression of original thought for the press.

The systematic and conscientious use of the pen as a means of mental development has long since been fully attested by the recorded experience of the best thinkers and writers of every age. It is the duty, then, of every physician, not only to select as his daily companions medical journals devoted to the interests of the profession at large, and work of the general practitioner, and also some publications designed for special fields of labour as his inclination may dictate, but he should also contribute his mite or of his abundance to their columns, recording his observations, his mature thoughts, his conclusions, and sustaining with generous sympathy every effort of real progress in his profession. . . .

JOHN G. ORTON, MD
(*Trans NY State Med Assoc* 1890; 7:18–36)

Gene technology may help predict risk of breast cancer recurrence

Researchers have discovered a correlation between specific abnormalities in gene structure and an increased risk of recurring breast cancer following surgical removal of the primary tumor (*Science* 1988; 241:535). If clinical studies support this finding, genetic analysis of tumor tissue could be used to predict the prognoses of patients with breast cancer and to decide which patients might best benefit from further preventive treatment, such as chemotherapy.

At the National Cancer Institute, researchers have identified two different genetic alterations in breast cancer cells that correlate with a poor clinical prognosis. One abnormality results in increased activity of the *int-2* oncogene, which codes for a protein that aids in the development of malignant tumors. The second abnormality involves a deletion of genetic material, which researchers hypothesize may include a gene that suppresses tumor growth and malignancy.

Preliminary application of these findings to clinical medicine has yielded conflicting results. One problem is the relatively small number of patients that have been studied. Another possible problem arises due to the mixed composition of tumor tissue—only a percentage of tumor cells are cancerous. Large clinical studies incorporating standardized genetic and clinical procedures are needed to clarify the role of gene analysis in determining breast cancer therapy and prognosis.

Alcohol consumption and breast cancer: Are they related?

An association has been proposed between alcohol consumption and increased risk of breast cancer. However, studies aimed at demonstrating this relationship have yielded inconsistent findings. Longnecker et al (*JAMA* 1988; 260:652–656), from the Harvard School of Public Health (Boston, Mass), performed a meta-analysis, or quantitative review, of existing epidemiologic data in an effort to determine whether there is a dose-response relationship between alcohol consumption and an increased risk of breast cancer.

The data for this study were drawn from a MEDLINE search of the literature for the period 1966–1987, from the references cited in these articles, and from all available abstracts and manuscripts in press at the time of the study. Two investigators then independently evaluated the data contained in the methods and results sections of these 21 documents. Separate analyses were performed for case-control and follow-up studies. The authors used a weighted, least-squares regression model to analyze the data, such that data from larger studies were given greater weight in the overall analysis.

In both the follow-up and case-control studies, the authors found a direct correlation between alcohol consumption and risk of breast cancer. The association was strongest at heavier levels of alcohol intake—$\geq$ 1 oz/day, or about two or more drinks each day. Furthermore, the researchers calculated that "13% of all cases of breast cancer in the United States might be attributable to alcohol consumption."

The paradox is that although moderate alcohol intake—one half to one drink daily—correlates with a slight increase in risk of breast cancer, that quantity of alcohol may have a beneficial effect on one's overall health. Studies have suggested that moderate alcohol consumption is associated with a decreased risk of cardiovascular disease. "One's particular profile of breast cancer and cardiovascular risk factors might influence personal decisions regarding alcohol use," proposed the authors. Finally, it is important to note that although a dose-response relation supports a cause and effect hypothesis, there could be a third factor that influences both the tendency to consume alcohol and the tendency to develop breast cancer. Unfortunately, randomized studies needed to test this phenomenon are not feasible using human subjects.

AIDS antibodies may enhance viral infection

Scientists studying the activity of human immunodeficiency virus (HIV), which causes AIDS, have found that some antibodies against HIV, which normally hinder infection, may actually enhance infection (*Science* 1988; 241:533–534). Although this finding is still in question and, clearly, confirmatory studies are needed, the possibility of antibody-enhanced infection could have a negative impact on efforts to develop an AIDS vaccine. The vaccine would immunize individuals by stimulating the production of antibodies against AIDS.

Further studies of this phenomenon will focus on three main issues: whether antibodies to HIV actually enhance viral activity; the mechanism behind such antibody activity; and the clinical implications of antibody-enhanced infection. As yet, only in vitro studies have demonstrated this phenomenon.

Preliminary findings suggest that monocytes and macrophages, in particular, are affected by the infection-enhancing effect of the antibodies. Recent research findings suggest that these two blood cells may come under attack early in the course of HIV infection and may become "virus-producing factories in the body."

Unraveling the mystery of antibody-enhanced infection is a complex problem. For example, in vitro studies have shown that a single sample of human serum can neutralize HIV, enhance infection, or do both, depending on the viral isolate. Researchers are suggesting that volunteers involved in clinical trials of experimental AIDS vaccines should provide blood samples, which can then be tested in vitro for their ability to increase the rate or extent of HIV infection.

A survey of needle-stick injuries

The risk of acquiring AIDS as the result of a needle-stick injury has heightened interest in the causes, outcomes, and means of preventing such injuries. The Centers for Disease Control has issued guidelines aimed at

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NEWS BRIEFS are compiled and written by Vicki Glaser.

reducing the incidence of needle-stick injuries, and most hospitals have adopted needle-stick prevention policies, often including employee education programs. Jagger et al (*N Engl J Med* 1988; 319:284–287) reviewed needle-stick injuries caused by various devices in one university hospital. They observed that "the potential medical and psychological consequences of needle sticks for health care workers and their spouses or sexual partners remain great." Furthermore, they cited studies that have demonstrated the transmission of more than 20 pathogens via needle-stick injuries. In this study, the authors identified the types and designs of devices that most often cause needle-stick injuries, and they described the mechanisms of the injuries. They supported the need for alternative injection devices, as well as a new design for devices that require needles, which would include a cover or barrier to prevent unintended contact with a contaminated needle.

The study design established the following: "Each employee who reported a needle-stick injury . . . was interviewed by a trained interviewer according to a standard format to determine the nature of the device that caused the injury, the procedure for which it was used, and the setting and mechanism of the injury . . . and the outcome of the injury." Only injuries caused by hollow-bore needles or unidentified devices believed to be needles were reviewed in this study.

The hospital had advised employees not to recap, bend, or break used needles. Nurses' stations housed special disposal containers for sharp objects.

The 326 needle-stick injuries reported during the study period affected a range of hospital personnel: nurses and nursing students, 64%; laboratory technicians and auxiliary personnel in radiology and respiratory therapy, 20%; housekeeping personnel, 8%; attendants, 4%; and physicians and medical students, 3%. No infections were associated with these 326 incidents. The needle-stick rate was based on the number of injuries with a specific device and the quantity of that device purchased by the hospital during the study period.

The results showed that disposable syringes caused the most injuries but had the lowest needle-stick rate. Intravenous tubing and needle assemblies, which require disassembly, had the highest rate. Seventeen percent of injuries occurred while the employee was using the devices, 70% during preparation of the devices for disposal, and 13% during or after disposal. Recapping needles accounted for one third of all injuries. These injuries occurred due to the user's missing the cap during recapping, the needle's piercing the cap, or the cap's falling off an already recapped needle. Inappropriate disposal of used needles led to injuries among housekeeping personnel.

The authors concluded that "our data indicate that there is no single remedy for needle-stick injuries. Each device that causes such injuries must be modified according to its specific use and handling requirements." They stressed the need to reduce the incentives for recapping needles. Employees reported the importance of recapping contaminated needles to protect themselves from exposed needles while disassembling a device or transporting items to an appropriate disposal container, to store a syringe between multiple uses, and to protect fellow employees.

Possible solutions to the continuing occurrence of needle-stick injuries include alternative injection methods, such as a compressed air system, in-line stopcocks for administering medication through an intravenous system, or newly designed injection devices with safety features that prevent user contact with the needle during and after use. The safety features should be an integral part of the device and should be simple to operate, requiring little training or experience.

The authors viewed the need for a safer work environment due to the risk of AIDS and other infectious disease transmission as "a historic opportunity to accelerate the transfer of new technology into the work place."

Retroactive chart review required in three New York State counties to uncover cases of Lyme disease

In August 1988 a new §206-b was added to the New York State Public Health Law to address the need to identify treatable Lyme disease in individuals previously diagnosed as having juvenile rheumatoid arthritis (JRA).

In accordance with the provisions of Public Health Law §206-b, a physician practicing in Suffolk, Westchester, or Nassau County, upon receipt of a notice mailed in August 1988, is required to review the medical records of any patient treated since January 1, 1975, with a diagnosis of JRA. The law requires the physicians to review the circumstances of the earlier diagnosis and to consider whether the patient has suffered or is suffering from the complex, multisystem disorder caused by the bacterium *Borrelia burgdorferi*, commonly referred to as Lyme disease. Physicians are urged to pay particular attention to individuals whose illness might have been characterized by the following ICDA-9 codes:

711	Arthropathy associated with infections.
712	Crystal arthropathies.
713	Arthropathy associated with other disorders classified elsewhere.
714	Rheumatoid arthritic and other inflammatory polyarthropathies.
714.3	JRA.
715	Osteoarthritis and allied disorders.
716	Other unspecified arthropathies.

If a hospital or other health care institution or provider has custody or control of the medical records for a patient so diagnosed, the physician shall be entitled to review such medical records for purposes of complying with the law. Under the new law, if a review of any of these records indicates that a patient may have been or is suffering from Lyme disease, the physician shall, consistent with customary and acceptable medical standards, take such action as he or she deems necessary to inform the patient, or a minor patient's parent or guardian, of the findings, conduct additional tests, administer other necessary treatments, or refer such patient for further diagnosis and/or treatment.

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Ben Wolfe Aberman, MD, East Hampton. Died July 1, 1988; age 77. Dr Aberman was a 1936 graduate of the Medizinische Fakultaet der Ludwig Maximiliams Universitaet, Muenchen, Bayern, Germany. He was a member of the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Abdul Hamid Alizadeh, MD, Brooklyn. Died July 20, 1987; age 57. Dr Alizadeh was a 1957 graduate of the Medizinische Fakultaet der Universitaet Hamburg, Hamburg, Germany. He was a Qualified Fellow of the International College of Surgeons. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Norman Charles Bender, MD, Getzville. Died June 5, 1988; age 93. Dr Bender was a 1919 graduate of the University of Michigan Medical School, Ann Arbor, Michigan. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. His memberships included the New York Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Benjamin J. Ciliberti, MD, Boynton Beach, Florida. Died April 19, 1988; age 79. Dr Ciliberti was a 1935 graduate of the St Louis University School of Medicine, St Louis, Missouri. He was a Diplomate of the American Board of Anesthesiology and a Fellow of the American College of Anesthesiologists. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Samuel Lewis Cohen, MD, New York. Died March 7, 1988; age 92. Dr Cohen was a 1919 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Cardiology. His memberships included the New York Cardiological Society, the New York State Society of Internal Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Anthony Lewis Davidson, MD, Brooklyn. Died July 11, 1988; age 43. Dr Davidson was a 1972 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Marion Thomas Donato, MD, Buffalo. Died May 15, 1988; age 75. Dr Donato was a 1936 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the Medical Society of the County of Erie and and the Medical Society of the State of New York.

Milton Dorfman, MD, Rome. Died July 3, 1988; age 69. Dr Dorfman was a 1943 graduate of the George Washington University School of Medicine, Washington, DC. He was a Diplomate of the American Board of Radiology and a Fellow of the American College of Radiology. His memberships included the Radiological Society of New York State, the Radiological Society of North America, Inc, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Clarence Falk Friedman, MD, Sarasota, Florida. Died July 13, 1988; age 90. Dr Friedman was a 1921 graduate of the Vanderbilt University School of Medicine, Nashville, Tennessee. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Alexander A. Gaines, MD, Hallandale, Florida. Died June 1, 1988; age 86. Dr Gaines was a 1925 graduate of the New York University School of Medicine, New York. He was a Diplomate of the American Board of Radiology and a Fellow of the American College of Angiology. His memberships included the Pan American Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Jacob R. Ginsberg, MD, Jamaica, New York. Died June 28, 1988; age 83. Dr Ginsberg was a 1930 graduate of the New York Medical College, New York. He was a member of the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

George Gorin, MD, New York. Died July 13, 1988; age 79. Dr Gorin was a 1935 graduate of the Faculte de Medecine de l'Universite de Geneve, Geneva, Switzerland. Dr Gorin was a Diplomate of the American Board of Ophthalmology and a Fellow of the American College of Surgeons. His memberships included the New York Society for Clinical Ophthalmology, the American Academy of Ophthalmology and Otolaryngology, the New York County Medical Society, and the Medical Society of the State of New York.

Ralph Berthold Jacoby, MD, Lake Worth, Florida. Died November 8, 1987; age 73. Dr Jacoby was a 1938 graduate of the Medizinische Fakultaet der Universitaet Bern, Bern, Switzerland. Dr Jacoby was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the Academy of Psychoanalysis, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Charles H. Kinley, MD, Elmira. Died

June 21, 1988; age 68. Dr Kinley was a 1946 graduate of the University of Pennsylvania School of Medicine, Philadelphia. Dr Kinley was a Fellow of the American College of Emergency Physicians. He was a member of the Medical Society of the County of Chemung and the Medical Society of the State of New York.

Aaron Leifer, MD, New York. Died July 4, 1988; age 87. Dr Leifer was a 1923 graduate of the New York University School of Medicine, New York. Dr Leifer was a member of the American Public Health Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Henry Mednick, MD, Lynbrook. Died July 11, 1988; age 67. Dr Mednick was a 1945 graduate of the New York University School of Medicine, New York. His memberships included the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Bernard J. Mintz, MD, New York. Died June 23, 1988; age 85. Dr Mintz was a Diplomate of the American Board of Orthopedic Surgery. His memberships included the New York Academy of Medicine, the American Academy of Orthopaedic Surgeons, the New York County Medical Society, and the Medical Society of the State of New York.

Thomas Main Proctor, MD, Live Oak, Florida. Died March 18, 1988; age 91. Dr Proctor was a 1925 graduate of the Cornell University Medical College, New York. He was a member of the Dutchess County Medical Society and the Medical Society of the State of New York.

Isaac Skir, MD, Brooklyn. Died July 10, 1988; age 91. Dr Skir was a 1922 graduate of the New York University School of Medicine, New York. He was a Diplomate of the American Board of Colon and Rectal Surgery and a Qualified Fellow of the International College of Surgeons. His memberships included the American Society of Colon and Rectal Surgeons, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Stanley L. Wang, MD, New York. Died January 5, 1988; age 99. Dr Wang was a 1912 graduate of the University of Oregon Medical School, Portland. He was a Fellow of the American College of Chest Physicians. His memberships included the American Urological Association, the New York County Medical Society, and the Medical Society of the State of New York.

Charles J. Woeppel, MD, Buffalo. Died June 9, 1988; age 74. Dr Woeppel was a 1937 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456–462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

FOR MY PATIENTS

NUMBER 3/1988

KNOW YOUR CHOLESTEROL

Dear Patient,

Among men 35 to 74 years of age, about 28% have high levels of cholesterol. Among women in the same age group, about 32% have high levels, according to a recent national survey.

Cholesterol is a fat-like chemical substance found in the blood and tissues of many organs. Most of the body's cholesterol is made in the liver, but a large portion is obtained from the food entering the stomach. Eating fatty foods such as butter, ice cream and meats tends to raise the level of cholesterol in the blood. Although some people inherit a tendency to develop higher levels than others, diet is an important factor.

The total amount of cholesterol circulating in the blood contains both "good" cholesterol (HDL) and "bad" cholesterol (LDL). Therefore, the aim is to raise the HDL part and lower the LDL part.

DOING ITS DAMAGE

High cholesterol in the blood gradually forms arteriosclerotic deposits (plaques) along the inside walls of the arteries ("hardening of the arteries"). As time goes on, these arteries become narrowed by the buildup of these plaques. Circulation to the vital organs of the body becomes blocked. The next event can be damage to the heart (coronary disease or "heart attack") or to the brain (cerebrovascular accident or "stroke").

KNOW YOUR RISK FACTORS

If you have *two or more* of these risk factors:

- ☐ smoking
- ☐ high cholesterol
- ☐ high blood pressure
- ☐ overweight
- ☐ diabetes
- ☐ family history of early "heart attacks"

your chances of developing these heart and brain conditions are increased 300% or more. The more of these risk factors you have, the greater are your chances.

REASON FOR TESTING

Your cholesterol level is a sensitive indicator or clue to the amount of arteriosclerosis built up in your arteries. If it is too high, it is a danger signal that you may be on your way to coronary heart disease or stroke. Steps can then be taken immediately to reduce this high level and prevent these damages. If your first cholesterol level is 200 or greater, it is a good idea to repeat the test at least twice more over the next couple of weeks at the same laboratory. By taking the average of all 3 readings, you can more accurately find your true level.

WATCH THESE NUMBERS

The ***desirable levels are less than 200*** (milligrams per deciliter). If you are in these levels, recheck it every 5 years at least.

The ***borderline high levels are 200-239.*** If you are in these levels, recheck it ***each*** year. If you are in these levels ***and*** have any other risk factors listed above, consult me as soon as possible. Special low-fat diet, exercise program, or drugs may be recommended for you.

The ***high levels are over 240.*** If you are in these levels you may need other blood tests to see how much of your total cholesterol is of the "good" type (HDL)and how much is of the "bad" type (LDL). Consult me immediately so that special low-fat diet, exercise program, or drugs may be prescribed for you. The level should be rechecked frequently.

The goal for most adults is to bring their cholesterol level down to 200 or less and keep it there continuously.

BENEFIT OF LOWERING THE LEVEL

The National Institute of Health has stated that for every 25% drop in blood cholesterol level, there will be a 50% cut in the chances of having a heart attack. The moral of the story is that checking your blood cholesterol is like taking the pulse of your own life.

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This issue of the *New York State Journal of Medicine* contains a special additional item, "For My Patients." This feature will be run in future issues as well.

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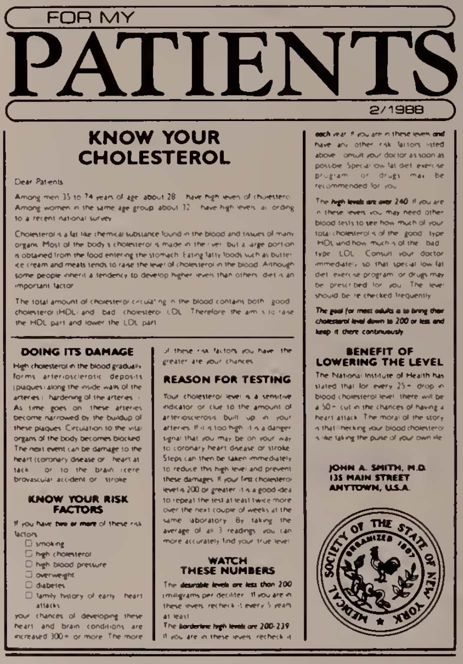

FOR MY

PATIENTS

2/1988

KNOW YOUR CHOLESTEROL

Dear Patients

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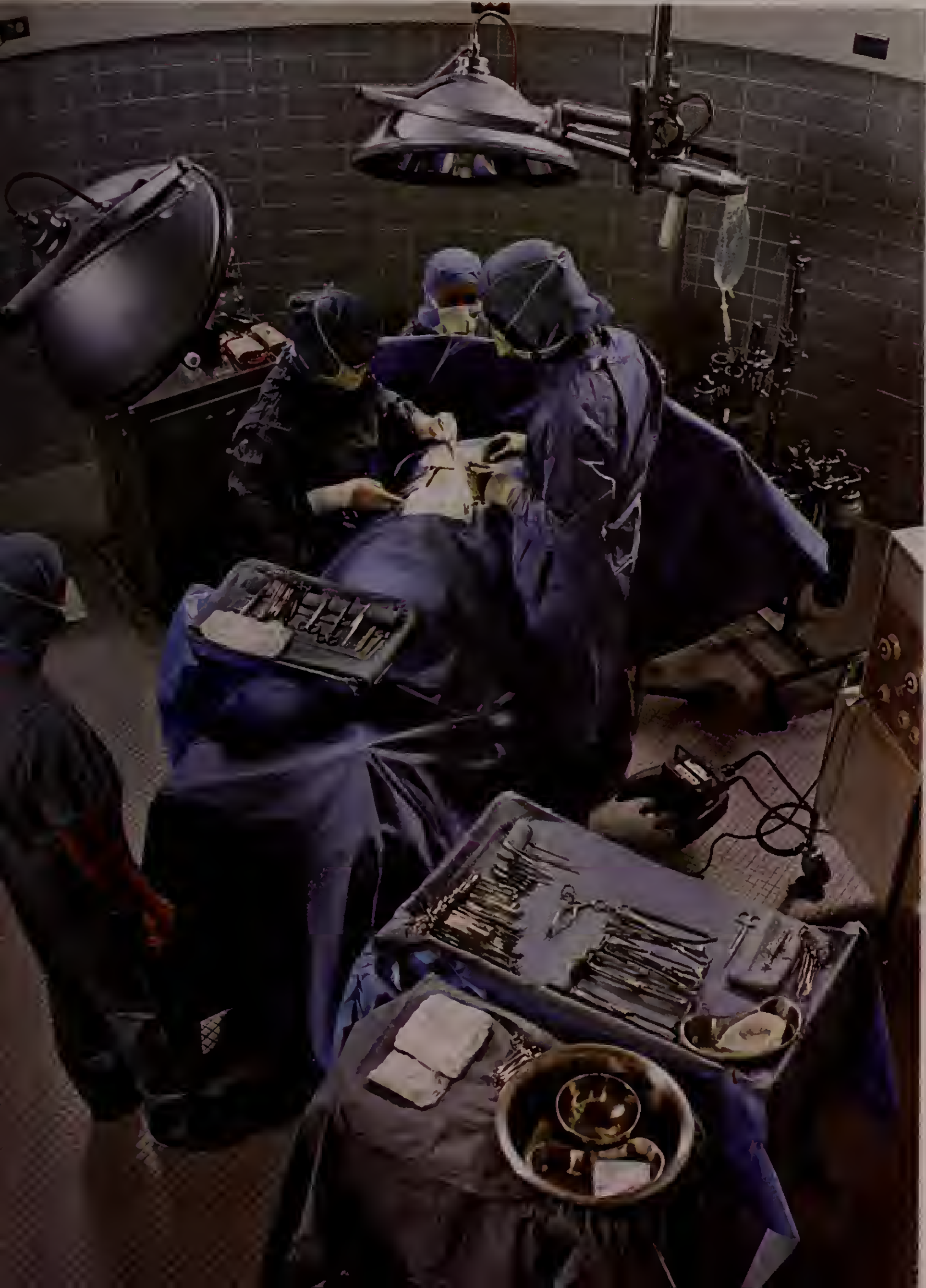

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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Nov 19. **Second Annual Course on Common Orthopedic Problems in Children.** 7 Cat I Credits. Schneider Children's Hospital. Contact: Ann J. Boehme, Associate Director for Continuing Education, Schneider Children's Hospital of Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

AROUND THE NATION

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Nov 4–6. **Eighth Annual Interventional Course.** 17½ Cat I Credits. Hotel del Coronado, Coronado (San Diego), California. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

Nov 11–14. **The Spine: Current Concepts of Diagnosis & Treatment.** Marriott Desert Springs Resort, Palm Springs. Contact: Spine Study Group, PO Box 33185, San Diego, CA 92103. Tel: (619) 692-9115.

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Nov 19–20. **1988 Regional Review Courses in Hand Surgery.** 12 Cat I Credits. Washington. Contact: American Society for Surgery of the Hand, 3025 S Parker Rd, Suite 65, Aurora, CO 80014. Tel: (303) 755-4588.

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(*continued on p 24A*)

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Nov 16–18. **a-Keto Acid Dehydrogenase Complexes.** Austin. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021.

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AROUND THE WORLD

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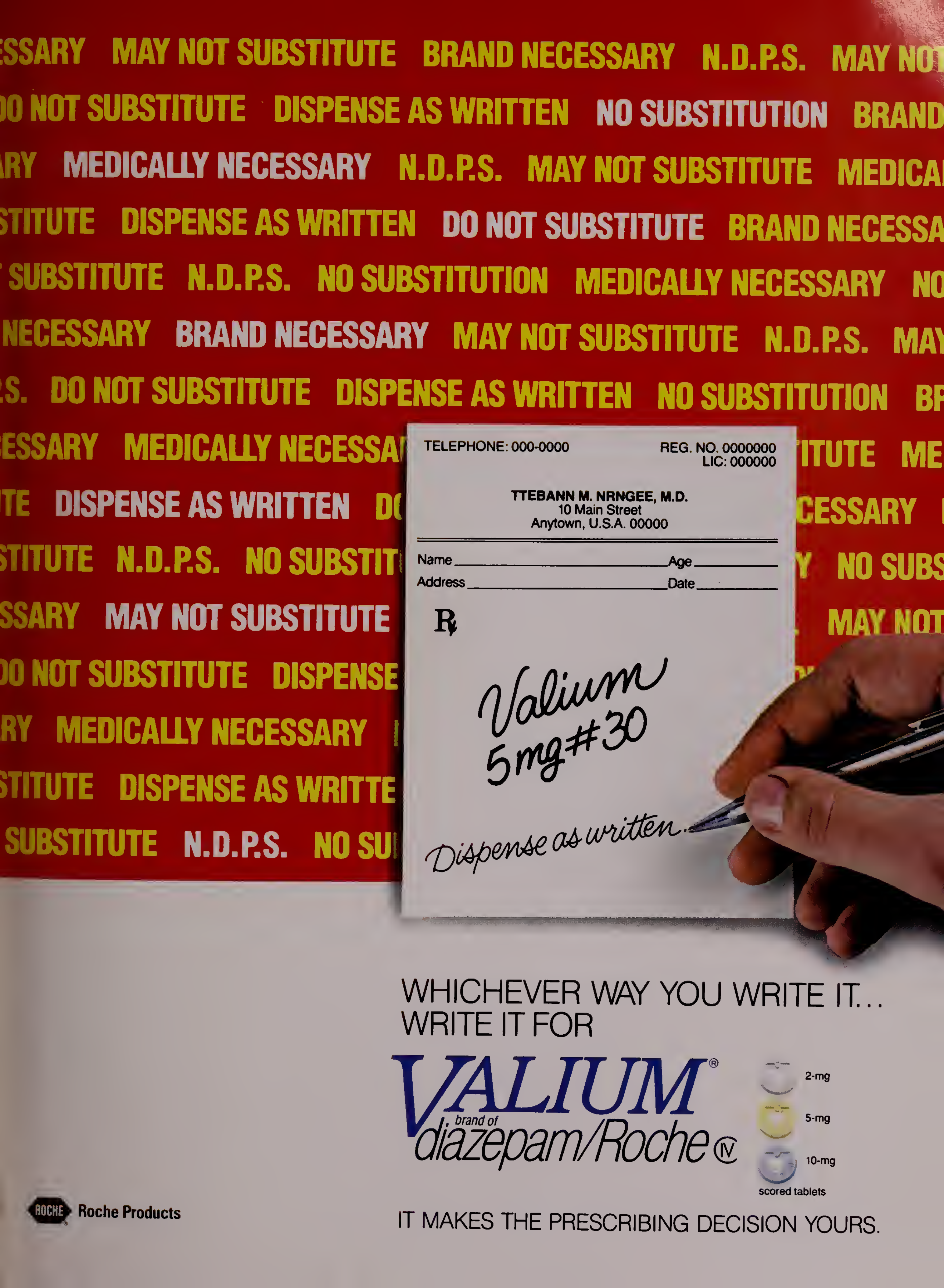
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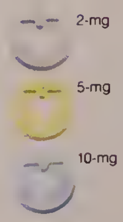

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Brief Summary

CLINICAL PHARMACOLOGY: Pharmacodynamics: Clinical studies of terazosin used in once a day (majority) and b.i.d regimens with total doses usually in the range of 5-20mg/day, in patients with mild or moderate hypertension. Because terazosin, like all alpha antagonists, can cause large falls in blood pressure after the first dose or first few doses, the initial dose was 1mg in virtually all studies, with subsequent titration to a specified fixed dose or titration to a specified blood pressure end point.

Blood pressure responses were measured at the end of the dosing interval (usually 24 hrs.) and effects were shown to persist throughout the interval, with usual supine responses 5-10mmHg systolic and 3.5-8mmHg diastolic greater than placebo. The responses in the standing position tended to be somewhat larger, although this was not true in all studies. The magnitude of blood pressure responses was similar to prazosin and less than hydrochlorothiazide (in a single study). In measurements 24 hrs. after dosing, heart rate was unchanged

Limited measurements of peak response (2-3 hrs. after dosing) during chronic terazosin administration indicate that it is more than twice the trough (24 hr.) response, suggesting some attenuation of response at 24 hrs., presumably due to a fall in blood terazosin concentrations at the end of the dose interval. This explanation is not established with certainty and is not consistent with the similarity of blood pressure response to once-a-day and b.i.d dosing. With the absence of an observed dose-response relationship over a range of 5-20mg, i.e., if blood concentrations fall to the point of providing less than full effect at 24 hrs., a shorter dosing interval or larger dose should lead to increased response. Measure blood pressure (BP) at the end of the dose interval, if response is not satisfactory, patients may be tried on a larger dose or b.i.d. regimen. The latter should be considered if side effects, such as dizziness, palpitations, or orthostatic complaints, are seen within a few hours after dosing

The greater BP effect associated with peak plasma concentrations (first few hours after dosing) appears somewhat more position-dependent (greater in the erect position) than the effect of terazosin at 24 hrs. In the erect position there is a 6-10 bpm increase in heart rate in the first few hours after dosing. During the first 3 hrs. after dosing 12.5% of patients had a systolic pressure fall of 30mmHg or more from supine to standing, or standing systolic pressure below 90mmHg with a fall of at least 20mmHg, compared to 4% of a placebo group

INDICATIONS AND USAGE: Indicated for the treatment of hypertension

CONTRAINDICATIONS: None known.

WARNINGS: Syncope and "First-dose" Effect: Terazosin, like other alpha-adrenergic blocking agents, can cause marked hypotension, especially postural hypotension, and syncope in association with the first dose or first few doses. A similar effect may occur if therapy is interrupted for more than a few doses. Syncope has been reported with other alpha-adrenergic blocking agents in association with rapid dosage increases or introduction of another antihypertensive drug. Syncope may be due to an excessive postural hypotensive effect, although occasionally the syncopal episode has been preceded by severe supraventricular tachycardia with heart rates of 120-160 bpm.

To decrease the likelihood of syncope or excessive hypotension, always initiate treatment with a 1mg dose at bedtime. The 2mg and 5mg tablets are not indicated as initial therapy. Increase dosage slowly, and add additional antihypertensive agents with caution. Caution patients to avoid situations where injury could result if syncope occurs during initiation of therapy.

In early studies, where increasing single doses up to 7.5mg were given at 3 day intervals, tolerance to the first dose phenomenon did not necessarily develop and the "first dose" effect was observed at all doses. Syncopal episodes occurred in 3 of 14 subjects given doses of 2.5, 5, and 7.5mg, which are higher than the recommended initial dose. Severe orthostatic hypotension (BP 50/0mmHg) was seen in two others and dizziness, tachycardia, and lightheadedness occurred in most subjects. These adverse effects all occurred within 90 min. of dosing

In multiple dose clinical trials involving nearly 2000 patients, syncope was reported in about 1% of patients, in no case severe or prolonged, and was not necessarily associated with early doses

If syncope occurs, place patient in recumbent position and treat supportively. There is evidence that the orthostatic effect of terazosin is greater, even in chronic use, shortly after dosing.

PRECAUTIONS: *General. Orthostatic Hypotension:* While syncope is the most severe orthostatic effect of terazosin, other symptoms of lowered BP, such as dizziness, lightheadedness and palpitations, are more common, occurring in 28% of patients in clinical trials. Patients with occupations in which such events represent potential problems should be treated with particular caution.

Information for Patients: Make aware of possibility of syncopal and orthostatic symptoms, especially at initiation of therapy, and to avoid driving or hazardous tasks for 12 hrs. after the first dose, after a dosage increase, and after interruption of therapy when treatment is resumed. Caution to avoid situations where injury could result should syncope occur during initial therapy. Advise to sit or lie down when symptoms of lowered BP occur and to rise carefully from a sitting or lying position. Bothersome dizziness, lightheadedness, or palpitations should be reported to physician.

Tell patients that drowsiness or somnolence can occur, requiring caution in people who must drive or operate heavy machinery

Laboratory Tests: Small but statistically significant decreases in hematocrit, hemoglobin, WBC, total protein and albumin were observed in clinical trials. The magnitude of decreases did not worsen with time. These findings suggest the possibility of hemodilution

Drug Interactions: In controlled trials, terazosin was added to diuretics, and several beta adrenergic blockers, no unexpected interactions were observed. Terazosin has also been used concomitantly without interaction in at least 50 patients on the following: 1) analgesic/anti-inflammatory (acetaminophen, aspirin, codeine, ibuprofen, indomethacin), 2) antibiotics (erythromycin, trimethoprim and sulfamethoxazole), 3) anticholinergic/sympathomimetics (phenylephrine HCl, phenylpropanolamine HCl, pseudoephedrine HCl), 4) antigout (allopurinol), 5) antihistamines (chlorpheniramine), 6) cardiovascular agents (atenolol, hydrochlorothiazide, methyclothiazide, propranolol), 7) corticosteroids, 8) gastrointestinal agents (antacids), 9) hypoglycemics, 10) sedatives and tranquilizers (diazepam).

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HYTRIN, administered in feed to rats at doses of 8, 40, and 250mg/kg/day for 2 yrs., was associated with a statistically significant increase in benign adrenal medullary tumors of male rats exposed to the 250mg/kg dose. This dose is 695 X max. recommended human dose (20mg/55kg). Female rats were unaffected. HYTRIN was not oncogenic in mice when administered in feed for 2 yrs. at a maximum tolerated dose of 32mg/kg/day

The absence of mutagenicity in a battery of tests, of tumorigenicity of any cell type in the mouse carcinogenicity assay, of increased total tumor incidence in either species, and of proliferative adrenal lesions in female rats, suggests a male rat species-specific event. Numerous other diverse pharmaceutical and chemical compounds have been associated with these tumors in male rats without supporting evidence for carcinogenicity in man.

Effects on fertility were assessed in a standard fertility/reproductive performance study in which male and female rats were administered oral doses of 8, 30 and 120mg/kg/day. Four of 20 male rats given 30mg/kg and 5 of 19 male rats given 120mg/kg failed to sire a litter. Testicular weights and morphology were unaffected. Vaginal smears at 30 and 120mg/kg/day appeared to contain less sperm than smears from control matings and good correlation was reported between sperm count and subsequent pregnancy

Oral use for 1 or 2 yrs. elicited a statistically significant increase in testicular atrophy in rats exposed to 40 and 250mg/kg/day, but not in rats exposed to 8mg/kg/day (> 20 X max. recommended human dose). Testicular atrophy was observed in dogs dosed with 300mg/kg/day (> 800 X max. recommended human dose) for 3 months but not after 1 yr. when dosed with 20mg/kg/day. This lesion has also been seen with Minipress®

Pregnancy: Teratogenic effects. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women and the safety of terazosin in pregnancy has not been established. HYTRIN is not recommended during pregnancy unless potential benefit justifies potential risk to mother and fetus.

Nonteratogenic effects: In a peri- and post-natal development study in rats, significantly more pups died in the group dosed with 120mg/kg/day (> 300 X max. recommended human dose) than in the control group during the 3-week post-partum period

Nursing Mothers: It is not known whether terazosin is excreted in breast milk, therefore, exercise caution when administering terazosin to a nursing woman.

Pediatric Use: Safety and effectiveness have not been determined.

ADVERSE REACTIONS: The prevalence of adverse reactions has been ascertained from 14 placebo-controlled studies conducted primarily in the U.S. The studies involved once-a-day administration of terazosin as monotherapy or in combination with other antihypertensive agents, at doses ranging from 1 to 40mg. All adverse events reported during these studies were recorded as adverse reactions. Adverse events where the prevalence rate in the terazosin group was at least 5%, where the prevalence rate for the terazosin group was at least 2% and was greater than the prevalence rate for the placebo group, or where the reaction is of particular interest are summarized below. Only asthenia, blurred vision, dizziness, nasal congestion, nausea, peripheral edema, palpitations and somnolence were significantly ($p < 0.05$) more common in patients receiving terazosin than in patients receiving placebo. Other events include **[%TERAZOSIN-%PLACEBO]:** asthenia (11.3%-4.3%), back pain (2.4%-1.2%), blurred vision (1.6%-0%), depression (0.3%-0.2%), dizziness (19.3%-7.5%), dyspnea (3.1%-2.4%), edema (0.9%-0.6%), headache (16.2%-15.8%), impotence (1.2%-1.4%), libido decreased (0.6%-0.2%), nasal congestion (5.9%-3.4%), nausea (4.4%-1.4%), nervousness (2.3%-1.8%), pain-extremities (3.5%-3%), palpitations (4.3%-1.2%), paresthesia (2.9%-1.4%), peripheral edema (5.5%-2.4%), postural hypotension (1.3%-0.4%), sinusitis (2.6%-1.4%), somnolence (5.4%-2.6%), tachycardia (1.9%-1.2%), weight gain (0.5%-0.2%)

Adverse reactions were usually mild or moderate in intensity but sometimes were serious enough to interrupt treatment. Adverse reactions that were most bothersome as judged by being reported as reasons for discontinuation of therapy by at least 0.5% of the terazosin group and being reported more often than in the placebo group **[%TERAZOSIN-%PLACEBO]** are: asthenia (1.6%-0%), blurred vision (0.6%-0%), dizziness (3.1%-0.4%), dyspnea (0.9%-0.6%), headache (1.3%-1%), nasal congestion (0.6%-0%), nausea (0.8%-0%), palpitations (1.4%-0.2%), paresthesia (0.8%-0.2%), peripheral edema (0.6%-0%), postural hypotension (0.5%-0%), somnolence (0.6%-0.2%), syncope (0.5%-0.2%), tachycardia (0.6%-0%)

Additional adverse reactions have been reported, but these are not distinguishable from symptoms that might have occurred in the absence of exposure to terazosin. The following additional adverse reactions were reported by at least 1% of 1987 patients who received terazosin in clinical studies or during marketing experience: abdominal pain, abnormal vision, anxiety, arrhythmia, arthralgia, arthritis, bronchitis, chest pain, cold symptoms, conjunctivitis, constipation, diarrhea, dry mouth, dyspepsia, epistaxis, facial edema, fever, flatulence, flu symptoms, gout, increased cough, insomnia, joint disorder, myalgia, neck pain, pharyngitis, pruritus, rash, rhinitis, shoulder pain, sweating, tinnitus, urinary frequency, urinary tract infection, vasodilation, vomiting

DOSAGE AND ADMINISTRATION: Dose and dose interval (12 or 24 hrs.) should be adjusted according to BP response

Initial Dose: 1mg at bedtime. Observe the initial dosing regimen strictly to minimize potential for severe hypotensive effects

Subsequent Doses: Slowly increase dose to achieve desired BP response. Usual dose range is 1mg to 5mg once a day. Some patients may benefit from doses up to 20mg/day. Doses over 20mg do not appear to provide further BP effect. Doses over 40mg have not been studied. Monitor BP at the end of dosing interval to assure control is maintained. It may be helpful to measure BP 2-3 hrs. after dosing to see if maximum and minimum responses are similar, and to evaluate symptoms which can result from excessive hypotensive response. If response is substantially diminished at 24 hrs. consider an increased dose or b.i.d. regimen. **If administration is discontinued for several days or longer, reinstitute therapy using initial dosing regimen.** In clinical trials, except for the initial dose, the dose was given in the morning

Use With Other Drugs: Caution should be observed when terazosin is administered concomitantly with other antihypertensive agents (e.g., calcium antagonists) to avoid the possibility of significant hypotension. When adding a diuretic or other antihypertensive agent, dosage reduction and retitration may be necessary

August, 1988 **Abbott Health Care Products, Inc.** North Chicago, IL 60064 8083873

References: 1. HYTRIN Product Information, Abbott Laboratories. 2. Deger G: Effect of terazosin on serum lipids. *Am J Med* 1986;80(suppl 5B):82-85. 3. Dzau VJ: Evolution of the clinical management of hypertension: Emerging role of "specific" vasodilators as initial therapy. *Am J Med* 1987;82(suppl 1A):36-43. 4. Weinberger MH: The effects of antihypertensive therapy on lipids. *Cardiovasc Med* 1986;11:10-11. 5. Ames RP: The effects of antihypertensive drugs on serum lipids and lipoproteins. II. Non-diuretic drugs. *Drugs* 1986;32:335-357.

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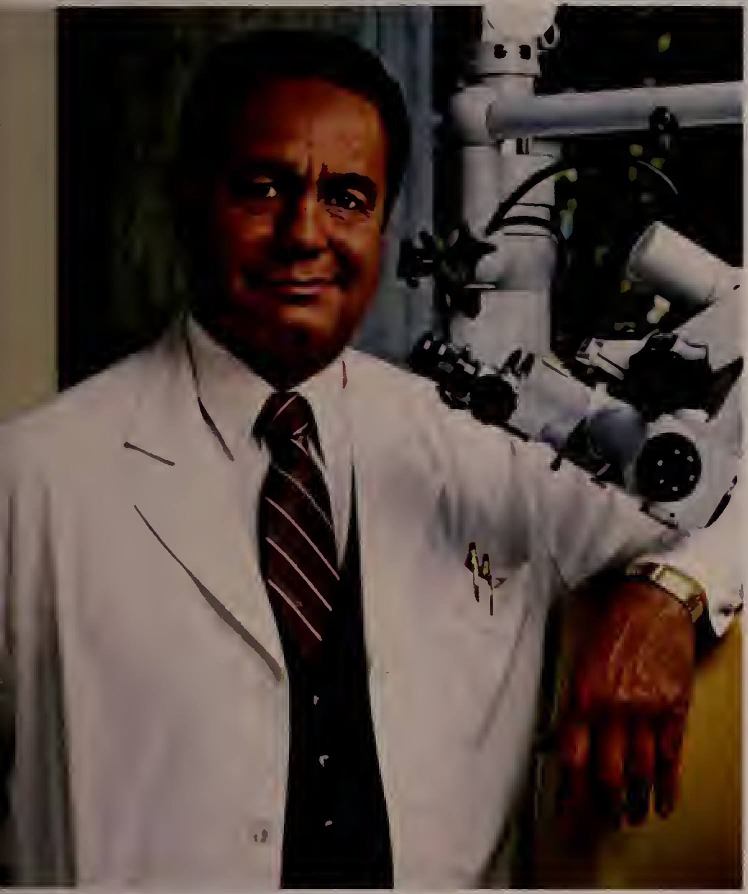

Dr. Tipton and residents examining post-operative patient in recovery room.

DALE L. TIPTON, M.D.

Associate Clinical Professor, Department of Otolaryngology, Head and Neck Surgery, University of California School of Medicine, San Francisco, California.
Chairman, Division of Otolaryngology, Franklin Hospital, San Francisco, California.
Lieutenant Colonel, U.S. Army Reserve.

EDUCATION University of California at Berkeley, A.B. Physiology; University of California School of Medicine, San Francisco, M.D. and Master of Science, Pharmacology.

RESIDENCY University of California School of Medicine, San Francisco: General Surgery—2 years; Otolaryngology—3 years.

FELLOWSHIPS National Institute of Health Fellow; Cancer Research Institute, University of California, San Francisco.

OUTSTANDING ACHIEVEMENTS Freshman Medical Student Research Award; Class President—2nd year medical school; Student Body President—senior year medical school; Special Award by National Institute of Health to attend and present paper at International Congress of Otolaryngology in Tokyo, Japan; Chairman, Department of Otolaryngology, San Francisco General Hospital 1970-76; Chief of Medical Staff, Franklin Hospital 1982-84.

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References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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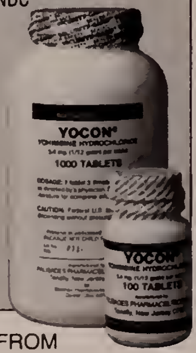

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AROUND THE STATE

BRONX

Dec 7. **A Conceptual Model for Forensic Psychiatry.** Lehman College. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 130 Gold Pl, Malverne, NY 11565. Tel: (516) 887-4691.

GARDEN CITY

Dec 1. **Growth and Growth Hormone.** Garden City Hotel. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Schneider Children's Hospital of Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

MANHATTAN

Dec 1. **Ventricular Hypertrophy: Good or Bad?** New York Helmsley Hotel. 2 Cat I Credits. Contact: The New York Cardiological Society, Inc, 84 Grove St, Suite Two, New York, NY 10014. Tel: (212) 924-5346.

Dec 2–3. **Application of New Techniques to Diagnostic Pathology.** 14 Cat I Credits. *Dec 10–11.* **Review of GU Pathology—Workshop for Urologists.** 16 Cat I Credits. NYU Medical Center. *Dec 12–17.* **CT and MRI—Head to Toe.** 40 Cat I Credits. Grand Hyatt Hotel. *Dec 14–16.* **Managing Clinical Problems in the Elderly.** 19½ Cat I Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Dec 2–3. **Immunologic Mechanisms in Neurologic and Psychiatric Disease.** Roosevelt Hotel. Contact: Ivan Bodis-Wollner, MD, Secretary-Treasurer, Association for Research in Nervous and Mental Diseases, Box 1052, Mt Sinai

Hospital, 1 Gustave Levy Pl, New York, NY 10029.

Dec 8–9. **Update in Gastroenterology & Hepatology.** 15 Cat I Credits. Columbia University. Contact: Center for Continuing Education, 630 First Ave, New York, NY 10032. Tel: (212) 305-3682.

Dec 9. **New Concepts in Medicine and Biology. A Symposium Honoring the Memory of Hans Popper, MD, PhD.** Mt Sinai School of Medicine. Contact: Swan N. Thung, MD, Department of Pathology, Mt Sinai School of Medicine, One Gustave L. Levy Pl, New York, NY 10029. Tel: (212) 241-9139.

Dec 9–11. **Acupuncture and Electro-Therapeutics in Clinical Practice.** 25 Cat I Credits. New York City. Contact: Y. Omura, MD, ScD, 800 Riverside Dr, (8-1), New York, NY 10032. Tel: (212) 781-6262.

Dec 12–14. **Regulation of the Acute Phase and Immune Responses: A New Cytokine.** Sheraton Centre. *Dec 15–18.* **Mechanisms & Regulation of Anion & Proton Transport.** Sheraton Centre. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021.

AROUND THE NATION

CALIFORNIA

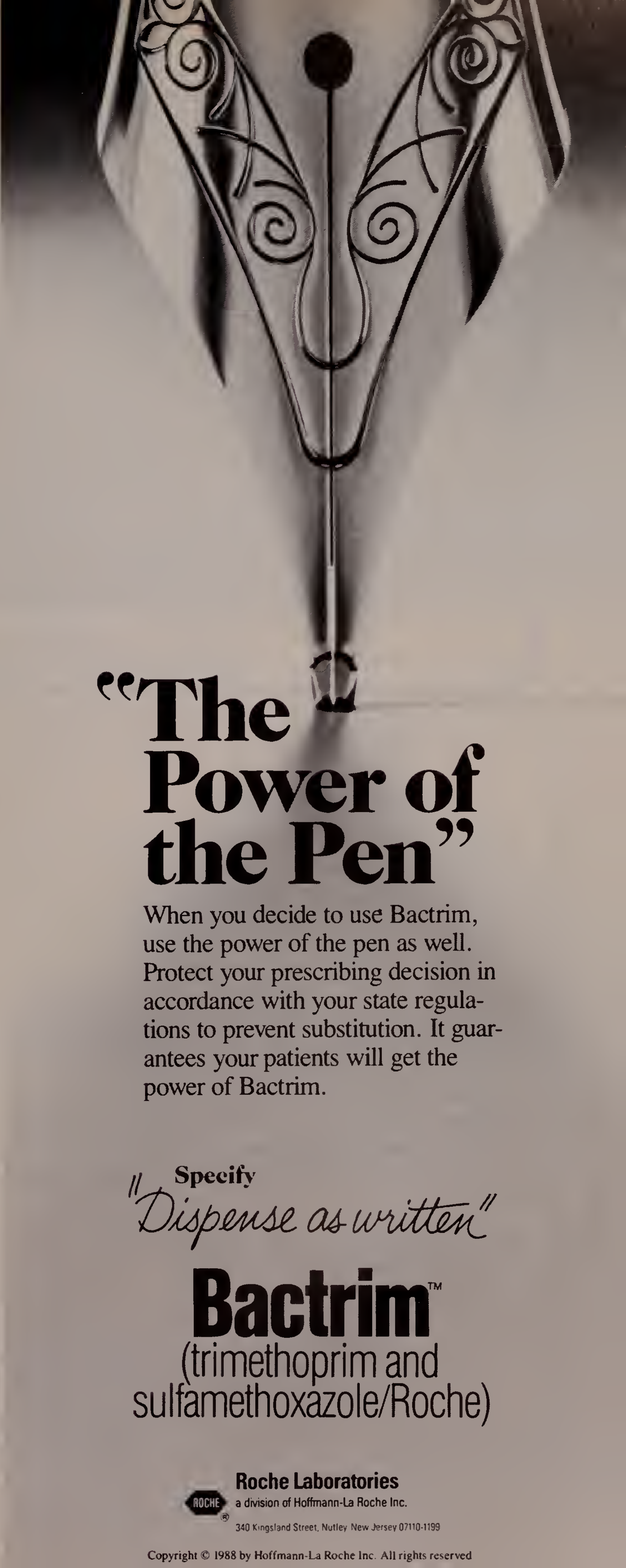
Dec 1–3. **American Back Society Fall Symposium on Back Pain.** In association with St Rose Hospital and San Francisco Spine Institute. 18½ Cat I Credits. Hyatt Regency Hotel, San Francisco. Contact: American Back Society, 2647 E 14th St, Suite 401, Oakland, CA 94601. Tel: (415) 536-9929.

Dec 8–9. **Advances and Controversies in the Management of Ocular and Periocular Malignancies.** 13 Cat I Credits. Hyatt on Union Square Hotel, San Francisco. Contact: Extended Programs in Medical Education, University of California, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

COLORADO

Dec 17–24. **Current Issues in Medicine.**

(*continued on p 26A*)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BRIEF SUMMARY

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Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that simultaneous administration of CARAFATE (sucralfate) with tetracycline, phenytoin, digoxin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The clinical significance of these animal studies is yet to be defined. However, because of the potential of CARAFATE to alter the absorption of some drugs from the gastrointestinal tract, the separate administration of CARAFATE from that of other agents should be considered when alterations in bioavailability are felt to be critical for concomitantly administered drugs

Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). There was no evidence of drug-related tumorigenicity. A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies were not conducted

Pregnancy: Teratogenic effects. Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4.7%).

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Reference:
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COMMENTARIES

Omphalocele at 15 weeks

The quality of images obtained by real-time diagnostic ultrasound units has improved dramatically in the last several years. Ultrasound, with its lack of ionizing radiation, has proved ideal in the diagnosis of obstetric and gynecologic problems. Indeed, in obstetrics the popularity of this modality is so great that many patients have come to expect an ultrasonic examination as a part of routine prenatal care. This increase in patient expectations combined with the relatively low cost of the equipment and the lack of any conclusive evidence that damage can be done to a fetus by sound waves has led to a great proliferation of ultrasound units and of people performing ultrasound examinations.

The training of physicians using ultrasound varies widely. Some have received extensive supervised training in radiology or obstetric-gynecology residencies. Others have received only two to three days of application training supplied by the company from which they purchased the ultrasound unit. This may be combined with attendance at conferences or visiting fellowships. The method of delivery of ultrasound service also varies. In some instances the physician personally performs the real-time examination. In other cases a technologist (whose training background is as variable as that of the physician) performs the study while preserving it on videotape for review by the physician. In still other cases, the technologist performs the study and the only record is a series of still images preserved on radiographic film, which are reviewed by the physician prior to dictating his report.

Not surprisingly, the quality of ultrasound studies being performed also varies widely. Some ultrasound laboratories can be remarkably precise in determining fetal age and weight and in documenting appropriate or inappropriate growth patterns on serial examinations. They can reliably identify fetal anomalies, blighted ova, or ectopic pregnancies. Other laboratories are far less reliable. In a given geographic area, referring physicians often become quickly attuned to who does "good" ultrasound and who does "bad" work and direct patients accordingly. However, even with this bias it still seems that the "bad" sonographers continue to get enough referrals to maintain their reputations. In areas where only a single poor ultrasound laboratory exists the reputation of the entire modality suffers.

Romano et al[1] describe in this issue of the *Journal* a case in which an omphalocele was diagnosed at 15 weeks' gestation using diagnostic ultrasound. They include a single image, taken from a real-time unit, which is so magnificently diagnostic that one glance is sufficient to confirm the presence of an abdominal wall defect, most likely an omphalocele. What the report does not state and what the picture does not demonstrate, however, is the great dependence of this modality on the experience and expertise of the person holding the transducer, that is, the person performing the examination. This image and this article are noteworthy because they demonstrate the tremendous potential of high resolution ultrasound in capable hands.

Like any acquired skill, the degree of proficiency obtained by a sonographer depends on many things, including the quality of teaching, the motivation of the pupil, and, probably most importantly, the number of examinations that the sonographer has performed. It takes repeated practice to manipulate the many variables involved in sonography to produce a quality study. Machine variables include transducer frequency and shape, distance-gain curve optimization, postprocessing enhancement, screen brightness and contrast, and film exposure and processing factors. Patient variables, such as degree of bladder filling, amount of bowel gas present, uterine and fetal orientations, and degree of cooperation of the patients, both maternal and fetal, are also important. Operator variables include position and pressure which the sonographer applies to the transducer, combined with the degree of angulation and/or sweeping motion used to visualize the fetus. All of these variables have a profound impact on the quality and orientation of the images obtained and thus on the interpretability of the study.

The novice finds it deceptively easy to run a transducer over an abdomen and produce reasonably acceptable still images. And yet just outside the plane of that image can easily be a large abnormality, completely missed. For it is a three-dimensional volume that is being evaluated and only imperfectly recorded in two dimensions. Learning to cover the entire uterine volume in a systematic way may sound easy, but given the variables described it can be incredibly difficult. In reviewing a series of still images one needs constantly to be concerned about what is not on the images as well as what is. Unless a physician knows himself to be blessed with exceptional ultrasound technologists he should ponder the ethical and medicolegal aspects of the above if the patient should be dismissed without being examined by the physician personally. And, unless a physician knows himself to be proficient in the use and interpretation of ultrasound sonography he should ponder also, before he enters the patient's presence.

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*The opinions expressed herein are those of the author and are not to be construed as official or as necessarily representing the views of the Department of the Air Force.

Filly[2] envisions ultrasonography in general as the stethoscope of the future, used by many and yet utilized to its full potential by only a few. In obstetrics the potential for this kind of "mal-use" is greater, I think, than in the other areas of sonography. This is because of the great demand present, as well as the rationalization that most of the fetuses are normal anyway so doing a less than optimal examination will be of little importance. Often the anomalies are very subtle, especially early, and can be difficult to detect even in the best hands. The omphalocele detected so elegantly by Romano et al[1] could easily have been missed in another setting, by another examiner, who was not as well trained. The patient, the fetus, and sonography deserve better than this.

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Orthostatic hypotension

In this issue of the *Journal*, Bhatt and Sharma[1] describe a case of recurrent syncope due to orthostatic hypotension. Orthostatic hypotension may escape early detection for several reasons. These include a lack of suspicion on the part of the evaluating physician; the evaluation of blood pressure in the afternoon or evening, instead of the early morning or after meals, when orthostatic hypotension is more prominent; symptoms of orthostatic hypotension other than syncope, such as leg weakness as the presenting manifestation; and, most importantly, the recording of orthostatic changes for only a few minutes.

Data collected by myself on 40 patients with orthostatic hypotension of diverse causes and 15 healthy volunteers were reviewed to standardize the duration of tilt required to elicit the onset of symptoms and/or a fall in systolic blood pressure exceeding 30 mm mercury. Blood pressure and heart rate were recorded after ten minutes of supine rest, and at one, three, five, seven and ten minutes of 90-degree tilt with foot support, if tolerated. Orthostatic symptoms occurred in 27.5% of subjects by one minute, in 70% by five minutes, and in 90% by ten minutes. A fall in systolic blood pressure exceeding 30 mm mercury was recorded in 60% by one minute, in 75% by five minutes, and in 85% by ten minutes. Combining both parameters produced a sensitivity of 67.5% by one minute, 87.5% by five minutes, and 97.5% by ten minutes.

This study demonstrated that orthostatic challenge should be given for at least ten minutes, and both orthostatic symptoms and blood pressure should be recorded. If the symptoms are not accompanied by a drop in blood pressure, one should look for another cause. The patients with dysautonomia usually had subnormal heart rate response. In my study patients, lightheadedness and leg weakness were the most frequent symptoms. Other symptoms, in decreasing frequency, were as follows: tinnitus and "dim" hearing, shaking of arms, palpitations, pressure in the chest, "heavy head," cloudy vision, drowsiness, tingling of head, general malaise, visual blackout, and bowel incontinence. Although a tilt table may be used for the assessment of orthostatic responses, it is usually sufficient to perform this test with the patient standing quietly, since both methods are comparable, though not physiologically identical, forms of orthostasis.

The maintenance of adequate blood pressure during upright posture requires an effective circulating blood volume and a baroreceptor-mediated feedback loop to stimulate the sympathetic nervous system. Abnormalities of either can present with orthostatic hypotension. Common causes of orthostatic hypotension include volume depletion (blood loss, dehydration), drugs (antihypertensives, neuroleptics, antidepressants, and monoamine oxidase inhibitors), and diseases of endocrine organs. Neurologic disorders that interrupt sympathetic outflow centrally or peripherally can also present with orthostatic hypotension. These include idiopathic parkinsonism, brainstem dysfunction, spinal cord lesions, Guillain-Barré syndrome, and neuropathies.[2-4] The patient described by Bhatt and Sharma[1] in this issue of the *Journal* had the striatonigral type of multiple system atrophy. Shy-Drager syndrome, another type of multiple system atrophy, involves progressive autonomic failure manifested by orthostatic hypotension, sexual impotence, anhidrosis, and urinary incontinence, and pyramidal, extrapyramidal, and cerebellar dysfunctions.[5] Although sympathetic insufficiency has been emphasized in the literature, parasympathetic dysfunction to a variable degree has also been observed in patients with central or peripheral autonomic failure.[6]

Recently, congenital biochemical defects have been added to the spectrum of causes of orthostatic hypotension. A patient with congenital ptosis, muscle hypotonia, nasal voice, and weak facial muscles had orthostatic hypotension with unmeasurable plasma levels of norepinephrine and dopamine beta hydroxylase, but a 7–12-fold rise in dopamine levels.[7] Tyrosine hydroxylase was present on immunohistochemical study. Orthostatic hypotension was attributed to failure of noradrenergic transmission because of a congenital defect in the release of norepinephrine.[7] A familial disorder with deficient kinin inactivating enzyme (bradykininase-1) but apparently normal sympathetic function was described by Streeten and colleagues.[8] When recumbent, these patients manifested flushing of the face, neck, and chest. On standing, purple discoloration of legs, decreased pulse pressure, and severe tachycardia were noted. These patients had normal plasma kallikreinogen concentrations, diminished bradykininase-1 concentrations, and increased bradykinin levels. Hypotension was attributed to vasodilation.

The evaluation of patients with orthostatic hypotension should include a careful history of drug intake and autonomic symptoms, a physical examination that includes orthostatic blood pressure and heart rate responses for a period of at least ten minutes, neurologic examination, and autonomic tests. Several physiologic maneuvers and pharmacologic tests can help to localize autonomic dysfunction: tilt-table study, the Valsalva maneuver, cold pressor test, mental arithmetic test, sweat test, hyperventilation, intravenous atropine administration, and supine norepinephrine levels.[9,10] Abnormally low basal plasma norepinephrine levels, along with absence of neurologic findings, may indicate a postganglionic or peripheral type of autonomic failure. A preserved heart rate response to the Valsalva maneuver (Valsalva ratio) or to atropine administration will indicate intact vagal function, which should be considered when managing patients with orthostatic hypotension, because a therapeutic attempt to raise blood pressure may be counteracted by excess bradycardia.[11]

Orthostatic hypotension in the absence of symptoms usually is not an indication for therapy. Treatment is necessary, however, when symptoms of orthostatic hypotension seriously interfere with the daily lives of patients or culminate in frank syncope. The aim of therapy is to reduce symptoms enough to permit day-to-day living. Treatment should be targeted, therefore, to times of the day when orthostatic hypotension is worst, for example, in the morning or after meals. In some patients, avoiding offending drugs or correcting the primary disease will suffice. In other patients, therapeutic measures can be divided into nonpharmacologic and pharmacologic groups. There is a variety of nonpharmacologic measures: gradual change in posture to allow more time for adjustment of vascular righting reflexes; sleeping with head of bed raised to reduce nocturnal diuresis and to allow adjustment of cerebral autoregulatory mechanism; use of Jobst stockings from metatarsals to the umbilicus as venous pressure gradient support; and attention to the timing as well as carbohydrate and sodium content of meals. Patients with orthostatic hypotension who have a fixed heart rate or intact vagal function may benefit from atrial tachypacing.[2]

The pharmacologic treatment of orthostatic hypotension has only been partially effective. This may be due to the lack of understanding of the pathophysiology of the syndrome and the precise mechanisms of drug action. Fludrocortisone is the single best drug for initial treatment. Its initial effect of increasing plasma volume gives way to increased sensitivity of the vascular smooth muscle to endogenous norepinephrine. When postural symptoms are not controlled with fludrocortisone, drugs to facilitate vasoconstriction or retard vasodilation are employed. Tyramine and hydroxyamphetamine, which facilitate release of norepinephrine from sympathetic nerve endings, are not easily available. DL-threo-3,4 dihydroxyphenylserine reduces orthostatic hypotension by increasing norepinephrine synthesis. Monoamine oxidase inhibitors, tranylcypromine, or phenelzine, which prevent the breakdown of catacholamines, may also be helpful. Dihydroergotamine taken intravenously or ergotamine tartrate by mouth, both direct-acting alpha-adrenergic agonists, have recently been used with some success, but ergotamine can provoke coronary spasm and impair blood flow to the extremities. Midodrine, another long-acting alpha-adrenergic agonist, is only inconsistently useful. Indomethacin and metoclopramide improve vascular responsiveness to endogenous vasoconstrictors. Pindolol, a beta-receptor antagonist with intrinsic sympathomimetic activity, and clonidine, an alpha-2 receptor stimulator, have been found useful in postganglionic sympathetic failure. The combination of fludrocortisone with dihydroergotamine or with monoamine oxidase inhibitors has been employed in refractory cases. Drugs intended to limit vasodilation include indomethacin which inhibits prostaglandin synthesis, metoclopramide which blocks dopamine receptors, propranolol which blocks beta receptors, cyproheptadine which blocks bradykinin in patients with hyperbradykininism, and somatostatin which blocks a vasodilating peptide.[5,12]

Patients with orthostatic hypotension have marked spontaneous fluctuations in supine blood pressure due to spontaneous changes in peripheral vascular resistance and altered circadian rhythm.[13] Many drugs that improve blood pressure simply shift the pressure curve higher, thus increasing the supine blood pressure into the hypertensive range in order to get acceptable orthostatic blood pressure. There is no ideal drug that can emulate precise central integration of sympathetic, parasympathetic, and other afferent stimuli, allowing selective vasoconstriction when a patient is upright. Attempts have been made to address these limitations. Polinsky et al[14] devised an electromechanical neural prosthesis which maintains blood pressure at predetermined levels by the chronic intermittent infusion of norepinephrine on postural demand. However, this device needs further work and refinement. Other therapies include short-acting vasopressor agents to provide increased blood pressure during times when it is most needed. For example, caffeine, ergotamine tartrate, or indomethacin by mouth in the morning and at lunch have been helpful.[15] These drugs are not given after 6 PM to avoid supine hypertension at night. Demand atrial tachypacing has also been considered a possible solution to this problem. In patients with postprandial accentuation of orthostatic hypotension, meals given at bedtime may counteract supine hypertension.

In brief, the management of orthostatic hypotension remains a challenge. The elucidation of basic pathophysiologic mechanisms should provide better ways of treating affected patients in the future.

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Gammaglobulin treatment in chronic lymphocytic leukemia

Renewed interest and new applications

The initial clinical experience with gammaglobulin therapy in chronic lymphocytic leukemia (CLL) in the early 1960s was not a successful one due to the fact that one could not administer and achieve meaningful elevations in systemic immunoglobulin levels using intramuscular preparations. With the newer intravenous preparations, normal to elevated serum levels of immunoglobulin G (IgG) are easily achieved with little toxicity and no risk of infection from human immunodeficiency virus (HIV) and non-A, non-B hepatitis.[1,2] The Food and Drug Administration (FDA) has approved these new and relatively expensive preparations for the hypogammaglobulinemic states (acquired and congenital forms) and for immune thrombocytopenic purpura (ITP). The reason why intravenous IgG was tried in these two seemingly divergent diseases was based on the observation of a high incidence of autoimmune diseases in hypogammaglobulinemic states and the fact that replacement therapy ameliorated autoimmune problems.[3]

Approximately 50% of patients with CLL can be considered to have hypogammaglobulinemia depending on the lower limits of normal. In this disorder low levels of immunoglobulins correlate with a high incidence of infections and death, particularly due to encapsulated microorganisms.[4,5] Clearly, a hypogammaglobulinemic CLL patient with serious infections or chronic persistent infections deserves to be treated with intravenous immunoglobulin, as shown in the article by Bottino et al[6] in this issue of the *Journal*. Patients with CLL who have one or more episodes of serious infection can justifiably be given maintenance intravenous immunoglobulin, as in the other hypogammaglobulinemic disorders, to avoid serious morbidity and mortality as well as the equally expensive hospitalizations and antibiotic therapy.

The role of prophylactic intravenous immunoglobulin in patients with CLL and with hypogammaglobulinemia regardless of history of infections, is currently under investigation. In a recent stratified, prospective, randomized double-blind study conducted in Europe and the United States, more than 100 patients with CLL and with hypogammaglobulinemia received either intravenous immunoglobulin or a placebo every three weeks for 12 months.[7] A total of 127 bacterial or viral infections occurred in 57 patients who could be evaluated. A significant decrease in bacterial infections (23 episodes) was observed in the intravenous immunoglobulin-treated group (ten of 28 patients), compared to 42 episodes in the placebo-treated group (16 of 29 patients)($p=0.001$). However, the incidence of viral infections was similar in both groups.[8]

The other issue concerning infections and CLL is the identification of those patients with hypogammaglobulinemia who can benefit from replacement therapy. These data may be forthcoming in the randomized study looking at subgroups of patients with IgG levels ≥400 mg/dL versus those with levels <400 mg/dL. Other investigators are measuring specific antibody titers to encapsulated organisms in order to identify patients with low titers who may be at high risk of infection. Because of the cost of therapy, the high risk subgroup of CLL patients should be identified. Until data become available, one should consider prophylactic intravenous immunoglobulin therapy in CLL as experimental.

Chronic lymphocytic leukemia and the congenital or acquired forms of agammaglobulinemia share a common problem, which is the paradoxical development of autoimmune disease in the setting of low IgG levels and B cell failure. The autoimmune problems in CLL such as ITP and autoimmune hemolytic anemia respond to intravenous immunoglobulin therapy,[10,11] as do those in patients with common variable immunodeficiency.[12] Furthermore, the leukemic process itself may be altered by intravenous immunoglobulin as shown in preliminary trials in CLL as manifested by a decrease in lymphocyte counts, lymphadenopathy, and splenomegaly in some patients.[10] These observations have indicated a larger role for intravenous immunoglobulin in CLL other than the treatment of infections.

The treatment of CLL patients with standard immunosuppressive and chemotherapeutic agents has not been shown to have any beneficial effects on their overall survival. In a disorder in which the major causes of morbidity and mortality are complications of immunosuppression, standard therapy that further suppresses the immune system is apparently not the best approach. There are concerns that standard therapy in CLL accelerates the development of hypogammaglobulinemia[13] as well as development of autoimmune complications.[14] Whether intravenous immunoglobulin will play a major role in the treatment of CLL will depend on future clinical trials.

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RESEARCH PAPER

Needle localization of occult breast cancer

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ABSTRACT. The clinical diagnosis of suspicious occult breast lesions has become more common, with both noninvasive and invasive methods being used to obtain accurate localization. This paper represents our preliminary experience with 152 patients using the Homer Mammalok needle-directed biopsy. Carcinoma was found in 28 (19.8%) of the patients; 19 (67.8%) of the lesions were invasive and 9 (32.2%) were noninvasive. Fourteen percent of the patients had axillary lymph node metastases. Needle localization is a safe, accurate method of diagnosing occult lesions with a minimum sacrifice of breast tissue.

(NY State J Med 1988; 88:574–576)

Occult breast cancer was not seen with any frequency until the introduction of clinically acceptable mammography by Egan[1] in 1960. It was usually found when a suspicious lump was biopsied that revealed predominantly fibrocystic changes and, in the slides, a focus of invasive or in situ carcinoma was identified. Carcinoma in situ (ductal or lobular) was rarely identified in 1978, representing less than 5% of all breast cancers; however, with increasing use of mammography, its incidence may well exceed 20%. Localizing and removing these clinically occult lesions represents a challenge for both the radiologist and the surgeon and requires their close cooperation. This study represents our experience with needle-guided biopsy of suspicious occult breast lesions.

PATIENTS AND METHODS

One hundred fifty-two consecutive patients with negative physical findings, who on mammography demonstrated clustered microcalcification, suspicious density, or both, had a biopsy using preoperative needle localization. Mammography was initially performed because of age, strong family history, previous cancer in the contralateral breast, or the presence of a lumpy breast that was difficult to examine.

Patients were admitted to the ambulatory surgical unit on the morning of the procedure and were discharged after recovery from the anesthesia used for the biopsy. Premedication for the biopsy was not given until after the needle localization procedure, which was performed with the patient seated in a padded, height-adjustable chair. Prior to needle placement, location of the abnormality on the previous mammogram was determined relative to the nipple. Medial or lateral position and depth from skin surface were measured on the craniocaudal view. When the patient arrived for the procedure, a true lateral view was taken to determine position superior or inferior to the nipple. This was necessary since a standard diagnostic mammogram usually includes an oblique mediolateral projection to show the axillary tail of the breast which distorts measurements in this plane.

The coordinates were marked on the skin surface, which was then prepped with povidone-iodine. Lidocaine hydrochloride, 1% injection USP, was used to infiltrate the skin. With the technologist compressing the breast in the mediolateral plane, a Homer Mammalok needle (Namic) was introduced from the shortest distance from the skin surface, according to the measurements previously obtained. Lower inner quadrant lesions in large breasts often required an anterior approach, due to the dependent position of the breast with the patient erect. Mammalok needles are available in several lengths (3.8 cm, 6.4 cm, 8.9 cm), which allows selection of a needle that would not project awkwardly from the skin surface. The inner wire core was then advanced beyond the needle tip, forming a smooth curve which anchored the needle within the breast. Craniocaudal and true mediolateral views were then obtained to show position of the needle relative to the lesion. An ideal placement was within 5 mm of the abnormality, but depending on the particular anatomy, up to 1 cm proximity proved adequate (greater accuracy is recommended). If repositioning of the needle was indicated, the wire guide was pulled back into the needle, the tip realigned, and the wire guide advanced again. The memory of the wire tip insured reaquisition of a smooth curve within the breast.

Once needle position was considered appropriate, 0.3 cc of 1% methylene blue injection USP, previously mixed with 1% lidocaine (one part lidocaine to five parts methylene blue), was injected to provide a blue mark in the tissue should the wire be displaced during surgery. Dressings were applied, and the patient was sent to the operating room for the biopsy.

There were no complications in the insertion of the needle per se, but while having the mammogram after the needle placement, an occasional patient experienced a vagal episode with nausea, hypotension, and bradycardia. When the patient returned to the supine position on the stretcher, the symptoms rapidly resolved without medication. Since these episodes were not predictable, it was essential to have the seated patient under constant observation, including during film processing.

To minimize the amount of breast tissue removed when biopsying these occult lesions, many methods for localization have been devised. Noninvasive techniques,[2-5] based on plotting coordinates from the nipple, are blind procedures that may result in unnecessary breast tissue removal, though in experienced hands these techniques appear accurate. Invasive techniques range from preoperative injection of radio-opaque dyes[6] or plain blue dye,[7] to preoperative placement of one or more localizing needles[8-14] or intraoperative placement of localizing needles.[15]

In the early part of this study, most patients received general anesthesia, but with increasing experience, more than 95% of biopsies are now performed using 1% xylocaine plus midazolam. The addition of midazolam not only provides excellent sedation for the anxious patient, but amnesia for the event has also been

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noted by many patients. When using midazolam, it is essential that patients receive continuous percutaneous oxygen monitoring. The excised tissue is sent for immediate specimen radiography and, while awaiting confirmation that the suspicious area has been removed, the medical personnel maintain meticulous hemostasis. Wounds are closed with running subcuticular sutures of 5-0 nylon and no drains. The pathologist sections and radiographically reexamines the specimen, maintaining orientation as to sequence. Areas of concern such as microcalcification are targeted and blocked, and sections are prepared. Frozen sections are not used.

RESULTS

One hundred and fifty-two patients were judged to have suspicious occult lesions on clinical and mammographic evaluation. Biopsies of these lesions were performed using the preoperative hooked-needle localization technique. There were 123 (80%) benign and 28 (19.8%) malignant lesions. In the benign group, fibrocystic changes predominated, representing 105 (85%), with the remainder being fibroadenoma, fat necrosis, lymph nodes, hemangioma, and one case of Kaposi sarcoma (most likely arising from an intramammary lymph node). Localization in this latter case was by intraoperative needle placement and was the single example of this method. Of the 28 carcinomas, there were 14 invasive ductal, two invasive lobular, two tubular, and one carcinoid lesion. The average age of these patients was 58 years. The noninvasive carcinomas consisted of eight intraductal and one lobular carcinoma in situ. Three of the 14 invasive ductal cancers (diameters of 8 mm, 15 mm, and 20 mm) had positive axillary lymph nodes, as did one of the lobular carcinomas in situ (diameter 10 mm). Three patients with intraductal carcinoma and one with lobular carcinoma in situ elected to be followed; the others were treated by modified radical mastectomy.

DISCUSSION

Occult breast cancer and minimal breast cancer are terms often used interchangeably. Not all occult cancers, however, are minimal, even though most minimal cancers are truly occult. An occult breast cancer may be of any size but cannot be detected by clinical means. Minimal breast cancer, a term introduced by Gallagher and Martin[16] in 1971, referred specifically to duct carcinoma in situ, lobular carcinoma in situ, and invasive carcinoma measuring less than 0.5 cm. Unfortunately, tumors up to 1 cm are being labeled minimal, but this group should not be considered minimal, early, or favorable, as there may well be a tendency for the unwary to be less than suitably aggressive in treatment.

Although breast imaging procedures such as magnetic resonance imaging, computerized axial tomography, sonography, telethermography, cholesterol plate thermography, radioisotopes, digital subtraction angiography, and diaphanography have been and are being evaluated, plain film/screen mammography and xeromammography remain the gold standard for detecting occult breast lesions. It is important to remember that until better adjuvant therapy becomes available, the best hope for cure remains early detection by mammography and appropriate local treatment.

The needle technique for localization of suspicious breast lesions has contributed immensely to the increase in positive yields in clinically occult cases. Libshitz et al[13] reported that 22% of biopsies by needle localization were malignant, and a study by Poole et al[17] detected cancer in 14.3% using the same approach. The series described here, with a malignancy rate of 19.8%, compares favorably with other series which report rates that vary from 10% to 35%.[11,18-23]

The Breast Cancer Detection Demonstration Project (BCDDP)[24] reported 13.4% axillary metastases in tumors less than 1 cm. The 1978 SEER Report[25] from the NCI found 20.2% axillary metastases in tumors under 1 cm, and Schwartz et al[26] showed 23.1% axillary metastases in occult tumors found only by mammography. Bedwani et al,[27] reporting on the American College of Surgeons Breast Cancer Survey, noted a 23% incidence of axillary metastases with invasive tumors under 0.5 cm. Finally, in those cases diagnosed as ductal carcinoma in situ, there is a reported incidence of 1% to 2% axillary metastases.[28-29] In the 3,557 cancers detected by the BCDDP study,[24] 41.6% were found by mammography alone. Noninvasive cancers accounted for 59%, 52.6% of which measured less than 1 cm. Of the greatest significance, 79.5% of the invasive cancers less than 1 cm were negative for axillary node involvement.

The increasing expertise in early diagnosis would have little real meaning unless it were reflected by increased survival. Despite some pessimism and expressions in the literature implying that variations in local-regional treatment have little impact on survival, several studies would suggest that screening and small tumor volume is associated with increased survival.[29-32] The only prospective randomized trial of breast cancer screening in the United States using mammography and physical examination was conducted by the Health Insurance Plan of Greater New York[33] and continues to show a mortality reduction by one third beyond 14 years. The Swedish National Board of Health Trial[34] using mammography alone also indicated a mortality reduction by one third beyond seven years. These two studies prove that screening has in some way intervened in the natural history of the disease and has added years of life to the screened group. Moreover, the aforesaid screening should put to rest the concept of lead-time bias and should encourage persistence in early diagnosis. Other studies[29-33] have shown that the patient's prognosis with these smaller tumors is better than the prognoses of patients who present with increasing tumor volume.

Evolving imaging techniques are detecting increasing numbers of occult lesions which require biopsy by a safe, simple, accurate method, such as preoperative hooked-needle localization, which removes a minimum of breast tissue.

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FROM THE LIBRARY

WHAT EVERY MEMBER OF THE MEDICAL PROFESSION SHOULD KNOW ABOUT PROTECTING WOMEN FROM DEATH FROM CANCER OF THE BREAST

It does not make any difference what your specialty is in medicine, or whether you are a general surgeon or general practitioner, or even whether you have retired from the medical profession, you still have an opportunity to help reduce the mortality of cancer no matter where it is first situated.

Those most familiar with cancer today agree that there is no treatment for malignant disease except some form of surgery the object of which is complete removal, or irradiation with x-rays or radium. There is also universal agreement that these therapeutic agencies accomplish the largest number of temporary and permanent cures the sooner they are applied after the first symptom.

Prolonged experience justifies me in making this statement to every lay and medical audience which is not yet fully appreciated by either. The prevention and cure of cancer today depend upon the view that in the beginning cancer is a local disease, and at first the cancer cells in that spot are not malignant, but simply abnormal cells changed by injury or chronic irritation of some character from the pre-existing normal cells in the spot. It is true that in some forms of malignant disease there are in the beginning multiple areas. These types are hopeless unless temporarily arrested and, in a very few instances, cured by irradiation with x-rays or radium.

The next fundamental conception justified by ample evidence is that the signs or symptoms of an abnormal area not cancer and which will never be cancer, or an abnormal area which precedes cancer, or the earliest stage of cancer itself, are identical. The danger of cancer is not so much its local growth, as the tendency of the cancer cell to enter the blood or blood stream and to be carried to spots beyond the possibility of surgical removal, and internal sites so far removed and so surrounded by other tissues that irradiation seems unable to destroy the function of the living cancer cell.

Therefore the protection against death from cancer, at present, is first a purely educational one. Every one must be informed and persuaded to report to their family physician or some medical adviser selected while they are well, the moment any warning sign or symptom appears. The greatest protection comes with routine annual examinations by a physician or clinic selected with the greatest care and visited the first time while well. . . .

JOSEPH COLT BLOODGOOD, MD
(*NY State J Med* 1932; 32:259-264)

Psychiatric teams and the psychiatrist's authority in the New York State mental health system

IRVING M. JABITSKY, MD

"**A sacred cow** of contemporary American life is the team concept."[1] Schools encourage good citizenship based on the Jeffersonian democratic principle that all men are created equal, by emphasizing the team spirit. The individual's contribution is not deemed as important as the team effort. In this context, the team concept usually relates to sports.

Another source of the team concept comes from World War II. Hutt, Menninger, and O'Keefe[2] attributed the emergence of the neuropsychiatric team in World War II to the necessity for maximum service with a minimum of staff. Under the pressure of war, it was necessary for psychiatrists, social workers, and psychologists to pool their talents and increase their interactions. Taxay[1] postulated that the use of the term *team* may have begun during World War II when a team was assembled to build an atomic device.

Does the team concept apply to psychiatry? Are all the team members equal? Who is the team manager or team leader? What are some of the problems facing psychiatrists who choose to play ball with the team?

EQUALITY VS UNIQUENESS

All members of the team are unique but not equal. Psychiatrists, social workers, psychologists, nurses, and occupational therapists each bring a unique orientation to the process of problem identification and assessment.[3] New[4] stated, "In our attempts to be too equal, we may have done a disservice to ourselves by failing to recognize that certain situations could only end in a deadlock if provisions are not made to allow a full expression of inequality. To put this more positively, we should realize that competence, by itself, implies a certain amount of inequality; that, indeed, some persons are more capable than others."

Trute and Macpherson[3] found the differing organization of sports teams to provide a humorous yet useful analogy to the psychiatric team. They reject the volleyball model because all team members are seen as having roughly equivalent roles and having equivalent decision-making authority. They praise the hockey team model because it permits the maintenance of the integrity of the roles of team members. Each player has a unique rationale in being a member of the team and each is appreciated for the difference in function that he performs as a team member. A team approach is a collaborative effort which utilizes the unique training, experience, and knowledge of the individual disciplines. "A team is the process whereby various professionals who make individual decisions concerning patients and who share a common purpose, meet together to communicate and share knowledge from which plans are made and thus future therapeutic decisions are influenced."[5] The emphasis is on a common purpose. For example, if one member of the team has research interests and another educational interests in training new professionals, there is a possibility that case assignments may hinge on whether the case offers research possibilities or poses an interesting educational problem. Another example is if the hospital and team leader want to keep the ward census low, team treatment planning may focus on discharge planning rather than on long term treatment goals.

Sifneos[6] set up a specialized Community Mental Health Training Program for psychiatrists, psychologists, and social workers at the Massachusetts General Hospital. Initially, the interdisciplinary seminars were poorly attended. While the faculty made an effort to integrate all the trainees as quickly as possible and to teach them the public health and social science approach, no effort was made to preserve the trainees' own professional identities. It was only after the faculty started to utilize more effectively the previous background of the trainees, and to emphasize the specific contribution each discipline could offer to the group as a whole, that they were able to develop a meaningful educational experience for all.

Even a team of strong individual parts, each with major contributions to make, may be a weak team as a whole. This paradox may exist not only when teamwork fails, but when it succeeds, and not only despite teamwork but because of it. If egalitarian teamwork brings about sufficient blurring of roles, and if jobs are sufficiently undifferentiated, the full thrust of each member's clinical effectiveness is never felt. Diffused identities and diffused social structure breed inefficiency and create adventitious anxiety that interferes with and undermines therapeutic work.[7]

Gomez et al[8] wrote that little attention has been paid to the managerial aspects of the team and to clinical under-

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standing of the interpersonal dynamics that are always present among team members. While those two factors are of primary importance in the delivery of mental health services in general, they are uniquely relevant in state hospital settings. In such settings, treatment priorities have always been so low, budget allocations so scarce, and the quality of staff so poor that extra attention must be paid to the composition and effectiveness of the multidisciplinary team and to understanding team dynamics. If those dynamics are overlooked, the impact of the team approach can be psychopathologic rather than psychotherapeutic and can result in major deficiencies in the quality of care delivered to the most needy patient group.

AUTHORITY, RESPONSIBILITY, EXPERTISE

The area of greatest confusion and intrateam conflict is the failure of team members to link the psychiatrist's authority with his legal responsibility by questioning his expertise. Obviously, this expertise is not absolute, which is the raison d'être of the psychiatric team. In the absence of the psychiatric team, lack of expertise is never grounds for challenging the authority of the psychiatrist. For example, a first-year resident in psychiatry who is unfamiliar with neuroleptics and dosage may be confronted by a psychiatric nurse of 25 years' experience. She will offer her advice, or the resident will seek it out, but both professionals understand that the resident will write the order. He has that authority because he has the legal responsibility. Almost everyone recognizes the legal responsibility of the psychiatrist in making decisions regarding admissions, discharges, legal status, passes, and medication. Yet the psychiatric team often challenges the authority of the psychiatrist to make those decisions.

When I began working in the state system, social workers were writing passes in my hospital. At a meeting, I questioned how this could be allowed if I was the one ultimately responsible. The argument was not persuasive, and one team member asked, "Why, do you know more about the patients than the social worker does to write a pass?" There was no linkage of authority with responsibility for a patient who leaves the hospital grounds.

Unfortunately, the psychiatrist's decision-making role is perceived by team members as a rejection of their expertise. Arguments like, "He doesn't understand the team concept," or "He doesn't listen to us" are used in lieu of, "He rejects our expertise."

The challenge to the authority and leadership of the psychiatrist is relatively new. In 1961, Crawshaw and Key[9] wrote, "In discussing the physician's role on the team, one is dealing with a special problem of leadership. The doctor and leader are almost always considered identical by the psychiatric staff. A few exceptions arise with a nurse or a psychologist leading a team, but these are quite rare and stem from either the absence of a physician or of one individual having outstanding qualities of leadership in a special setting which allows development. In the usual situation, the psychiatrist acts both as the administrative and therapeutic leader. The basis for leadership is broad and has been investigated by many writers, the most immediate emphasis being placed on the legal responsibility for a patient's care."

The linkage of responsibility and leadership is assumed by Brown and Ishiyama:[10] "In virtually all instances in which he is available, the staff's leader will be the psychiatrist. By virtue of his ultimate medical and legal responsibility in the medical setting, as well as his status in the hospital community, this becomes a largely inevitable choice." The proliferation of the professionals was a challenge to the psychiatrist's professional identity.

Ishiyama[11] wrote, "At one time, the patient knew who the doctor was. Then came the psychologist, who was a doctor, but not a doctor. Social Workers are now acquiring Ph.D.'s and D.S.W.'s in increasing numbers. Nurses are exchanging not only their uniforms for street clothes, but also their R.N.'s for Ed.D.'s and Ph.D.'s. Even the psychiatrist, who is supposed to be the real doctor, has long forsaken the art of doctoring. Consider the psychiatrist, who, upon ministering to a patient's physical ailment, is heard to say, 'It's sure good to get back to doctoring sometimes!' "

The team's inclusion of nurses, social workers, psychologists, and, in some cases, chaplains,[12] volunteers, and ward aides[13] created a new problem. Bower[14] stated, "A genuine problem for every hospital psychiatrist was the lack of clarity about which procedures, decisions, and treatments had to remain uniquely his and which he could and should delegate." Even the composition of the team members lacked clarity. I recall a management decision to include the ward secretary on a case conference before the hospital's director. A dozen or more people were present in the room when the director by chance called on the secretary for an opinion, not knowing her title. With much embarrassment, she said, "I am the secretary," thereby disqualifying herself from answering the question. The director correctly replied that if she participated as a team member, he had every right to call on her.

TEAM LEADER

By the mid-70s, the lack of clarity as to the authority, leadership, and role of the psychiatrist resulted in psychiatrists leaving positions of leadership, and the psychologists, social workers, and nurses filling the void. New York State created a new Civil Service title, "team leader," which was filled almost entirely by nonphysicians. Not only is the leadership of the team removed from the psychiatrist, but his authority is questioned as well. "Lip service is paid to the ultimate clinical authority of the physician, but in practice he is pressured to follow the demands of the team and its leader. The authority of the physician is diminished in the eyes of team members, who see the physician as just an equal member."[15] Talbott[16] discussed the increased readmissions in the public sector and a greater need for physicians. However, instead of hiring more psychiatrists, the number of nonmedical administrators was increased, which has left physicians feeling devalued. "Abuses of the team system have resulted in a 'democratized' ward team voting on discharges or medication, both of which are medical responsibilities."

Doyle[17] was the psychiatric supervisor of a new mental health center established in a setting of equality of all professionals, including the physician. She concluded, "Total equality without the underpinnings of a delineation of authority can have a deleterious effect on the very excellence of patient care that is sought."

In June 1968, Syracuse Psychiatric Hospital appointed nonphysicians to the position of psychiatric team leader on three treatment teams. Greiff and McDonald[18] studied the difficulties that arose between the nonphysician team leaders and psychiatrists. Some doctors felt that the psychiatrists' authority was not commensurate with their legal responsibilities. A structured interview dealt with two issues, discharge and medications; afterwards, a questionnaire was filled out. The authors of the study concluded that conflicts arose because of overlapping of roles and suggested that greater clarity in role delineation would be helpful in recruiting new staff members.

Democratization of the team has introduced into the literature the idea of voting. Fischer and Weinstein,[19] in discussing the team approach, referred to "one-man-one-vote" in deciding whether to use medication and certain treatments like electroconvulsive therapy.

Favis[20] stated that pathologic coalition and conniving cannot be halted if the psychiatrist is given one vote in the treatment team. The psychiatrist, by having only one vote, leaves the impression that his expertise and judgment are no better than those of the other members. This inevitably leads to an overt or covert challenge to his authority.

SUBMIT, FIGHT, OR FLEE

The psychiatrist's response to the challenge to his authority can be to either submit, fight, or flee. Few psychiatrists are interested in fighting. Indeed, why fight if it is so much easier to submit or resign? "More and more he relies on the treatment team and no longer uses his discretion, ignoring even his legal responsibilities."[20] Of course, submission requires compromising one's professional integrity. Resignation appears much more appealing for both clinical and administrative psychiatrists. The void left by the resignation of American graduates has been filled by foreign medical graduates (FMGs).

In 1975, slightly more than half of the psychiatrists and other physicians in state mental hospitals were FMGs; in some states this proportion neared 90%.[21] FMGs are culturally and linguistically alien to the patients they serve.[22] Psychiatrists need to know the language and culture well in order to communicate effectively and to understand the sociocultural psychologic factors of illness.[23] The University of South Florida established a language-cultural course for foreign psychiatric residents to address this problem.[24] However, premature judgment on the knowledge and expertise of FMGs should not be based on cultural and linguistic deficits. Neither should the contribution FMGs make to the mental health system be undervalued.

Keill[25] studied the resignation rate of senior administrative psychiatrists in the New York State Department of Mental Hygiene between March 1975 and 1978. In 1975, 48 psychiatrists held senior positions in the department, including 31 psychiatric hospital directorships; 11 central office positions as assistant, associate, or deputy commissioner; and six regional directorships. Three years later, 27 (56%) of these physicians, including the commissioner, had voluntarily left the state positions for more gratifying activities.

Greenblatt[26] reported that between 1971 and 1981 the number of psychiatrists serving as state commissioners of mental health fell about 56%, while the number of nonpsychiatrist commissioners rose by 78%. In 1971, there were 41 psychiatrists, six nonpsychiatrists, and seven nonphysicians serving in the states and territories. In 1981, there were 18 psychiatrists, four nonpsychiatrists, and 32 nonphysicians.

Beigel, Sharfstein, and Wolfe[27] reported that between 1971 and 1977 the percentage of community mental health centers that had psychiatrists as directors decreased from 55% to 26%. Meanwhile, the percentage of center directors with backgrounds in social work increased from 17% to 33%, and in administration, from 3% to 12%.

MANAGEMENT VS LABOR

The establishment of the title "team leader" within the framework of governmental civil service created additional problems. The team leader is part of management, and the psychiatrist is part of labor and its union. The collaborative nature of the team is now divided along contractual issues. The team leader evaluates all members of the team, including the psychiatrist, and determines salary increases. The psychiatrist must consider what effect his decisions will have on the leader. Will they antagonize and alienate the leader, and if so will they impact negatively on the physician's evaluation and salary?

The team leader is the boss, and the physician has been forced into a subordinate role. For example, an agitated patient was screaming in the hallway. A PRN medication order had been written several days before. The staff and the psychiatrist were discussing whether to use it, or to attempt to talk with the patient and quiet her. The psychiatrist believed it appropriate to try talking first and so stated. The team leader, who was not initially present at the meeting, heard the patient screaming and entered the meeting room. He asked the nurse what she wanted to do, and she answered that she wished to give an injection. The team leader ordered that it be given, without consulting the psychiatrist, and the nurse immediately proceeded to give the medication. It made no difference that the psychiatrist had recommended not to give the medication. The team leader had spoken and he was the boss.

Although the team leader is supposed to function as administrative chief, and the psychiatrist as clinical chief, administrative and clinical decisions overlap. Roth[28] wrote, "Supervision of the unit physician by the unit chief or the team leader extends to administrative matters only; in professional matters he is subordinate only to the medical clinical director or his delegate."

Favis[20] agreed: "With regard to the team leader, the position of coordinator can be misused, and a supervisory and decision-making position be assumed. When a nonphysician team leader starts making decisions for the group, the danger of pathological decisions exists, especially if the process cannot be controlled by the supervisory capacity of the psychiatrist."

In the New York State Office of Mental Health, the psychiatrist has no supervisory role over the team leader. Indeed, disciplinary proceedings can be initiated by the team leader against the psychiatrist and not vice-versa because the team leader is management and the psychiatrist is labor.

Those psychiatrists who do not have tenure or are unlicensed will tend to withdraw and acquiesce to the team leader's decisions, fearing alienation. Unfortunately, senior administrative psychiatrists, who are also in management, tend to support nonmedical chiefs of service and team leaders and not on-ward clinical psychiatrists, who are labor.

Sletten[29] discussed the problems that occur when a nonmedical administrator is given control of the fiscal and personnel policies of a state mental hospital. He argued that supplies, equipment, physical environment, personnel policies, patient policies, and staff orientation directly affect patient care and treatment programs. Administrative decisions have direct bearing on clinical issues.

Racy[30] discussed the inherent ambivalence of feelings between the leader and the led. "Those who have power desire it but feel guilty because of it. Those who are subject to another's power both welcome and resent it. The ambivalence remains even in the situation, where the leader, by virtue of personal or professional qualifications, is the obvious choice to lead. For all leadership implies an inequality between leader and led."

Miller[31] explained why psychiatrists are often resented when they attempt to impose their leadership on the treatment team. For years many of the hospitals had been run chiefly by aides, nurses, and psychologists, who came to feel that they could provide most of what patients needed. Physicians were often considered useful primarily to sign medical orders for treatment that had been prescribed by nonphysicians. As a result, the hospitals had learned out of necessity to do without psychiatrists. This trend has had a resurgence of late, most notably in California and New York, where psychologists are seeking admitting privileges.

Schindler et al[32] studied the causes of conflict between psychiatrists and psychologists. They found that a major disagreement was over perceived competency to perform mental health tasks; each group thought itself more competent in a variety of regular activities. Psychiatrists perceived psychologists as more competent than themselves only in conducting psychological testing. Psychologists viewed psychiatrists as more competent only in the area of medication management.

The rivalry between members of the treatment team is less acute than the competition between the team and the psychiatrist. Ribner[33] summed it up, "In perhaps no other medical specialty does the physician find himself expected to work as a team member with those who are jealous of his power and certain that their abilities equal his."

Rappaport[34] attributed the unique position of the psychiatrist to his training in both medicine and psychology. He evaluated the interplay between psyche and the soma. Brill[35] believed that the physician is trained to be captain of the team, while Taxay[1] urged us to recognize the difference between cooperative ventures and the singular responsibility of the ship's captain, the airline pilot, and the physician. A committee of passengers does not navigate or fly, and only the physician directs health care. The practice of medicine is not a team activity, however well-intentioned and trained the members of the supporting services may be.

SOLUTIONS

The changing role of the psychiatrist and the perception that he simply signs prescriptions and medical records has resulted in psychiatrists leaving the public sector.[16,36] This trend can be reversed if we restore the prestige and clinical authority that belongs to the psychiatrist.

In New York State, I have recommended changing the Civil Service title, "team leader," to "administrative assistant to the physician." Secondly, the psychiatrist should have a management status, with responsibility and authority to supervise and evaluate the members of the team.[15]

Several states have contracted with a university department of psychiatry to provide university residents to work in a state mental hospital.[22,37,38] Knesper and Hirtle[39] found that system changes rather than financial benefits were the most persuasive to attract psychiatrists to state mental hospitals. Weintraub et al[38] asserted that recruitment strategies used by most states ignore administrative problems and focus on financial variables that are more subject to manipulation, such as salaries, fringe benefits, and bonuses.

Dr Krajewski, superintendent of Springfield State Hospital (Sykesville, Md), said that psychiatrists who pass up higher-paying positions to work in the state facilities are attracted by the opportunity to put their theories into practice with a varied and difficult patient population. "We give our staff the ball and let them try out their own game plan. We give a lot of flexibility in choosing therapies and we try to give a lot of support."[40]

The physician must have the support of the system in order to exercise his legal responsibility.[15] That support must come from Civil Service through appropriate titles for team leadership. These titles must leave no doubt as to who is clinical leader versus administrative leader. Secondly, hospital administrators, whether medical or nonmedical, must support the authority of the psychiatrist for his legal responsibilities. The physician must be allowed to exercise his clinical judgment free of intimidation concerning what the nonmedical team leader will think and how he will be evaluated. Finally, psychiatrists must achieve the authority that society usually assigns to them.[41] The psychiatrist must utilize his unique expertise in asserting his leadership of the team, thereby achieving the respect of the psychiatric team members for his authority.

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FROM THE LIBRARY

RAPPORT BETWEEN PSYCHIATRISTS AND PSYCHOLOGISTS

. . . Every psychiatrist realizes that emotional problems are closely connected with many factors: physical and psychic development, diseases incurred, and the environment in which one lives. The psychiatrist, in order to help people with emotional problems, must know the general pathology of childhood disease, all infectious diseases, all dystrophies, congenital or acquired; he must know the "disease process" which involves etiology, diagnosis, and treatment, all of which are closely knitted together, one in function of the other.

The psychologist believes he is qualified to make a diagnosis of mental abnormalities and to treat accordingly by relying only on psychologic testing.

. . . The psychologic technics give to psychiatry a scientific tool extremely helpful, and it would be unwise not to appreciate it to its full extent. However, this is only one part of the examination, and it is indispensable to study the patient as a whole in his somatic-psychic unit; it is necessary to see the constitutional factors or the acquired ones of his personality, and in many cases it is also necessary to study the physical-chemical components of the humors and of cells in order to reach a complete diagnosis with its therapeutic implication. The approach of the psychiatrist is a global one; it reaches a diagnosis and treatment by taking into consideration medical knowledge, psychologic findings, and social history. He makes a synthesis of all data and reaches his conclusions. It is easy to understand why he is better qualified to do this than the psychologist, the teacher, or the social worker; in fact, none of them has the indispensable medical knowledge to make a diagnosis and reach a therapeutic conclusion. . . .

The psychologist has to be the collaborator of the psychiatrist, who should ultimately and exclusively have the responsibility of the treatment. Many fantastic, useless, and outright dangerous initiatives in the field of therapy could be avoided if the treatment were left to the most qualified in the field. A common sense approach indicates that "nervous and mental disorders" should be treated by a physician who has specialized in that branch of medicine, that is, psychiatry.

This is an extremely important problem to which we are particularly sensitive because we feel that a lot can be done in the field of preventive mental hygiene, that the completely specialized and trained physician is not hampered in his therapeutic and research efforts. In the broad field of mental hygiene a more beneficial and effective contribution could be made by the psychologist if he would concentrate his efforts on the progress and development of psychologic testings and collaboration with the psychiatrist. The collaboration between the psychiatrist, the psychologist, and the social worker is, however, necessary and indispensable for the understanding of the dynamics of the situation. It is only through this teamwork that the best results are achieved in diagnosis and therapy. The practice of medicine and its different forms of therapy, however, should be restricted to the medical profession which should have entire responsibility.

G. EMIL PIANA, MD
(*NY State J Med* 1955; 55:672-673)

Rhabdomyolysis

Case Presentation

Robert Feldman, MD (Medical Intern): A 36-year-old black man was admitted for the first time to the New York Infirmary-Beekman Downtown Hospital with progressive generalized abdominal pain, lethargy, and confusion. The patient's history was fragmentary and was primarily obtained from his relatives.

Four to five days prior to admission, the patient had begun to pass dark brown urine, with a subsequent decrease in urine volume. For several days, he experienced diffuse myalgias, malaise, and vague abdominal pain, particularly in the suprapubic region, without radiation. He denied nausea, vomiting, and diarrhea.

The limited history included hypertension for 20 years, a left hip dislocation, and a seizure disorder. There was no history of diabetes, recent viral infections, muscle disease, sickle cell disease, nephrolithiasis, prostatitis, renal disease, or vascular disease. He denied the use of any medications but admitted to intravenous drug abuse in the past. A review of systems was otherwise not helpful. The family history was negative for polycystic renal disease, metabolic disease, and sickle cell disease. The patient was employed as a vendor in a fish market and lived alone; he denied abuse of alcohol and had discontinued cigarette smoking several years previously.

Physical examination revealed a lethargic but arousable black man, well developed and well nourished, in no acute distress. His blood pressure was 148/92 mm Hg; pulse rate, 88/min; respiratory rate, 18/min; and temperature, 101°F. His skin was dry with fair turgor; old healed scars at intravenous injection sites were present in both anticubital fossae. The sclerae were clear and nonicteric; the conjunctivae were slightly injected; the fundi were not visualized due to the patient's confused state. The pharyngeal mucous membranes were dry. The lymph nodes were diffusely enlarged, with a firm tender node palpable in the right axilla. The lungs were clear to percussion and auscultation. The heart rhythm was regular; a grade I/VI systolic ejection murmur was loudest at the left lower sternal border; no rubs or gallops were noted. The abdomen was flat and soft with tenderness to light palpation over the suprapubic area, but without rebound or guarding; bowel sounds were normal. The liver edge was felt 3-4 cm below the right costal margin; location of the spleen edge could not be determined. No abdominal masses were palpable. There was moderate costovertebral angle tenderness bilaterally. The rectal examination indicated a normal rectal sphincter tone, normal size and consistency of the prostate, and a small amount of brown stool which was negative for blood on Guaiac test. The extremities showed no cyanosis, tenderness, or edema.

The neurologic examination indicated a fluctuating mental status, with the patient oriented only to his own name, but unable to concentrate for additional mental testing. No motor or sensory deficits were detected. Deep tendon reflexes were normal; plantar responses were flexor; gait could not be observed.

The urine was dark brown and cloudy, with a pH of 5.0, specific gravity of 1.025, a trace positive test for glucose, three-plus test for protein, and a four-plus Guaiac test for blood. Microscopic examination showed 2–6 red blood cells, 1–3 white blood cells, and many bacteria. Random determination of electrolyte levels in the urine revealed the following: sodium, 40 mEq/L; potassium, 96 mEq/L; and chloride, 21 mEq/L. Toxicology screen of the urine revealed the presence of cocaine. During later hospitalization, the urine became progressively lighter in color and increased in volume.

The hemoglobin on admission was 16.7 g/dL; hematocrit, 48.7%; mean corpuscular volume (MCV), 94 femtoliters; white blood cell count, 4,000/μL, with 56% mature polymorphonuclear leukocytes, 8% immature polymorphonuclear leukocytes, 33% lymphocytes, 2% monocytes, and 1% eosinophils. The platelet count was 63,000/μL; prothrombin time (PT), 11.9 sec (control, 11.9 sec); and partial thromboplastin time (PTT), 34.2 sec (control, 26.4 sec). During later hospitalization the hemoglobin fell to 7.0 g/dL, and the hematocrit to 22.6%; the white blood cell count remained persistently low at 2,900/μl.

Serum electrolyte determinations indicated the following: sodium, 133 mEq/L; potassium, 5.6 mEq/L; chloride, 94 mEq/L; bicarbonate, 22 mEq/L; calcium, 7.7 mg/dL; and phosphate, 8.4 mg/dL. The blood urea nitrogen level was 103 mg/dL; serum creatinine, 10.5 mg/dL; blood glucose, 121 mg/dL; serum amylase, 347 U/L. The total serum protein level was 6.6 g/dL; the serum albumin was 3.6 g/dL.

Arterial blood gas determinations with the patient breathing room air indicated a pH of 7.4; $P\text{CO}_2$, 28 mm Hg; $P\text{O}_2$, 93 mm Hg; bicarbonate, 17 milliosmoles per liter; oxygen saturation, 97%.

The electrocardiogram showed normal sinus rhythm with a rate of 83/min; there were peaked T-waves in many leads but no acute S-T wave changes.

The opening pressure of cerebrospinal fluid obtained by

Based on a clinical conference held at New York Infirmary-Beekman Downtown Hospital, New York, NY, on October 16, 1987

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lumbar puncture was 17 cm water; the fluid was transparent and colorless, with a protein level of 44 mg/dL; glucose, 68 mg/dL, and chloride, 120 mEq/L. Microscopic examination revealed only one red blood cell and one white blood cell per microliter. Gram stain, India ink stain for toxoplasma, and acid-fast stains for tubercle bacilli were negative. Routine cultures of the fluid were subsequently reported as showing no growth.

The patient was admitted with a diagnosis of hypertension, acute renal failure, thrombocytopenia, and mental status changes possibly due to uremia or to cocaine; bacterial endocarditis was suspected. Peritoneal dialysis was carried out during the first five days in the hospital. However, the abdomen became increasingly tender following dialysis catheter insertion. As the patient became more alert though still confused, he became more uncooperative. After five days, he removed his dialysis catheter, which was replaced two days later; dialysis was continued for an additional two days. His urine output increased—up to 200-300 cc per day—and reached normal volume after one week. Urine cultures were negative. The serum myoglobin level was greater than 700 mg/dL (normal level, less than 50 mg/dL).

Initially, a diagnosis of endocarditis was strongly considered in this patient, in view of the fever, the heart murmur, and the history of intravenous drug abuse. On admission, the patient was treated with vancomycin and cephtrioxone, which were discontinued when a two-dimensional echocardiogram and blood cultures were reported as negative. However, in the light of increasing abdominal tenderness, even with negative cultures and cell counts on the peritoneal dialysate, the patient was started on vancomycin and cephoxitin. The abdominal tenderness subsequently resolved.

During this interval, the blood urea nitrogen (BUN) at first rose to 141 mg/dL, and thereafter during dialysis fell slowly to 91 mg/dL, though the serum creatinine remained elevated at 13.1 mg/dL, and the serum phosphate at 6.8 mg/dL. The serum bilirubin remained within normal limits; the serum alanine aminotransferase level (SGOT/ALT) initially was 325 units per liter, but later decreased to 81 U/L; the serum aspartate aminotransferase (SGPT/AST) was 92 U/L, and later 53 U/L. The serum lactate dehydrogenase initially was 2,214 U/L, decreasing slowly to 820 U/L; the MB fraction was 888 U/L initially, and after two weeks was 284 U/L. The initial serum creatine phosphokinase level was 7,858 U/L (normal range, 0-130 U/L), with a slow, steady decrease during hospitalization to a level of 331 U/L.

Mental confusion, agitation, and fluctuating states of consciousness alternated with periods of combativeness and refusal to permit further laboratory investigations. The patient was given oral haloperidol, but an acute dystonic reaction developed which necessitated discontinuance of the medication and treatment with an antihistamine. The patient was treated with transfusions for anemia, and propranolol for hypertension, with good control.

Our clinical impressions included acute renal failure, with changes of mental status secondary to renal failure and cocaine abuse; rhabdomyolysis; and hypertension.

RADIOLOGIC PRESENTATION

George Braff, MD (Chief of Radiology): The chest film showed initially prominent hilar markings, no doubt vascular in origin, but no acute infiltrates. During the subsequent hospital course, there were thought to be infiltrations at the lung bases.

The plain film of the abdomen showed absence of one psoas margin; however, absence of one or both psoas margins can be seen in 25% of patients. The measurement of kidney size usually equals the linear height of three to four vertebral bodies. Here, the upper margins of the kidney could not be seen due to overlying bowel gas. The overall hazy appearance of the abdomen on this film suggested the presence of intraperitoneal fluid, which presumably was present prior to the peritoneal dialysis.

The ultrasound examination of the kidneys showed both to be slightly enlarged; they appeared round, with accentuation of the renal pyramids. These findings suggest some sort of acute insult that contributed to the renal failure.

Mary Chaglassian, MD (Medical Resident): Acute renal failure is defined as a deterioration of kidney function over the course of a few hours or days, resulting in the accumulation of nitrogenous wastes, leading to imbalances of electrolyte, fluid, and acid-base metabolism. Frequently, it is accompanied by oliguria and anuria; however, normal volumes of urine may be seen with progressive decline of renal function; the total urine volume may be highly variable. The causes of acute renal failure can be divided into three general categories: pre-renal azotemia; post-renal azotemia, due to obstructive uropathy; and intrinsic renal damage.

Pre-renal azotemia is associated with reduction in both renal perfusion pressure and renal blood flow. The kidney is not intrinsically diseased but is responding to a state of apparent or actual intravascular volume depletion by maximally conserving sodium and water in order to maintain adequate fluid and electrolyte homeostasis. The assessment of this condition should include measurement of orthostatic changes in blood pressure and pulse, assessment of skin turgor, and evaluation of possible signs of congestive heart failure. Other findings include urine osmolality of more than 500 milliosmoles per kilogram, a urine sodium level of less than 20 mEq/L, a urine-to-plasma urea nitrogen ratio of more than 8, a urine-to-plasma creatinine ratio of more than 40, and a fractional excretion of sodium of less than 1.

Post-renal azotemia can be secondary to obstructive anatomic changes, as with prostatic enlargement; or it can be functional. Bladder neck obstruction can be evaluated by suprapubic palpation and percussion, and by urethal catheterization with measurement of postvoiding residual volume. Obstruction of both upper urinary tracts may be due to retroperitoneal fibrosis, space-occupying lesions (tumors or abscesses), surgical accident, or intra-urinary tract occlusions caused by stones, sloughed renal papillary tissues, blood clots, or pus. Unilateral ureteral obstruction may lead to azotemia in the presence of a single kidney, or with a severely diseased contralateral kidney.

Obstruction to urine flow can also occur within the kidney due to intrabular precipitation of poorly soluble material such as uric acid, oxalic acid, methotrexate, sulfonamide, and perhaps myeloma protein.

Symptoms of intermittent gross hematuria, flank or groin pain, pyuria, hyperuricemia, prior history of analgesic abuse, sickle cell disease, or diabetes mellitus all may suggest an underlying condition that could result in ureteral obstruction. In addition to a good medical history, a careful rectal and pelvic examination is important in evaluating obstructive uropathy. A plain radiograph of the abdomen may detect retroperitoneal disease or urinary tract calculi. If obstruction of the upper urinary tract cannot be excluded by ultrasound, then infusion pyelography, computed tomography, or retrograde pyelography may be required.

The diagnosis of acute renal failure is one of exclusion, since pre-renal blockage, post-renal obstruction, and other internal disorders may all produce identical clinical syndromes of deteriorating renal function. Underlying causes of acute renal failure include vascular diseases such as vasculitis, malignant hypertension, arterial or venous occlusion, or thrombotic thrombocytopenic purpura; glomerulonephritis; interstitial nephritis secondary to drugs, hypercalcemia, infection, or idiopathic causes; acute tubular necrosis secondary to pigment deposition, as in hemolysis after transfusion reaction or with malaria, or in instances of rhabdomyolysis from muscle trauma, coma, heat stroke, severe exercise, muscle disease, or potassium and phosphate depletion, and as a result of nephrotoxins such as radiographic contrast materials, anesthetic agents, heavy metals, and antibiotics.

Acute renal failure may be precipitated by either renal ischemia or a nephrotoxic agent and can be perpetuated by tubular obstruction due to cast formation, accumulation of cellular debris, development of interstitial edema with a decreased osmotic pressure coefficient, or backleak of filtrate across the damaged tubular epithelium.

The composition of the urine may assist in differentiating between pre-renal azotemia and intrinsic acute renal failure. In the former, the tubular system maximally reabsorbs sodium and water in order to maintain total body fluid homeostasis. The amount of urine formed is small and is essentially devoid of sodium; its high concentration is due to the presence of urea and other nitrogenous wastes being excreted in the smallest possible volume of water.

In acute intrinsic renal failure, the limited glomerular filtrate may be modified only minimally by the damaged tubules; thus the levels of the chemical constituents in the urine tend to correspond closely to those of the plasma. The urine concentration will be less than maximal and will contain unreabsorbed sodium. When the urine osmolality is less than 350 mosm/kg, the urine sodium content greater than 40 mEq/L, the urine-to-plasma ratio of urea nitrogen less than 3, and the urine-to-plasma ratio of creatinine less than 20, the diagnosis is more likely to be acute renal failure due to intrinsic kidney disease.

In general, when acute renal failure follows a hemodynamic event such as shock or is secondary to nephrotoxicity, the resulting urinary sediment may contain epithelial cells, cellular debris, and typical pigmented brown tubular casts. A small amount of microscopic hematuria, pyuria, and one-plus or two-plus proteinuria are also compatible with the diagnosis of intrinsic acute renal failure. A protein content in the urine of more than 3 g per 24 hours and the presence of red cell casts suggest glomerulonephritis. When the urine is largely free of sediment abnormalities, azotemia is most likely due to either pre-renal or post-renal causes. In pre-renal azotemia, the sediment may contain only granular casts without tubular cells or tubular casts.

Three findings constitute the characteristic triad seen in patients with rhabdomyolysis: pigmented granular casts in the urine sediment; a positive orthotolidine (Hematest) reaction in the urine supernatant indicating the presence of heme; and a marked elevation in the plasma level of creatine phosphokinase (CPK) due to release of this enzyme from damaged muscle cells.

This patient's important findings consisted of oliguria with dark brown cloudy urine, a three-plus positive orthotolidine reaction for occult blood in the urine, pigmented granular casts in the urine sediment, urine spot-electrolyte determinations with a sodium content of more than 40 mEq/L, a serum CPK of more than 7,800 U/L, a plasma myoglobin level of more than 700 mg/dL (normal range, less than 50 mg/dL), hyperkalemia, hypocalcemia, hyperphosphatemia, marked azotemia with a BUN of 103 mg/dL, which continued to rise at the rate of more than 10 mg per day, and a serum creatinine of 10.5 mg/dL, which continued to rise at the rate of more than 0.5 mg per day. Bilateral mild kidney enlargement with absence of obstruction was revealed by ultrasonography. All of these findings pointed to the acute event of rhabdomyolysis, which Dr Maharam will discuss.

Irwin Sharkey, MD (Chief of Medicine): What is your management plan from this point on?

Dr Chaglassian: The patient has been transferred from the medical intensive care unit to an inpatient nursing unit. Two days ago, the BUN was 60 mg/dL, and the creatinine was 8.5 mg/dL, which represented significant decreases in each level. His urine output has increased to more than 1.5 L/day and he appears to be entering the diuretic phase of acute renal failure. He has abdominal distention and has been febrile, so his therapy with vancomycin has been continued in order to control the suspected peritonitis.

Dr Sharkey: Does the patient have any skeletal muscle symptoms now, such as pain or tenderness?

Dr Chaglassian: No.

Yehuda Handelsman, MD (Medical Resident): Why does the serum amylase remain so high? Do you think it is secondary to poor excretion of the enzyme because of the renal failure, or is there likelihood of pancreatitis?

Arturo Constantiner, MD (Director of Nephrology): A urine amylase determination was requested but was difficult to obtain. In acute renal failure, the serum level of amylase tends to be high. In this patient, the serum amylase content was fluctuating up to a level of 1,000 U/L or more, which is much higher than we ordinarily see with acute renal failure alone. However, in view of his improvement and the subsidence of his gastrointestinal symptoms, we felt that the serum amylase level was most probably due to the acute renal failure only.

Dr Sharkey: One or two patients with rhabdomyolysis are seen here every year. All of the cases in my own experience occurred in alcoholics; yet we could never be certain that the patients did or did not have polymyositis. I find it hard to believe that trauma to muscle tissue during and after an alcoholic binge can be caused simply by lying for long periods of time on a hard floor, leading to myoglobinuria of such severity as to require hospitalization. If the cause of rhabdomyolysis were such a simple one, we should see more of such instances, since drug abuse and alcoholism are so common.

Lewis Maharam, MD (Medical Resident): Rhabdomyolysis literally means striped muscle dissolution, that is, skeletal muscle injury, reversible or irreversible, which alters the muscle cell membranes sufficiently to allow the escape of cell contents into the extracellular fluid. These cell contents include creatine phosphokinase, glutamic oxalacid, oxalacidic transaminase, lactic dehydrogenase and aldolase, the heme pigment myoglobin, potassium, phosphates, and purines. Rhabdomyolysis may or may not result in visible myoglobinuria with brown urine, depending on the amount of the myoglobin released into the plasma, the glomerular filtration rate, and the urine concentration. Therefore, the more appropriate descriptive term for the syndrome is rhabdomyolysis, rather than myoglobinuria.

The earliest modern reports related rhabdomyolysis to crush injuries in World War I. Later, Bywaters and Beall[1] in 1941 were the first to study this syndrome during the Blitz of London in World War II.

The clinical presentations commonly include muscle swelling, pain, and tenderness; systemic symptoms include anorexia, vomiting, nausea, and frequent tiredness, but occasionally only the complaint of dark urine. However, some patients have no subjective complaints. The presenting clinical picture may in great part reflect the immediate cause of the rhabdomyolysis, such as in crush injury with muscle trauma or compression, or a fascial compartment syndrome.

The most common causes of rhabdomyolysis[2] are as follows:

- Trauma, the leading cause, resulting in muscle injury, myoglobinuria, acute renal failure, and sometimes vascular and fascial compartment compromise.
- Acute exertional rhabdomyolysis associated with excessive muscular activity, usually in a hot environment. Its particular occurrence in athletes in poor physical condition is controversial.
- Physical seizures, leading to rhabdomyolysis secondary to excessive muscular activity during the seizure, and possibly to a minor component of muscle compression during the period of post-ictal stupor and confusion.
- Delirium tremens with myoglobinuria due to excessive muscular activity.
- Drug-related rhabdomyolysis resulting from muscle compression during prolonged coma. Virtually every drug that can cause prolonged coma has been blamed, most commonly heroin and barbiturates. Yet some drugs can cause rhabdomyolysis without associated coma. Intravenous amphetamines can be associated with a syndrome of cardiovascular collapse, disseminated intravascular coagulation, and rhabdomyolysis. Succinylcholine, especially in prepubertal patients, and rarely amphotericin B, can be associated with rhabdomyolysis. Cocaine intoxication can result in severe hyperthermia, bizarre behavior, and rhabdomyolysis with acute renal failure.[3] The cause of the renal failure may be a combination of exertion, hyperthermia, and direct toxic effects of the drug.
- Arterial occlusion resulting in significant ischemia, muscle injury, and release of enzymes.
- Heat injury as with heat stroke, heat cramps, malignant hyperthermia, or electrical injury.
- Infections, especially with influenza, with *Clostridia* involving myonecrosis and gas gangrene, or rarely with *Leptospira* and *Shigella* organisms.[2]
- Myopathies, both hereditary and acquired.
- Toxins in rare instances, such as sea snake bites, spider bites, and Haff disease associated with a lagoon in the Baltic area. Between 1920 and 1950, fishermen in this vicinity were suddenly seized with severe pain in the limbs, great weariness, and myoglobinuria. These symptoms were attributed to poisoning by a resin deposited in the Haff lagoon from the wastewater of cellulose factories.[2]

In 1982, a study was conducted at the Denver University Hospital of 87 patients with a discharge diagnosis of rhabdomyolysis recorded over a three-year period.[4] Alcohol abuse, muscle compression, and seizures were the most common etiologic factors; multiple factors contributing to muscle injury were identified in 51 of the 87 instances (59%). Other factors that can potentiate rhabdomyolysis are dehydration, hypokalemia, hypophosphatemia, and acidosis, including lactic acidosis with exercise.

An alcoholic who is suffering from starvation, volume depletion, and potassium and phosphate depletion is at particular risk for the development of rhabdomyolysis.

Four mechanisms for the acute renal failure seen with this syndrome have been proposed, without certainty as to which is definitely responsible: intratubular obstruction due to precipitation of myoglobin in the ascending limb of the loop of Henle; intratubular precipitation of uric acid crystals with resultant obstruction; renal ischemia due to potent vasoconstrictive substances released by proteolytic enzymes from the damaged muscles; and the direct nephrotoxicity of myoglobin or its breakdown product ferrihemase, with tubular necrosis, intranephronal obstruction, and backleak of the filtrate.[5]

Myoglobin, an iron-containing, oxygen-binding respiratory protein in skeletal and smooth muscles, is a single-chain protein, with a molecular weight of 16,800 daltons. It binds loosely to alpha-2 globulin in the plasma but not to haptoglobin. The maximum myoglobin-binding capacity of alpha-2 globulin is approximately 20 mg/dL of plasma; the small amount of myoglobin normally present in the plasma is detected only by sensitive radioimmunoassay. The renal threshold for myoglobin is just above the level of maximal binding of myoglobin to plasma protein (approximately 21 mg/dL of plasma).[6]

Above this threshold plasma concentration, the fractional clearance of myoglobin is about 75% of the level of the glomerular filtration rate. The plasma begins to assume a pinkish color at myoglobin concentrations above 100 mg/dL. The renal clearance of myoglobin is normally high at plasma concentrations above the plasma protein binding capacity, so that the plasma is not likely to present the pinkish color of myoglobin, unless the renal glomerular filtration rate is significantly reduced and/or the destruction of muscle is severe. Myoglobin is distributed over approximately 40% of the body mass. The myoglobin content of skeletal muscle is about 4 mg per gram of muscle tissue.

Myoglobin in the urine, initially pink, is rapidly converted to the brownish-black pigment met-myoglobin. Myoglobinuria is usually not detectable until plasma levels exceed 1.5 mg/dL, an amount about equal to the dissolution of 100 g of skeletal muscle. Physical training can greatly increase the myoglobin content of skeletal muscle and, therefore, for any quantitative amount of muscle destruction, a well-trained athlete will have a greater likelihood of developing myoglobinuria than would a sedentary individual.[6]

Myoglobin and hemoglobin are both soluble when heated in dilute acetic acid, whereas plasma proteins are usually insoluble. The fact that myoglobin is soluble in 3.2 molar ammonium sulfate, whereas hemoglobin is not, serves as a chemical test to distinguish between hemoglobinuria and myoglobinuria. At a pH of less than 5.4, myoglobin and hemoglobin dissociate to globin and ferrihemate; the latter is nephrotoxic.

The diagnosis of rhabdomyolysis depends on the history and physical examination; the clinical setting probably provides the most important clues. Muscle swelling and tenderness should be sought on physical examination, as well as evidence of a muscle-fascial compartment syndrome. The laboratory findings include a dark amber color to the urine; in the absence of red blood cells, a positive benzidine test in a fresh urine specimen suggests myoglobinuria or hemoglobinuria. The serum is usually clear in myoglobinuria and is usually pink in hemoglobinuria. Spectrophotometric determinations will assist the differentiation; also the ammonium sulfate test will precipitate the hemoglobin and not the myoglobin. The most reliable method of differentiation is by electrophoresis or immunophoresis; radioimmunoassay has also been used.[2]

Release of intracellular creatine, which can be converted to creatinine, can result in elevated serum creatinine levels out of proportion to the level of blood urea nitrogen. Release of intracellular potassium from injured muscle tissue may lead to increases in serum potassium, sometimes to life-threatening levels. Serum creatine phosphokinase, phosphate, and uric acid levels are often increased, with decrease of serum calcium possibly secondary to hyperphosphatemia and to deposition of calcium in damaged muscle.

The initial management of these patients, after careful assessment to rule out volume overload, emphasizes the induction of diuresis, using mannitol intravenously or a normal saline infusion with intravenous diuretics. Alkalinization of the urine with sodium bicarbonate should be attempted only after assessment of the patient's serum calcium level. Careful measurement of fluid intake and output is required in order to avoid volume overload. When muscle-fascial compartmentalization is a problem, prompt fasciotomy is indicated.

If renal failure has developed and the initial attempts at inducing diuresis with optimization of the volume status fails, management methods are those usually used in cases of established renal failure. Special attention should be paid to the patient's serum potassium level to avoid life-threatening hyperkalemia. Hyperphosphatemia can be treated with oral phosphate binders such as aluminum hydroxide gel. The level of serum calcium should be monitored; severe hypocalcemia should be treated so as to include correction of the hyperphosphatemia. Elemental calcium, if used, must be administered carefully due to the potential for severe rebound hypercalcemia during the recovery phase following renal failure.[2] Peritoneal dialysis or hemodialysis may be necessary to correct severe azotemia, volume overload, or severe hyperkalemia. The clinician must carefully observe the patient for development of the muscle-fascial compartment syndromes.

Most patients with acute renal failure secondary to rhabdomyolysis improve and will eventually recover with normalization of renal function; the time course is variable between seven and 20 days, the mean being 16 days. Five to ten percent of these patients have died, but usually with other complications.[2-6]

Acute exertional rhabdomyolysis was first described in military recruits who were subjected to vigorous exercise, including squat thrusts, marching, and running. It is characterized by pain, swelling, and tenderness due to necrosis of skeletal muscle following such strenuous exercise. Prospective studies at the Marine Corps Recruit Depot at San Diego[7] revealed a 39% incidence of asymptomatic elevated serum myoglobin levels after the first six days of basic training. At the Marine Corps Recruit Depot in South Carolina, myoglobinemia developed in one third of 63 recruits[8]; one sixth of the recruits (11 cases) were hospitalized with exertional rhabdomyolysis and evidence of renal failure, but there was no mortality.

As a sidelight on the present widespread commitment to jogging, the first case of acute renal failure and rhabdomyolysis in a marathon runner was reported in Israel in 1979.[9] The subject had had a viral infection with sore throat which had spontaneously resolved one week prior to the race. Beginning a few hundred meters into the race, he developed severe gluteal-region cramps extending to the thighs and calves; these decreased, however, and he finished the race. The first urine he passed after the race was very dark, and during the next 30 hours he passed no urine but gained 3 kg in weight. On the day of admission, he was nauseated, vomited several times, and had severe muscle tenderness in the thighs and gluteal regions. After dialysis, the patient's renal function returned to normal within ten days.

Many other instances involving marathon runners and runners in general have subsequently been reported.[10] A South African study reported ten cases over a nine-year period during the Comrade Marathon, a 90-kilometer (56.25 miles) course. Nine runners developed renal damage, three patients required hemodialysis, and one required peritoneal dialysis; all survived.[11]

A 24-year-old man was admitted to the Charity Hospital in New Orleans with myalgias, muscular weakness, and dark urine after a basketball game.[12] He had not engaged in strenuous activities for the three previous months. He was oliguric for 36 hours after admission, but treatment with a low protein diet, fluid replacement, and bed rest was followed by return of renal function; he remained well.

A master of tae kwon do (a Korean martial arts discipline) broke 2,035 boards in four hours but eight hours later back ache and acute renal failure developed.[13] Examination showed extensive bruising of the right forearm; the urine was positive for blood on dip-stick test, but no erythrocytes were seen on microscopic examination. The patient recovered after four hemodialysis treatments.

Acute renal failure due to exertional rhabdomyolysis was reported in a 22-year-old man who engaged in a daily intense weight-lifting routine; the complication resolved within four days and the patient did well.[14]

A 19-year-old university student presented with oliguria and myalgia after an intramural wrestling match.[15] Although an experienced wrestler, he had had insufficient opportunity to condition himself for the match. After receiving intravenous mannitol, he did not require hemodialysis, and he recovered.

Several days after a three- to four-hour period of jet-skiing, a 31-year-old patient developed rhabdomyolysis, with an initial complaint of pain and swelling of the right arm.[16] The serum CPK was reported as 40,900 U/L (normal, less than 140 U/L). Vigorous normal saline hydration preserved renal function, and the patient did well. Many marathon runners and even common daily joggers may not be as well trained as they should be or are training incorrectly.

James Rahal, MD (Director of Infectious Diseases): Should not fever occur with rhabdomyolysis?

Dr Maharam: It occurs irregularly, but without a consistent relationship to the degree of rhabdomyolysis.

Arturo Constantiner, MD: The persistence of fever in this patient cannot be adequately explained on the basis of the rhabdomyolysis. In the early phase of this patient's hospital course, certain clinical data created difficulties in establishing a correct diagnosis. We had to consider the possibilities that a condition other than rhabdomyolysis was causing the acute renal failure, or that rhabdomyolysis was superimposed on underlying renal disease.

The patient's history of hypertension since age 12, extremely unusual in itself, had to be considered as one cause of underlying renal insufficiency. In addition, his presentation with three-plus proteinuria is not the typical presentation of the patient with rhabdomyolysis. Among the initial diagnostic possibilities was bacterial endocarditis, which can present with proteinuria and hematuria. The negative blood cultures and the four-plus positive dipstick test for blood in the urine, but with only two to six red blood cells visible per high-power field, were not supportive of the diagnosis of bacterial endocarditis, nor was the hematocrit of 46%. Amyloidosis was suspected in view of the three-plus proteinuria, the large kidneys, and the history of drug addiction with "skin popping" to which this patient admitted. Skin-popping has been reported as a cause of amyloidosis in patients with chronic renal insufficiency.[17]

The prompt decision to dialyse this patient shortly after admission was based on two main indications:

- The altered mental status. A BUN of 103 mg/dL in a patient with chronic renal insufficiency would probably be well tolerated, whereas this patient was quite lethargic with such an elevation of urea nitrogen. We opted to lower that level of blood urea by dialysis, in order to determine if his mental status was due to a uremic encephalopathy or to some other condition.
- The development of progressive acidosis. His serum bicarbonate level on admission was 22 mEq/L, but within a few hours it dropped to 9 mEq/L.

The mode of dialysis for rhabdomyolysis can be either the peritoneal or the vascular route. Both forms of therapy are adequate.

Hemodialysis is used depending on the ease of obtaining a dialysis machine in the unit and on the availability of the nursing staff. In view of this patient's history of drug addiction and thrombocytopenia, we considered that he might be at high risk for HIV infection, and that therefore peritoneal dialysis would represent less risk of exposure to HIV infection for the dialysis staff and for other hemodialysis patients. The most recent recommendations of the Centers for Disease Control concerning dialysis patients with positive tests for HIV are really the same as for patients with hepatitis, ie, an isolation room, gowning and use of gloves for nurses with change after each contact with the patient.[18] Unfortunately, in this hospital, we did not have an isolation room available; therefore, at that point, for the reasons I have just stated, we felt it was safer for all concerned to use peritoneal dialysis.

The matter of alkalinizing the urine for excretion of myoglobin is controversial. You should alkalinize with bicarbonate when the patient is acidotic; but if you must alkalinize the urine while the patient has a primary respiratory alkalosis, the use of acetazolamide is preferable.

The effectiveness of mannitol is not substantiated by adequate studies in patients in whom acute renal failure has already developed. Mannitol can be effective by increasing the osmotic load within the lumen of the renal proximal tubule and thus decreasing the reabsorption of toxic substances, including myoglobin, in that location. Mannitol may induce a diuresis and facilitate the management of acute renal insufficiency, but I doubt that mannitol will prevent the persistence or progression of the renal failure.

FINAL CLINICAL DIAGNOSES

Acute renal failure, most probably due to rhabdomyolysis with secondary toxic effects of myoglobin on the kidneys.

History of intravenous drug abuse with "skin-popping."

History of hypertension.

History of seizure disorder.

Note: Subsequent to the original presentation of this conference, cocaine has been reported as an increasingly frequent cause of rhabdomyolysis.[19,20]

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FROM THE LIBRARY

MYOGLOBINURIA

. . . The dark brown color of the urine is typical of myoglobinuria. It is generally described as reddish at first, and it turns brown because of the reduction of the oxymyoglobin that is excreted to metmyoglobin which is a brown color. There are many causes of myoglobinuria. Among them are the crush syndrome and high-voltage electric shock. Some cases have been associated with dermatomyositis or polymyositis. Others have been reported with thrombosis of a large vessel resulting in a considerable degree of muscle necrosis. . . .

The clinical picture of idiopathic paroxysmal myoglobinuria is characterized by recurrent attacks. The patients have perhaps two or three attacks a year, characterized by muscle pain and tenderness, weakness, soreness, and stiffness. The muscles commonly involved are in the calves, thighs, buttocks, and back, generally the lower extremities, although any muscle groups may be affected. The most typical precipitating factor is exercise. The amount may vary from patient to patient. Some patients can predict exactly how much and what kind of exercise will produce pain and dark urine. Other patients will tolerate varying amounts of exercise at different times. Associated symptoms that may occur include fever, malaise, abdominal pain, and, in severe cases, the patient may look like one who is having an acute hemolytic crisis. This is unusual, however. Most of the patients who have been described have had moderate amounts of fever and malaise while they were excreting dark urine. The urine becomes dark a few hours after the exercise is performed. Incidentally, exercise is only a precipitating factor in about half of the patients who have been reported. . . .

The dark urine appears two or three hours after the exercise, generally lasts for a day, and is gone in about twenty-four hours. The course of disease may show striking variations. Occasional patients have been admitted to the hospital with a first attack and have died within twenty-four hours. In others the disease has followed a mild course and appeared to be a benign illness. Typically the onset is early in life, before the age of fourteen years, as reported in about a third of the known patients. In other patients it has been recognized in the third or fourth decade, but there has usually been a history of muscle weakness and intolerance to exercise in early youth. . . .

RICHARD WEISKOPF, MD
(*NY State J Med* 1963; 63:2512-2517)

Anything for Thanksgiving?

PASCAL JAMES IMPERATO, MD

On cool Thanksgiving mornings in the 1940s children in many areas of New York City dressed themselves as tramps and hobos and with paper bags in hand went from house to house shouting, "Anything for Thanksgiving?" The leaves that had fallen in the weeks before carpeted the sidewalks and lawns in my neighborhood and we purposely shuffled our feet through them as we made our way towards the stoops. We did this more to pre-announce our arrival than for the pleasure of it. Those of us with Thanksgiving begging experience thought that people who were forewarned of our arrival were better disposed to giving us something than those who were suddenly startled by the ringing of their doorbell.

Begging is among humanity's last grasps for survival, a final attempt on the part of people to actively control their fate. It carries historical legitimacy, is socially approved, and is subject to abuse. It is the latter fact, coupled with the view that government should meet these needs, that has lessened tolerance for begging in the United States. Even in those years when I went begging on Thanksgiving, intolerance for beggars was announced on small signs attached to the front of houses. They read: "No peddlers or beggars allowed." I thought it strange that peddlers and beggars should be grouped together since in my child's view their only shared characteristic was itinerancy. Yet many peddlers of those years were little better off than beggars. What set them apart was the peddler's accumulation of a small amount of excess capital with which to purchase goods for resale. Still they were despised by many and viewed as a nuisance by most.

Thus in donning the attire of beggars on Thanksgiving we temporarily entered the world of those who were despised. We were consciously aware of this, to the point that we didn't beg at those houses where warning signs were posted. What we weren't aware of were the root causes of this intolerance which in part lay in the Great Depression of the 1930s. Unemployment and bank failures had reduced many to paupery. Those who were barely holding on to their homes and jobs were constantly besieged by beggars and peddlers and by confidence men who duped them at their doorsteps with ingenious pretenses and schemes. No wonder, then, that the signs went up.

Begging began early on Thanksgiving morning and ended at noon as if by convention. We put on our fathers' old jackets, coats, and fedoras and streaked our faces with cork blackened over the flames of the kitchen stove in imitation of the smudges seen on hobos and tramps.

Traveling in groups, we moved through the neighborhood within the geographic limits set down by our parents. This encompassed the area of Tudor Village, a development of some 300 homes that had been built in 1928–1929 in southwestern Queens County, New York. Surrounding it were farms, woods, and open fields that had been given a reprieve from development by the Great Depression and World War II.

As the door of a house opened we shouted, "Anything for Thanksgiving?" That shrill cry carried a nuance of threat and intimidation, of possible negative consequences if our demand weren't met. People shared with us the little they had, a walnut, a peanut, a penny, an apple, and on a rare occasion a piece of candy. As the doors opened pleasant kitchen aromas drifted out over the stoops. But on some stoops no aromas came out through the opened doors. Some people didn't open their doors at all and others sadly said they had nothing to give. We understood this because we ourselves had so frequently heard our parents speak of the Depression and saw it as some abstract monster that took people's homes away from them and put men out of work.

Despite the hard times, most people made an attempt to feast on Thanksgiving. To be sure these feasts were modest by today's standards. But they included turkey, cranberry sauce, sweet potatoes, nuts, fruits, and desserts. Through the vehicle of their children's begging, neighbors shared their feast with one another. For what we brought home was in turn given to everyone.

By late morning we reached the eastern limit of our begging area, the fence to Balsam Farm's northwest pasture. There were 150 milking cows on the farm, chickens, ducks, geese, and, to our delight, calves in the spring. Two blocks from the farm was a row of stores, several of which were open for business on Thanksgiving morning. We first stopped at Ida and Harry Rosen's candy store where Ida, dressed in a maroon dress trimmed with a white collar provided by the Borden Dairy Company, gave us a piece of candy. At John Schriefer's delicatessen we received pretzels from a large slanted tin near the door and at Solomon Fierer's drugstore a penny each. Our final stop was Big Bob's Bar and Grill, later renamed Monahan's Bar and Grill, which stood at 85th Street and Old South Road next to Rosen's candy store. By mid-day a number of men

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from the village were usually seated at the bar. They were always in a gay mood from a few drinks which moved them to a rewarding level of largesse. My mother viewed Big Bob's and all bars as dens of iniquity. Although she said my father would never set foot in Big Bob's, I once saw him exiting from the side door but didn't tell her. We were told not to cross the threshold but that we could beg from outside the door. It was hard to obey this order, especially since other children always exited with fistfuls of pennies. However, we did obey but found an ingenious solution to our dilemma. We stood at the open door and asked the men to throw coins from their bar stools into our open bags. This game usually netted us our greatest cash take of the morning.

The custom of begging on Thanksgiving morning began to die out in my neighborhood around 1949. My parents said it had originated in the early 1930s during the Great Depression and that it was out of place in better times. It was replaced by a then-new custom which my younger brothers participated in on Halloween called "Trick or Treat."

In 1987 Halloween fell on a Saturday and my wife asked me to accompany our three children as they went trick-or-treating. In place of wearing old clothes they were dressed in elaborate costumes, one as a witch, another as a pirate, and our two-year-old son as a pumpkin. They didn't carry paper bags but large plastic pumpkins whose contents would be carefully inspected for pins, glass, razors, and poison after we got home. It had been close to 40 years since I had last gone from door to door on a quest for treats. Whereas we had gone alone, these children had to travel under the watchful eye of an adult. But unlike us they were met with a material bounty we could never have imagined. People greeted them with baskets and bowls full of packaged candies whereas we had picked our peanuts and pennies from opened palms.

There is something almost mechanical, unfeeling, and dutiful about this transfer of trick-or-treat goods. Children don't receive treats unknown to them the rest of the year, as we did, but increased quantities of well known luxury and excess. This deprives them of the joy we felt when we begged and of the excitement of telling others what they received. It also struck me that the focus had shifted from the act of receiving to parading one's attire for others to see. Trick-or-treating seems but a vehicle through which to display the uniqueness and quality of costume and in so doing to best one's fellows. Yet when we begged we all looked alike and no one gave it a thought.

After about an hour the pumpkins were almost filled to capacity. I looked at them and realized that each one contained more than any of us had ever received in several years of Thanksgiving begging. Some children, their bags, pumpkins, and other receptacles completely filled, playfully ran past houses where people waited in vain to give them treats.

When we entered the last street my two-year-old son followed a group of children up a steep slope to get to the front door of a house where a woman waited with a tray full of candy bars. It struck me that his struggle up that slope was not only physical but also symbolic. For he was weighed down by the bounty represented by both his costume and the contents of his pumpkin. As I looked at him I thought of another slope and of another time and place where children climbed in search of necessities. They were weighed down not by clothing, because they didn't have any, but by hunger and malnutrition. I followed them up the side of a sand dune in northern Mali, taking them to a feeding station set up by the US Agency for International Development. They were grateful for powdered milk and sorghum whereas my son was indifferent to most of the food he had acquired. They reached out with hope while he reached out because he had been told to. They knew nothing of luxury and comfort; he knew nothing of misery and starvation.

Those of us who filled the autumn air with the cry of, "Anything for Thanksgiving?" had more in common with the children on the sand dune than with those on the grassy slope. We knew hardship and adversity, had tasted deprivation, and lived under the constant threat of losing the little we had. Although we were symbolic beggars for but a few hours a year, we thought and reflected and came to the conclusion that this could become our permanent lot. For beggars and poverty were not mere abstractions or flashes on a television screen of faraway places and people unlike ourselves. Beggars frequented our neighborhood, often with children in tow. People gave them food and money and in return were reminded what they themselves might become. Thus in becoming beggars on Thanksgiving, we slipped into a dreaded world that might one day be ours. No wonder, then, that we hurriedly took off our beggar's clothes as soon as we returned home. We cherished the treats but not the disguise.

We had to be home by noon to help my mother set the table and get other things ready for dinner, which usually began in the early afternoon. The turkey, usually weighing 20 pounds or more, had been in the oven since early morning. There were no frozen turkeys in those days and no home freezers so my mother had to order the turkey from the butcher at the A & P well ahead of time and pick it up the day before, head and feet still attached. Jell-O desserts were put out on a wall in the garden to harden and the cider was chilled in the empty milk box in the back yard.

Our Thanksgiving table was a work of magic. It looked splendid to our eyes, with its shining crystal, silver, and heirloom napkins. We never gave a thought as to how our parents created this special feast. For in our view they were magical themselves, and had a share in the powerful world of spirits, tooth fairies, and Santa Claus. When my parents gave thanks before the meal began they spoke with feelings that had been forged in adverse times among people who had won the struggle to survive. The meaning of their thanks has now disappeared in the glow of abundance that has touched most of our lives. That abundance silenced the autumn air and made Thanksgiving beggars but a vision from yesteryear.

Roswell Park

A pioneer in the surgical treatment of epilepsy

EMILIO D. SORIA, MD; EDWARD J. FINE, MD; MARGARET W. PAROSKI, MD

In 1883, the Chair of Surgery of the Medical School of the University of Buffalo (New York) was made vacant through the retirement of Edward M. Moore, and Roswell Park was chosen to fill the position[1] (Fig 1). His arrival in Buffalo was opportune; this was the transitional period at the threshold of modern surgery, and during the next 30 years his became one of the leading names in the field of medicine.[2]

In 1898, Dr Park created in Buffalo the first facility in the world dedicated specifically to cancer research, known as the New York State Pathological Laboratory of the University of Buffalo.[3] The cancer research program soon grew too large for this laboratory, and the Gratwick Research Laboratory was built in 1901.[3] This later became the Roswell Park Memorial Institute, the oldest and one of the best known educational facilities in the world for the research and treatment of cancer.[4]

In 1901, the Pan-American Exposition was held in Buffalo, and Dr Park was made Medical Director. The exposition attracted thousands of visitors to the city. Among the many illustrious guests was President William McKinley. On his visit to the Exposition, President McKinley was shot at the Temple of Music.[5] Park was out of town at the time, and it was hours before he could reach the President.[6] The President's severe abdominal injuries required immediate surgery, which was performed by Dr Matthew D. Mann. The failure to avert the death of President William McKinley, however inevitable, was a bitter disappointment in Park's life, from which he suffered for years.[6]

Park is still well remembered for his considerable contributions to the medical sciences. He wrote 167 articles, textbooks, and monographs and lectured before numerous societies throughout the world.[7] His interests were not confined to the domain of medicine: he was widely read in the humanities, and he wrote extensively on areas as diverse as classical mythology, thanatology, and history.[8,9]

Much less well remembered, although probably of even greater value, is his pioneering work in the field of neurosurgery. He was in the vanguard of the neurosurgical world of his day, and in many respects his work opened the way for later developments by Harvey Cushing and others. A review of Park's neurosurgical work not only records his contributions, but also provides captivating insights into the prevailing concepts of brain function at the turn of the century. In those years, the medical community was regularly supplied with revolutionary neurosurgical news. In 1884, the first successful removal of a brain tumor was accomplished in Great Britain. (The tumor

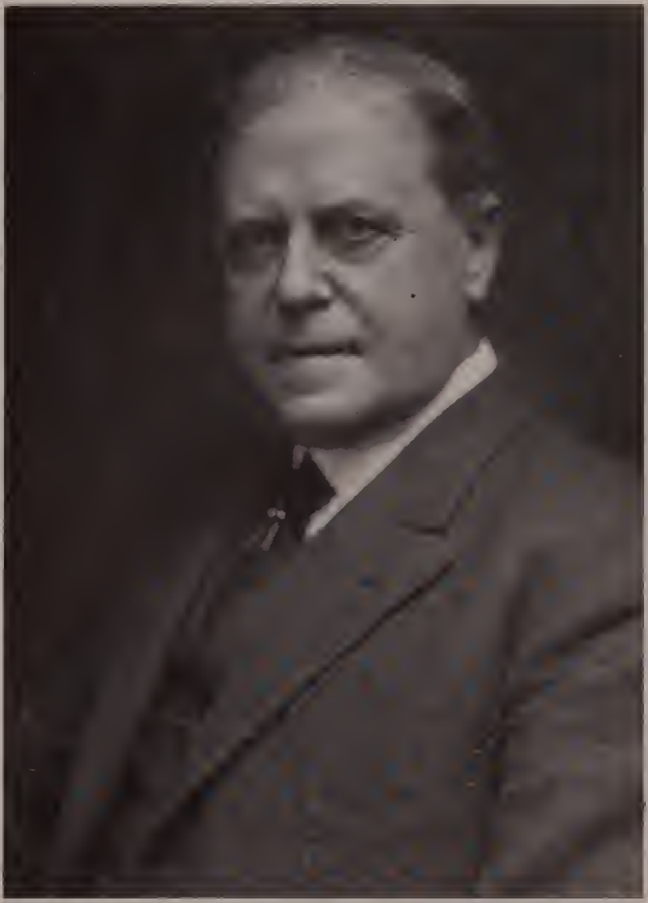

FIGURE 1. Roswell Park, one of the most prominent figures in American surgery, came to Buffalo at the age of 31 to become professor of surgery (photograph courtesy of the Medical Photography Department, Roswell Park Memorial Institute, Buffalo, NY).

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was diagnosed and localized by A. H. Bennett, who persuaded R. J. Godlee to operate.[10] In 1887, Gowers diagnosed a spinal tumor; Victor Horsley was invited to operate, and a "fibromyxoma" was removed in the theatre at Queen Square.[11] The patient made an uneventful recovery.

Anatomic localization is the cornerstone of clinical neurology. The modern era of cerebral localization began in the late 19th century, when surgeons became confident enough to intervene in the brain with the topographic guidance of clinical neurology. A mind as encyclopedic as Park's could not remain isolated from these innovations. He wrote on the topic of neurosurgical techniques and became very interested in the area of cerebral topographic anatomy for surgical landmarking.[12]

A crucial milestone for American neurosurgery took place in Washington, DC, in 1888, on the occasion of the First Congress of American Physicians and Surgeons. One session was devoted to a symposium on brain surgery. Sir Victor Horsley attended the meeting, as did Park.[13] Present at this congress were many other influential scientists, including Sir David Ferrier, the brilliant British neurophysiologist, who had demonstrated with his famous cerebral ablation experiments in monkeys some of the basic principles of cerebral localization. The participants brought to debate the rapidly accumulating discoveries in cerebral localization of the time. Park declared the date as one that was ushering in the dawn of a new era.[13] His hopes with regard to the possibilities of brain surgery were stirred by the expectations that "in a short time the topography of the brain would be charted out for the surgeon as are obstructions in the navigation charted out for the marine pilot"[13] (Fig 2).

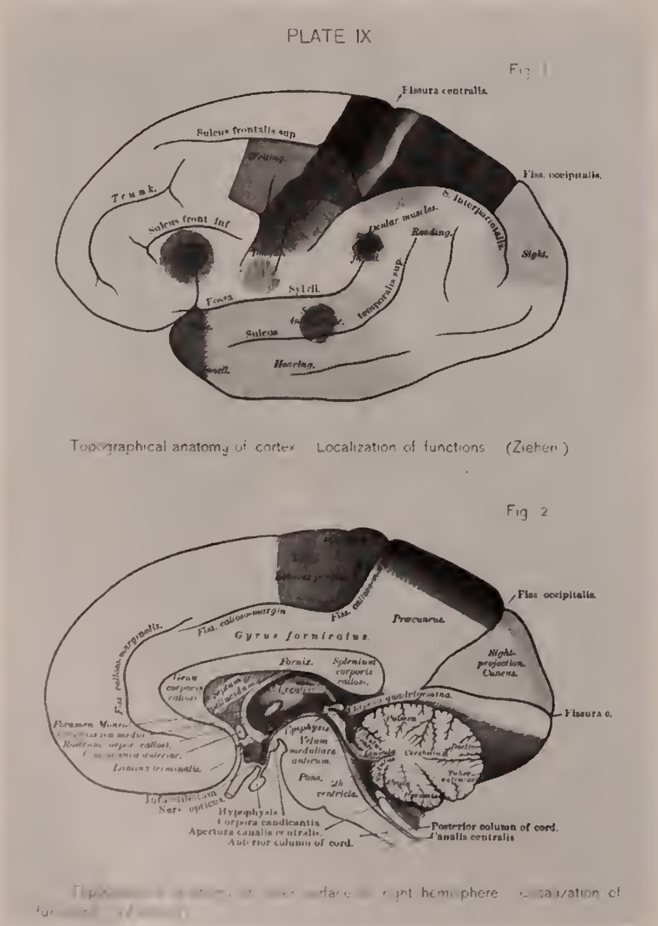

FIGURE 2. Park's concept of cerebral localization from his book, *Treatise on Surgery by American Authors*,[28] 1901 (photograph courtesy of the Medical Photography Department, Roswell Park Memorial Institute, Buffalo, NY).

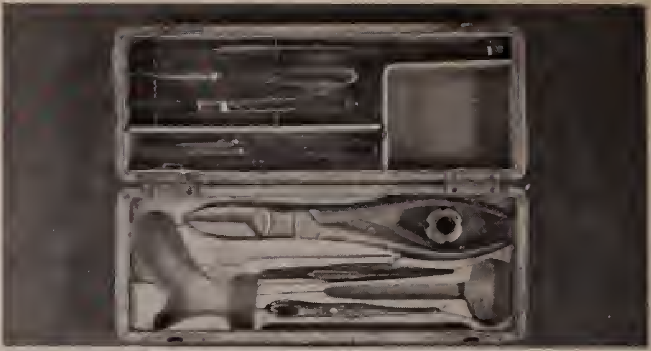

FIGURE 3. Some of the surgical instruments used by Park, on display at the Roswell Park Memorial Institute (photograph courtesy of the Medical Photography Department, Roswell Park Memorial Institute, Buffalo, NY).

In this day and age, when there is renewed interest in the surgical treatment of epilepsy, we are seldom aware of the enormous debt owed to the medical scientists of the late 19th century. By modern standards, these early attempts to treat epilepsy may appear to be crudely performed experimentation, but they certainly opened the way to later developments. The early neurosurgical work on epilepsy was instrumental in the formulation of the postulate that the cerebral hemispheres were not equipotential, proving beyond doubt some of the cardinal principles of cerebral localization.

Park was among the first to treat epilepsy with a direct approach on the brain and its membranes by trephination, and he insisted on the need for opening the dura (a sancta sanctorum shunned by most surgeons of his time) to survey the brain.[14] He was always proud of his pioneering neurosurgical work. In fact, Dr Park operated on the first patient in the US whose mass was accurately localized and successfully operated on based on the then new principles of cerebral localization. The patient's meningeal cyst was removed by Dr Park at the Buffalo General Hospital in 1886[13] (Figs 3, 4).

These attempts were certainly not devoid of flaws and misunderstandings. At the turn of the century, the vascular theory of the etiology of epilepsy was the prevailing

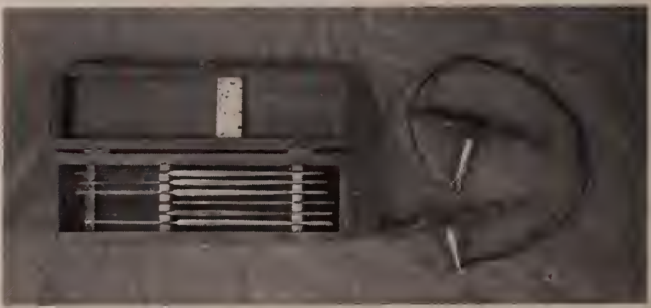

FIGURE 4. A valuable addition in the surgeon's outfit was the Gigli saw (right), which simplified craniotomies greatly (photograph courtesy of the Medical Photography Department, Roswell Park Memorial Institute, Buffalo, NY).

one. Park, among others, ventured to treat certain forms of epilepsy by increasing cerebral blood flow with cervical sympathectomies.[15,16]

Park was a physician of great moral fiber and dedication. His last years were punctuated by adversity.[6] He contracted diphtheria while intubating a child dying of the disease, and later suffered from a series of protracted illnesses. He developed a severe laryngeal stenosis for which he underwent numerous operations, enduring great pain until his death in 1914.[6]

SURGICAL TREATMENT OF EPILEPSY

Trephinations have been performed since prehistoric times for the treatment of epilepsy, to permit evil spirits to escape, and for many other reasons. As postmortem examinations became common practice in the 19th century, brain tumors, depressed fractures, and many other pathologic findings were implicated as etiologic factors of epilepsy. The new emphasis on anatomic abnormalities prompted many surgeons to perform trephinations to treat epilepsy. Most of the trephinations carried out at the turn of the century were based on the belief that pressure exerted by the cerebrospinal fluid on the brain was a common cause of seizures. Several techniques were devised to prevent fluctuations in the pressure of the cerebrospinal fluid. Kocher[17] described a "movable valve" to drain the fluid collections sometimes seen over the cerebral hemispheres in epileptics. Alexander,[18] in 1911, started performing "fenestrations of the dura mater" by making a number of parallel slits in the dura, with a glass tube being used for temporary drainage. It was not until 1917 that Dandy[19] finally proved that such collections of fluid were merely a sign of cerebral atrophy and not a cause of epilepsy.

In 1892, Sir Victor Horsley,[20] using his theory of the "primary spasmodic center," showed that in focal epilepsy there is a point in the cerebral cortex that gives rise to the seizure. The excision of this area was often followed by cessation of the attacks.

The study of epilepsy proved very fruitful in the localization of brain functions.[21] Jackson's[22] intensive study of patients with focal seizures led the way to mapping the motor cortex, as it was discovered that areas governing specific body movements existed in the brain. Further localization of distinct functions was done in the two decades between 1860 and 1880 by Broca and Wernicke. After their discoveries, the principle of cerebral homogeneity was no longer tenable.[21,23]

Until the later part of the 19th century, brain surgery was limited to physical injuries, in which the site of the lesion was obvious by the location of the lacerations or bruises over the scalp. The knowledge of cerebral localization opened a new chapter in the history of neurosurgery, as topical diagnosis based on clinical examination showed where to operate.

Some of the surgeons of the time operated under the direction of a clinician who made the diagnosis of localization. As mentioned, the first successful removal of a intraspinal tumor, performed by Victor Horsley, had been accomplished after the localization was accurately outlined by Gowers.[11] Park was a keen diagnostician himself. He was informed about the subtleties of diagnosis of localization to a degree that was reached by only a handful of surgeons of his era. For instance, in expressing his views on cerebellar abscesses, he wrote the following as early as 1888: "Certain peculiar features are alleged to pertain to abscess in the temporal region; thus, Wernicke has stated that there is a peculiar disturbance of speech which points especially to this lobe as the locality of the disturbance. This is the confusion of correct with incorrect or fictitious (erdichteten) words."[12]

Park enthusiastically tried the new developments about which he was knowledgeable. His fruitful work in the surgical treatment of epilepsy was promoted by a longstanding association with William P. Spratling, the medical superintendent of the Craig Colony in Sonyea, NY, which was one of the world's first facilities dedicated to the care of epileptics.[24] The Craig Colony was a model institution with a large community of epileptics, which gave Park ample opportunity to try some of the new surgical methods of treatment. Harvey Cushing,[25] in his chapter on surgery of the head, wrote about the Craig Colony: "For the general education and regulation of life of the incurable and idiopathic forms of epilepsy in general, the colony system, such as that established at Sonyea, New York, possesses many obvious advantages." Spratling[15,16] was a prolific writer on epilepsy and coauthored several papers with Park, reporting their ideas and results.

Park became very involved in the study of cerebral surgical anatomy. In 1888, in an article entitled, "Surgery of the brain based upon the principles of cerebral localization," he wrote with his characteristically colorful style: "Not much less astonishing than the discovery of the planet Neptune at the spot determined by the computations of Le Verrier was the first discovery of a cerebral lesion at exactly the point indicated by a careful study of somatic disturbances; both were wonderful examples of inductive reasoning."[12] In this article, he enumerated a wealth of information that could still guide modern neurosurgeons. He observed that the Torcular Herophili is seldom exactly centrally located, most commonly being a little to the left. Other facts that need to be borne in mind are that children and the aged have no diploe; that the crania of the aged may be extremely thin; that the frontal sinus is not to be disregarded when operating in its neighborhood; that the superior longitudinal sinus is beneath the middle line of the vault; and that the middle meningeal artery lies about an inch and a quarter in back of the external angle of the orbit, and is sometimes almost buried in a bony channel.

To trephine the skull safely, he advised that the first application of the trephine should be over those well-known areas of the skull which do not overlie large vascular channels. He was particularly wary with regard to opening one of the venous sinuses of the skull for fear of two dangers; one, of fatal air embolism; the other, of profuse hemorrhage. He also disagreed with Schellmann in his opinion that the integrity of a sinus may be destroyed with impunity: "One must perhaps hold to the opinion that the liability of edema of the brain will thereby be increased."[12]

At the time, some surgeons were performing trephinations for the treatment of epilepsy in a ritualistic way: a piece of bone in the calvarium was removed and no attempt was made to open the dura mater. The rationale for such interventions was rather hazy. Park became an impassioned proponent of opening the dura to survey the underlying brain.[26,12] His endeavors were rewarded by several claimed remarkable cures. Considering the abysmal mortality rate from brain surgery at the time, these notable results are a tribute to Park's dexterity.

Some of Park's case histories are noteworthy. For example, a soldier was injured in the charge of San Juan Hill in the Spanish-American war. A bullet penetrated his skull and injured the brain. Three years later he presented to Park with spastic hemiplegia and seizures that began in the thumb. Park performed a craniectomy over the motor areas and excised the hand and arm centers; the seizures almost disappeared.[14] In another case, Park operated for Jacksonian epilepsy of the left arm. On opening the dura a "fibroma" (meningioma?), the size of a hickory nut, showed itself. The tumor was removed, and the patient made an excellent recovery.[27]

Park was painfully aware of the limitations of surgical methods. In 1892 he wrote, "When operating for pronounced epilepsy we have to combat not only a somatic lesion, but an epileptic habit, so to speak, and a mere removal of the lesion is not necessarily or always enough to break up the well formed habit."[26] Park made a careful selection of his patients and stated that epilepsy is the last disease in which surgical measures should be indiscriminately applied.[14]

The classification of seizures was in an embryonic stage at that time, but Park undertook the task of delineating the surgical indications. He summed up his observations as follows: "We first single out the epilepsies that mostly affect the motor side of the body as promising most for surgical treatment, to the exclusion of those that invade the psychic side to the greatest degree."[14] Park's associate, Spratling, stated in 1903 that the operations for the possible relief of epilepsy should be confined to cases in which the attacks are grand-mal or Jacksonian, and will seldom be found of any use in petit-mal or psychic types.[15]

Park clearly understood the significance of an aura as pointing to a focal, localized pathologic process amenable to surgery. In his *Treatise on Surgery by American Authors*,[28(p 693)] Park wrote (in his chapter on injuries and surgical diseases of the head),

> A certain proportion of the cases, especially those of traumatic origin, are characterized by a localized and definite aura and by a systematic and practically invariable order of muscle involvement according, it would seem, to some fixed law, and pointing definitely to a certain area of the brain from which apparently the irritation arises and spreads. This form of epileptic seizure is that generally known as the Jacksonian, and is that in which operation is most often of real service [Fig 5].

To quell any overenthusiastic intervention in epilepsy, the always cautious Park gave this sensible caveat: "The temptation is always to remove scars, and the tendency to forget that every scar is followed by another. Here we are forced to limit our effort to the endeavor to make the second scar less irritating or disquieting than the first."[13]

TREATMENT OF EPILEPSY WITH CERVICAL SYMPATHECTOMIES

The intricate history of the surgical treatment of epilepsy is certainly not free from many false steps. Vascular procedures were commonly practiced at Park's time for the treatment of epilepsy. Episodic disruption of the cerebral blood circulation was considered to be related etiologically to convulsions. Based on this conjecture, Alexander[29] began in 1883 to treat epilepsy by cervical sympathectomy.

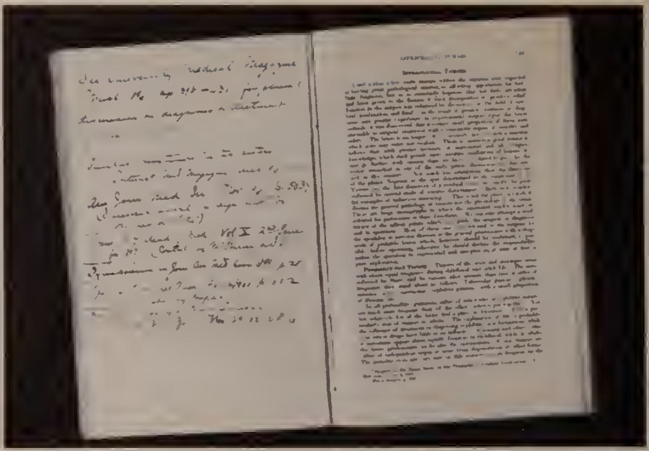

FIGURE 5. A copy of *Treatise on Surgery by American Authors*,[28] 1901, with handwritten notes by Park (photograph courtesy of the Medical Photography Department, Roswell Park Memorial Institute, Buffalo, NY).

Park, though aware of the differences of opinion between authors concerning the blood supply of the brain during epileptic attacks, became a proponent of the vascular theory and performed numerous sympathectomies. His experiences were written in a series of articles published at the beginning of this century.[14-16]

However erroneous these early procedures may appear in light of the modern understanding of epilepsy, they were undoubtedly based on empirical observations of that era. Brown-Séquard[30] considered that the facial pallor seen in convulsions was a manifestation of a more generalized cephalic vasoconstriction affecting the brain. Even Jackson,[31] the leading author who radically changed existing views on the etiology of epilepsy, wrote in 1864 that the theory of constriction of the blood vessels in epilepsy was the established theory of the day. Physiologists had produced convulsions experimentally in animals by a sudden and simultaneous ligation of both carotids. A surgeon, Doyen, claimed to have witnessed the occurrence of a seizure during a neurosurgical procedure and to have noted at that moment a pronounced vasoconstriction in the brain. There was some dissent in this controversy: Victor Horsley,[20] in 1892, wrote that vascular changes were not an etiologic factor of epileptic spells but a mere epiphenomenon of the attack.

As a follower of the vascular theory, Park devised a way to detect the patients likely to benefit from a sympathectomy: "In those cases characterized by blanching of the face, when the seizures can be warded off or mitigated by the prompt use of amyl nitrate, we may well consider the propriety of an exsection of the cervical sympathetic."[14]

In summary, the surgical treatment of epilepsy was made possible at the turn of the century by the then rapidly accumulating knowledge of anatomic localization of brain functions. Roswell Park was in the vanguard of neurosurgery in his time. In many ways, his pioneering work opened the way for Harvey Cushing and others. He was among the first to treat epilepsy through a direct approach on the brain and its membranes by trephining. A review of Park's work gives insights into the fascinating history of the localization of brain functions.

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CONCLUSIONS DRAWN FROM A QUARTER CENTURY'S WORK IN BRAIN SURGERY

EPILEPSY (INCLUDING THE OTHER LEPSIES). In but few studies of this kind are statistics of less value than here. Personal experiences are always of interest and should be of importance, but those of different surgeons differ so widely that any advice given must be based upon almost anything rather than mere statistics. Have large gains been made in the surgical treatment of epilepsy? In reply I would say that such gains have been relatively small, and that we know little more about the prime factors of this syndrome than we did thirty years ago, and that operative technique has made but small advance. Operations indiscriminately performed must always be unsatisfactory, and this discrimination has been too seldom evinced in this direction. True we have much with which to contend, including especially vagueness of history, uncertainty of observation, and the fact that a period of years is often allowed to elapse between injury or even the first symptoms of the condition and the attempt at operative relief. During these years cicatricial and other changes have taken place, and the epileptic habit has become formed, while dementia or even mania have so altered the patient's finer brain characteristics as to ruin almost every prospect of the success that might have been attained had the interval been much shorter. Disappointment, then, so frequently results that operations for epilepsy are now quite frequently discouraged, since they have brought surgery into a sort of ill-repute which it does not deserve. The trouble is not so much that operation itself is a mistake, since it is a perfectly justifiable late or even last resort, but patients are led consciously or unconsciously to expect too much. So soon as the term "experiment" or "legitimate experiment" is used in this connection a large proportion of people jump at the conclusion that we mean something by the expression which it is really not intended to convey. Personally I believe in operating in many instances where the outlook is even unpromising, simply because this outlook is still more unpromising without it. In the strict meaning of the term such intervention *is* an experiment, justifiable of course, but an experiment in the sense that a voyage of discovery is such. If intelligently accepted in this significance no blame attaches even to a fatal result; in fact this may often be in effect a blessing in disguise. . . .

ROSWELL PARK, MD, LLD
(*NY State J Med* 1913; 13:303-309)

CASE REPORTS

Omphalocele: Sonographic detection at 15 weeks' gestation

KENNETH R. ROMANO, MD; ORLANDO L. MANFREDI, MD; TIMOTHY FARLEY, MD;
JOSE E. PEREZ-GUMA, MD

An omphalocele is a developmental defect in the anterior abdominal wall of the fetus which occurs in approximately one in 2,500 births. One cannot discuss this congenital deformity without including another rare anomaly of the anterior abdominal wall—gastroschisis. An omphalocele is a herniation of the abdominal contents into the base of the umbilical cord, whereas gastroschisis is a full thickness anterior abdominal wall defect that is located lateral to and usually to the right of the umbilicus. Prenatal sonographic diagnosis of omphalocele and gastroschisis is of paramount importance for definitive obstetric and perinatal management.

CASE REPORT

A 29-year-old woman was referred for obstetric sonography at the 15th week of gestation. Sonography was performed with a commercially available linear array real-time system with a 3.5-mHz transducer. A single fetus in migratory positions was identified with an estimated biparietal diameter of 3.12 cm and a recorded femur length of 2.16 cm. The placenta was fundocorporeal with extension along the posterior wall. A circular anechoic image was noted along the anterior aspect of the pelvis, representing the urinary bladder; however, a persistent, dominant, 3 × 2.2 cm elliptical, sonolucent image with a thin echogenic periphery was noted extending anteriorly from the lower abdominal wall, with an umbilical cord present in the region of the apex of the defect (Fig 1). The fetal heart was recorded at a normal rate. The choroid plexuses were well outlined, and there was no evidence of ventricular dilation or displacement. The initial impression was consistent with a lower anterior abdominal wall defect secondary to an omphalocele. The patient submitted to voluntary interruption of pregnancy.

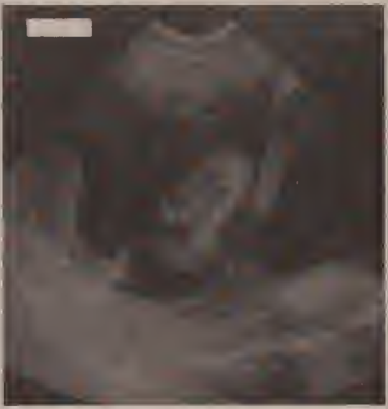

FIGURE 1. Real-time sonogram showing a true omphalocele. A sagittal section shows a fetus of 15 weeks' gestation with a 3 × 2.2 cm elliptical, anechoic image with a thin echogenic periphery extending anteriorly from the lower anterior abdominal wall. Insertion of umbilical cord into the sac is demonstrated.

The specimen was a female fetus weighing 260 grams and measuring 23 cm in crown–heel and 15 cm in crown–rump length. Multiple external anomalies were reported. The abdomen was scaphoid. The most proximal 5 cm of the umbilical cord were widened out into a sac-like structure which appeared to contain loops of bowel, no other organs, and over which the umbilical vessels splayed. The base of the defect measured about 2.6 × 2.0 cm. The defect was slightly larger in the superior inferior direction. The genitalia were those of an immature female fetus. On opening the anterior abdominal sac it was evident that it did not contain intestinal tract, but the urinary bladder was protruding approximately 2 cm into the sac. The fetal abdominal cavity contained intestines which were malrotated. The liver and spleen were unremarkable. The uterus was normal, although the ovaries were unusually wide and extended to the flanks on both sides at the level of the pelvic rim. The urinary bladder was unusually large.

Numerous cardiac abnormalities were noted involving the tricuspid valve, intraventricular septum, and pulmonary artery.

The kidneys were normal in size, shape, and weight. The urinary bladder was markedly dilated, and its tip, which might have been urachal, extended into the umbilical hernia. Two umbilical arteries flanked the urinary bladder. The umbilical hernia had a sharp rim and was approximately 2 cm long and 1.5 cm wide. It contained only the tortuous umbilical vessels and the dome of the bladder. The sac measured approximately 5 cm and was filled with Wharton's jelly.

Chromosomal investigation disclosed a trisomy 18.

DISCUSSION

In normal development, the mid-gut and superior mesenteric artery protrude out of the abdomen into the umbilical cord forming a physiologic umbilical hernia at approximately the fifth fetal week. In the ensuing six weeks, the gut rotates, and at the 11th week the gut returns into the abdomen, having completed a full 270° rotation.[1] If the mid-gut fails to return, an omphalocele is formed. It may consist of only herniated mid-gut, although other viscera can be included, covered by a translucent membrane. This membrane consists of peritoneum inside and amnion outside separated by Wharton's jelly. The umbilical cord is attached to the apex of the mass.[1] The incidence of omphalocele has been reported as one in 3,000 to one in 10,000 births.[2]

Gastroschisis is a defect that occurs in the embryonic fold just lateral to its apex, which results in a full thickness abnormality in the lateral wall adjacent to the umbilicus. The abdominal contents can then protrude through this defect.[1,2] The hernia is devoid of an amniotic layer or Wharton's jelly, and the umbilical cord lies separate in its normal position. During fetal life, the herniated contents, being bathed in amniotic fluid, undergo a form of sterile

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chemical peritonitis and can present as a clumped mass held together by adhesions.[1] The incidence of gastroschisis has been reported to be approximately one third the incidence of omphalocele.[3]

The purpose of this paper is to describe a case in which antenatal sonographic diagnosis was achieved at 15 weeks' gestation. Recognition of ventral abdominal wall defects is important for parental counseling, method of delivery, and management of pregnancy.

Obstetric sonography plays an important role in prenatal diagnosis and subsequent management of omphalocele. Antenatal recognition of abdominal wall defects assists the obstetrician and patient in the plan of delivery. Meticulous real-time sonography can detect these abnormalities as early as 15 weeks' gestation. Armed with this information, the parents can be fully informed and prepared for the consequences.

Serial examination may be obtained with special attention directed toward the size of the abdominal defect and associated congenital anomalies. Ultrasound-guided amniocentesis reveals useful information regarding the titer of alpha-fetoprotein and karyotypes. Omphaloceles may be associated with other anomalies such as intestinal atresia or stenosis as well as Meckel diverticulum. Chromosomal aberrations may be related to genitourinary and cardiac abnormalities.[4]

Gastroschisis is rarely associated with anomalies other than Meckel diverticulum. Therefore, fetuses with gastroschisis have a better prognosis than those with omphalocele.[5,6] Sonographic differentiation of omphalocele from gastroschisis is based on the presence of a covering membrane, the site of cord insertion, and the type of viscera contained in the defect. In omphalocele a membrane is present, the cord inserts into the membrane, and the liver is commonly herniated. In gastroschisis, a covering membrane is absent, the cord inserts into the abdominal wall, and thickened bowel is commonly contained in the defect. Additionally, fetal ascites is frequently present in omphalocele and not in gastroschisis.[7]

Acknowledgment. The authors thank Mrs Julie Werner for her assistance in preparing this report.

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See also pp 569-570.

Immunoglobulin therapy in a patient with chronic lymphocytic leukemia and recurrent infections

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Several investigators have demonstrated the beneficial effects of immunoglobulin in reducing bacterial infection in patients with hypogammaglobulinemia.[1-5] Patients with chronic lymphocytic leukemia (CLL) commonly have hypogammaglobulinemia, which may be severe. The major consequences of this acquired deficiency of immunoglobulin are increased susceptibility to bacterial infection and a subsequent increase in morbidity and mortality. This report details a successful experience with weekly intravenous immunoglobulin replacement therapy in a patient with CLL complicated by frequent severe infections.

CASE REPORT

A 65-year-old man had CLL, clinical stage II, which had been diagnosed in 1963. Between that year and 1972 he was treated intermittently with cyclophosphamide, vincristine, and prednisone for lymphadenopathy, and between 1974 and 1977 he received vincristine and methotrexate for the same problem. In 1979, pancytopenia developed, which was corrected by splenectomy. During the same year, the patient experienced two episodes of severe pneumonia, in one of which both *Haemophilus influenzae* and *Bacillus fragilis* were present. Bronchopulmonary infections occurred repeatedly over the next three years, and in 1983 the patient experienced a near-fatal viral pneumonia and meningitis. Recurrent pneumonia continued to dominate his clinical course, and bronchograms in 1984 showed bronchiectasis. Bronchial biopsy revealed lymphocytic infiltration and chronic inflammation.

In 1985, rectal fistulas developed and were associated with multiple bouts of diarrhea and abdominal sepsis. Repeated hospitalization for intravenous antimicrobial therapy became necessary, and during short home stays, the patient took multiple oral antibiotics. Clinical reevaluation revealed significant generalized adenopathy and mild anemia.

The white blood cell count was 20,000/mm^3 with a predominance of small lymphocytes. Serum IgM was undetectable, IgA was 20 mg, and the IgG level was 200 mg. Lymphocyte typing disclosed a marked decrease in CLL cells from a previous count and a reversal of the OKT4/OKT8 ratio.

Following an episode of an acute abdomen in May 1986, the patient was thought to have abdominal abscesses. Considered to be too fragile for surgery, the patient was started on a course of intravenous antibiotics and immunoglobulin (Sandoglobulin,

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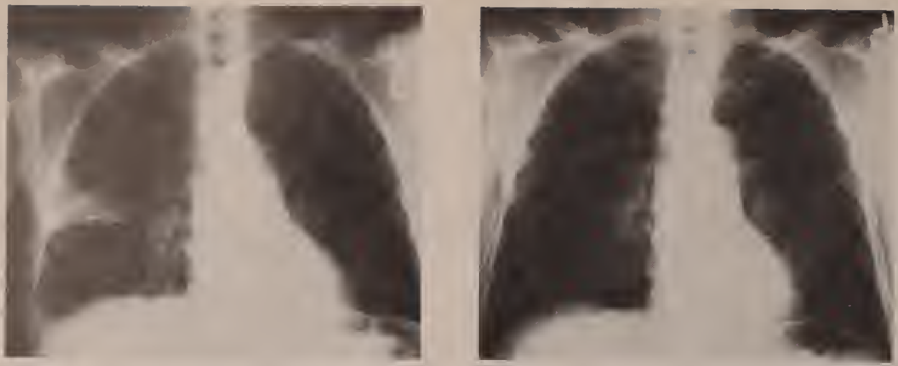

FIGURE 1. **Left,** radiograph of chest prior to intravenous immunoglobulin; **Right,** radiograph of chest after intravenous immunoglobulin.

Sandoz Pharmaceuticals). The antibiotics were discontinued after three weeks, and intravenous immunoglobulin was continued alone. Initially, to treat the infection, the patient received 0.4 g/kg of immunoglobulin every three days; however, this dose was gradually reduced to 0.2–0.4 g/kg weekly.

Marked clinical improvement occurred on this therapy. Especially noteworthy was the complete resolution of radiographic changes of chronic pulmonary inflammatory disease (Fig 1). Once stabilized, the patient was subjected to an exploratory laparotomy. During surgery, a thinly encapsulated abdominal abscess was excised, and a fistulous tract with an involved segment of colon was removed. Several smaller tracts in the rectum had already closed in response to treatment with gamma globulin and antibiotics. Microscopic examination of the excised colon revealed infiltration with small, well-differentiated lymphocytes. Because of the patient's chronic incapacitation resulting from recurrent severe infection, he has been continued on intravenous immunoglobulin therapy as prophylaxis against infection. His maintenance dose of intravenous immunoglobulin is about 0.4 g/kg/month.

Since starting the intravenous immunoglobulin infusions, the patient has had no infections, even in the absence of antibiotic prophylaxis. Lymphocyte typing now shows a normal OKT4/OKT8 ratio, while the total white cell count, including CLL cells, remains depressed. Lymphadenopathy has markedly regressed. Immunoglobulin levels have stabilized, with an IgG level of approximately 1,500 mg%; however, IgA and IgM levels remain subnormal (Fig 2).

At present, the patient returns to the hospital once weekly for his immunoglobulin infusions. The only adverse effect has been an allergic rash which developed six months after the start of treatment and which responded to single doses of benadryl, cimetidine, and prednisone administered before and after each immunoglobulin infusion.

DISCUSSION

Replacement therapy with immunoglobulins for patients with hypogammaglobulinemia is effective in reducing infections. The major mode of administration has been the intramuscular route, and the traditional dose recommended is 100 mg/kg per month. One of the restrictions imposed by intramuscular administration is the small dose that can be given, which results in insufficiently high levels of circulating antibody. One of the major benefits of the new intravenous immunoglobulin is the opportunity to use higher doses sufficient to maintain immunoglobulin levels within the normal range.

Recurrent infections, often associated with very low levels of serum immunoglobulins, are common in B-cell CLL. The recent availability of intravenous immunoglobulin preparations that are effective and well tolerated in patients with hypogammaglobulinemia allowed us to assess the role of exogenously administered immunoglobulin in this patient with CLL, hypogammaglobulinemia, and recurrent severe bacterial infections.

The patient's clinical response to such therapy has been dramatic. Not only has he remained free of infection, but he is enjoying a sustained partial remission of CLL as evidenced by regression of his lymphadenopathy and reduction of his leukocyte count with a pronounced decrease in CLL cells. These changes have occurred during a period when only immunotherapy was being administered. Although the consequences of immunotherapy on tumor burden are poorly understood and have been quite variable, the administration of immunoglobulin in this patient with CLL has been associated with a favorable effect on tumor mass. A similar effect on tumor mass has been reported by Besa.[6] Our experience in this case parallels the observations of others. Schedel,[7] in a prospective randomized study, administered intravenous immunoglobulin (12 grams at intervals of 18–26 days) to ten patients with CLL, five of whom were displaying symptoms of chronic bronchitis. The mean serum concentrations before treatment were 5.16 g/L ± 2.85 g/L. With this treatment, four of the five patients showed improvement of their symptoms.

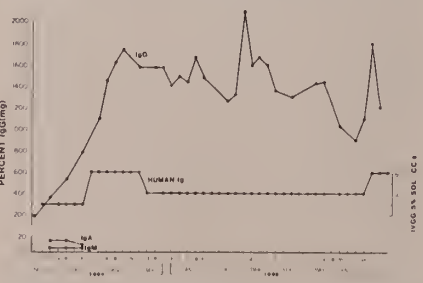

FIGURE 2. Normalization of serum gamma globulin levels (% mg) in response to the intravenous administration of immunoglobulin.

Besa[6] noted control of infections in two of three patients with clinical stage III or IV CLL and subnormal immunoglobulin levels whom he treated with intravenous IgG for recurrent infections. The dose used for prophylaxis and control of recurrent infections was 0.15 g/kg for two doses 14 days apart, and then maintenance with 0.3 g/kg every three to four weeks.

The administration of immunoglobulin to replace endogenous IgG is currently the treatment of choice for individuals with primary antibody deficiency syndromes. Benefit in patients with primary hypogammaglobulinemia and sinopulmonary infections has been demonstrated.[8] However, the published literature contains little information documenting a role for intravenous immunoglobulin in the management of patients with B-cell CLL acquired hypogammaglobulinemia and recurrent serious or life-threatening infections. Our experience with this immunocompromised patient with CLL and severe recurrent bacterial infections suggests that immunotherapy may be an effective means of reducing or preventing infection in such patients.

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See also pp 572–573.

Extreme rhabdomyolysis in a patient with acute leukemia

Association with *Candida kruseii* fungemia

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Rhabdomyolysis represents a constellation of clinical and laboratory findings associated with striated muscle injury and release of intracellular muscle contents into plasma. Subsequent myoglobinuria has been associated with acute renal failure. Excessive exercise, trauma, alcohol, and drug use account for the majority of cases of rhabdomyolysis reported among western nations. Less common etiologies include immune and metabolic causes, along with toxins, infections, and rare genetic disorders of muscle metabolism.[1,2] This report describes the coincident finding of rhabdomyolysis and disseminated candidal infection in a patient with acute leukemia in whom the peak creatine phosphokinase (CPK) level reached 165,000 IU/L.

CASE REPORT

A 29-year-old woman was found to have acute myelogenous leukemia (AML) while pregnant with her second son. After the induction of a successful remission, she developed *Mycoplasma gordonae* pneumonia diagnosed by open lung biopsy, and was treated for 18 months with isoniazid and rifampin. The patient was admitted to Strong Memorial Hospital (Rochester, NY) for treatment of the third relapse of her AML. A partial remission was achieved with high doses of amascarine, cyclophosphamide, and cytosine arabinoside.

Because of persistent petechiae, refractory thrombocytopenia (platelet counts 1,000–4,000/μL), and intermittent episodes of vaginal bleeding, therapy with epsilon-amino caproic acid was begun. Two weeks later, while the patient was granulocytopenic, receiving epsilon-amino caproic acid, isoniazid, rifampin, clindamycin, and ceftazidine, and after she had received 700 mg of prophylactic amphotericin B, a new rash developed. Multiple erythematous papules, measuring 3–4 mm with surrounding macular erythema, were noted on the left forearm. A punch biopsy showed deep dermal intravascular yeast forms with perivascular inflammation (Fig 1). Blood cultures, on two separate occasions, subsequently grew *Candida kruseii*.

The Hickman central line was removed, and 5-flucytosine was added to the amphotericin B. Additional potassium supplementation was required for a serum level of 2.8 mEq/L. Granulocyte transfusions, given daily for eight days, were begun, and three days later the patient complained of myalgias of both thighs and the right deltoid. Her urine was noted to be heme positive with 0–1 red blood cells per high power field. The skin lesions extended to more than 20 circumscribed areas involving both arms, hands, thighs, forehead, back, and legs. An echocardiogram revealed no valvular vegetations. Urine myoglobin was detected, and an initial CPK level was 7,606 IU/L (normal, 0–148 IU/L) with less than 4% MB forms. During one day, the CPK rose to 22,144 IU/L, and therapy with epsilon-amino caproic acid was stopped.

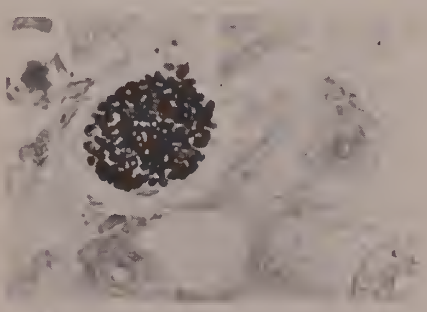

FIGURE 1. Punch biopsy showing deep dermal intravascular yeast forms with perivascular inflammation (Gomon's methenamine silver).

At this time, the amphotericin B minimum inhibitory concentrations (MIC) were therapeutic against the *Candida kruseii*, and the white blood cell count (WBC) rose to 1,500/μL with 77% neutrophils. Granulocyte transfusions were halted, but the patient continued to have spiking fevers, with temperatures to 39–40°C. Cultures of blood, urine, and sputum were negative. Amikacin, ceftazidine, and clindamycin were stopped, but muscle pain continued and the CPK rose to 100,000 IU/L. The patient underwent plasmapheresis twice daily for three days because of persistent symptoms, elevated CPK, and a low CH_{50}. The serum creatinine level increased to 2.6 mg/dL, which reversed with aggressive hydration. The CPK peaked at 165,352 IU/L ten days after the onset of symptoms. Mild hypercalcemia and hyperphosphatemia were reversed, and, 37 days after the onset of the rhabdomyolysis, the CPK level had fallen to 278 IU/L. At that time the patient was beginning rehabilitation and aggressive physical therapy after achieving a partial remission from her relapsed AML. Her serum potassium level at discharge was 4.1 mEq/L.

DISCUSSION

The possible causes of rhabdomyolysis in this patient are numerous and probably multifactorial. In one review of this disorder, 51 of 77 patients had multiple factors as the cause of the rhabdomyolysis. Alcohol use, muscle compression, and generalized seizures made up the bulk of the risk factors.[1] There were as many as five possible causes for the rhabdomyolysis in this patient.

The use of epsilon-amino caproic acid, a specific inhibitor of plasminogen activation, has been associated with muscle injury in doses of 24–32 g/day,

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after four to eight weeks of continuous therapy.[3] Its mechanism of injury remains unclear, but the absence of vascular involvement in biopsied muscle suggests a direct myotoxicity.[4]

Hypokalemia is an important predisposing factor in muscle injury, usually at levels below 2.3 mEq/L.[5] Suppressed potassium-mediated vasodilation in muscle or destabilization of myocyte membranes can explain this phenomenon.[6] Amphotericin B-induced rhabdomyolysis has been reported in a patient with a serum potassium level of between 1.5 mEq/L and 2.2 mEq/L.[7]

Immune-mediated muscle injury has also been reported as a cause of polymyositis.[8] The patient suffered renal failure, presumed to be due to a myoglobin-mediated decreased glomerular filtration rate and altered intrarenal blood flow. A biopsy showed mesangial widening and increased mesangial cells in the renal glomerulus.

Infections with bacteria and viruses have been associated with rhabdomyolysis.[9-11] A patient with CPK levels of 216,000 IU/L was reported with a *Staphylococcus aureus* abscess and muscle necrosis, illustrating the potential severity of such a presentation.[11]

The patient presented in this report is unusual in that she experienced extremely high levels of serum CPK lasting more than 36 days despite optimal therapy. The persistent severe thrombocytopenia made direct muscle biopsy undesirable. The prolonged pattern of muscle injury is not typical for acute drug-related injury. Epsilon-amino caproic acid myotoxicity typically develops after more than four weeks of therapy; this patient received only two and a half weeks of therapy. The serum potassium level never fell below 2.8 mEq/L, and no case of amphotericin-mediated muscle injury has been reported with only moderate potassium depletion. The serum phosphorus level was within the normal range prior to the onset of the rhabdomyolysis. Although plasmapheresis was performed on clinical grounds, the evidence for immune-mediated injury cannot be documented. The isolated decrease in serum complement is a nonspecific finding, compatible with severe muscle injury, systemic candidal infection, and thrombocytopenia.

While the exact causative factor in this case is impossible to determine, the severity and prolonged nature of the muscle injury in a patient with evolving return of circulating leukocytes raises the possibility of an infectious source. The presence of fungemia with direct or indirect myotoxicity and myoglobinuria suggests a cause that must be seriously considered. Detection of candida by skin biopsy has been correlated with direct muscle invasion in a patient with myalgias and disseminated candidemia.[12] Certainly this patient's chronology of elevation in white blood cell count was correlated with worsening myalgias and rash, as well as peak CPK determinations. The hypothesis of extensive muscle inflammation and necrosis after fungemia and slow return of immune function cannot be the only one considered here, but it remains an important association and could be the causative agent in this patient with extreme and sustained muscle injury. If bleeding problems do not supervene, a muscle biopsy is recommended in patients with similar rhabdomyolysis.

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See also pp 582-588.

Perforated appendix in the incarcerated femoral hernia

A place for preperitoneal repair

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Herniation of the appendix into the femoral hernia sac is unusual, and appendicitis in the femoral hernia sac is rarer still, to our knowledge. There are only 63 reported cases of the latter in the literature.[1] The first case of a femoral hernia with appendicitis was reported by DeGarengot in 1731.[2] The first successful appendectomy performed through a hernia sac, albeit an inguinal hernia, was by Amyand in 1736.[3]

An acutely inflamed appendix in a femoral hernia is almost never diagnosed prior to surgery. Most often the diagnosis of this condition is incarcerated or strangulated hernia. Three cases of perforated appendicitis in incarcerated groin hernias are reported in this paper, followed by a discussion of the preperitoneal surgical approach in the treatment of this phenomenon. We believe that the diagnosis of a perforated appendix in a femoral hernia is rarely

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made preoperatively, and that the treatment of choice is an appendectomy with a preperitoneal repair.

CASE REPORTS

Case 1. A 72-year-old woman presented to the emergency room with tenderness of a mass in the right side of the groin of one week's duration. The mass had first appeared one month prior to admission. The patient was otherwise asymptomatic and afebrile. Physical examination showed an erythematous, nonreducible, fluctuant, right-sided groin mass. The rest of the abdominal examination was unremarkable. The white blood cell count was 11,000/mm^3 with a left shift. The abdominal radiograph showed subcutaneous emphysema in the right lower quadrant.

The diagnosis was a strangulated femoral hernia, and the patient was taken to the operating room. A right groin incision was made, and a femoral hernia was identified. A perforated appendix was noted within the hernia sac. A right paramedian incision was necessary to perform the appendectomy. A Cooper ligament repair was carried out, and the skin was left open. The patient was discharged on the fifth day after surgery. The wound healed by second intention without incident.

Case 2. A 53-year-old woman was admitted in July 1986 because of the sudden onset of sharp, right lower quadrant pain that began earlier that day. The pain was accompanied by anorexia without nausea or vomiting. The patient was febrile to 101°F. Abdominal examination disclosed hypoactive bowel sounds with tenderness and guarding in the right lower quadrant. There was a tender, nonreducible mass in the right groin. The skin overlying the mass was erythematous and nonfluctuant. Tenderness was noted on rectal examination. A complete blood cell count, blood chemistry screen, and chest radiograph were within normal limits. One air-fluid level was evident on the abdominal radiograph, and there was air in the rectum. Intravenous fluids and antibiotics were administered. The preoperative diagnosis was an incarcerated inguinal hernia. A preperitoneal surgical approach was used, and an incarcerated femoral hernia with a gangrenous appendix within the hernia sac was found. Appendectomy and Cooper ligament herniorrhaphy were carried out. A Penrose drain was placed in the preperitoneal space, and the patient was continued on antibiotics. The patient did well after surgery and was discharged one week later.

Case 3. An 86-year-old woman presented to the emergency room with a two-day history of a painful mass in the right groin. There was accompanying anorexia without nausea or vomiting or diarrhea. The patient had had a left groin hernia repaired twice in the past. On physical examination, she was febrile to 102.8°F. There was a 4 × 5-cm, nonreducible mass in her right groin with extensive overlying erythema. Bowel sounds were present, and the abdomen was mildly distended. The patient's white blood cell count was 23,800/mm^3 with a left shift. Abdominal radiographs showed several air-fluid levels and air in the rectum. The diagnosis of an incarcerated femoral hernia was made. The patient underwent a groin exploration using the preperitoneal approach. An incarcerated femoral hernia was noted. The sac contained a perforated appendix with 30 mL of pus. The femoral canal was irrigated with saline. The peritoneum was opened and an appendectomy was performed. The McVay repair was then carried out. The preperitoneal space was drained with a Penrose drain. The skin was left open and healed by second intention. The patient did well and was discharged five days after surgery.

DISCUSSION

An incarcerated femoral hernia can contain any of several intraabdominal contents. Omentum, small bowel, colon, stomach, and Meckel diverticulum have all been reported.[4] The hernia sac is usually empty. Findings of an appendix within the femoral hernia have also been reported. Wakely[5] encountered such a finding in 1% of patients in a series of 655 femoral hernias. An inflamed appendix is a less common finding, with 62 reported cases.[1] Rarer still is the case of the perforated appendix within a femoral hernia. Twelve cases have been previously reported, and three additional cases are reported here (Table I).

The preoperative diagnosis of a femoral hernia containing an inflamed appendix is seldom if ever made.[6] Acute appendicitis in a femoral hernia often occurs in women in their 60s with a right-sided femoral hernia. In the review by Thomas et al,[7] the correct preoperative diagnosis was never made. The symptoms of appendicitis are overshadowed by the findings of an incarcerated femoral hernia. Pyrexia and leukocytosis are variable, and their more than occasional absence may be explained by a tight femoral hernia neck which prevents the spread of pus throughout the abdomen.

Some think that the presence of subcutaneous air over the right groin in the clinical setting of an incarcerated femoral hernia lends support for a preoperative diagnosis of gangrenous appendicitis within the hernia.[6] This finding, however, is rare and cannot be considered pathognomonic. More commonly, radiographs of incarcerated femoral hernias with appendicitis show a picture of small bowel obstruction.[8]

Treatment is controversial. Watkins[1] proposed initial drainage and interval appendectomy and hernia repair. Voitk et al[6] performed incision and drainage concurrent with appendectomy and hernia repair. In their second patient, they performed celiotomy and appendectomy. Voitk et al concluded that drainage may be life saving, and subsequent fistula can be dealt with at a later time. Carey[8] performed an appendectomy and a Cooper ligament repair. Cuotolo and colleagues[9] performed incision and drainage, followed three days later by celiotomy, appendectomy, and hernia repair.

The preperitoneal approach in the repair of certain inguinal hernias has definite advantages. In recurrent inguinal hernias, the scar tissue normally encountered is avoided, making this ap-

TABLE I. Operative Approaches to a Perforated Appendix in a Femoral Hernia

Operation	Results
Appendectomy and Cooper ligament repair	
Waring (1891)[10]	Uneventful
Wakely (1938)[5]	Uneventful
Carey (1967)[8]	Wound infection
Voitk (1972)[6]	Uneventful
Thomas (1982)[7]	Wound infection
Rose and Cosgrove (1987)	Uneventful
Appendectomy and delayed hernia repair	
Hodgson (1925)[11]	Uneventful
Incision and drainage, appendectomy, and hernia repair	
Holliday (1953)[13]	Uneventful
Incision and drainage, delayed appendectomy, and hernia repair	
Gerami (1969)[12]	Uneventful
Cuotolo (1978)[9]	Wound infection
Watkins (1981)[1]	Uneventful
Appendectomy with packing	
Voitk (1972)[6]	Expired
Appendectomy and hernia repair using the preperitoneal approach	
Rose and Cosgrove (1987)	Uneventful
Rose and Cosgrove (1987)	Uneventful

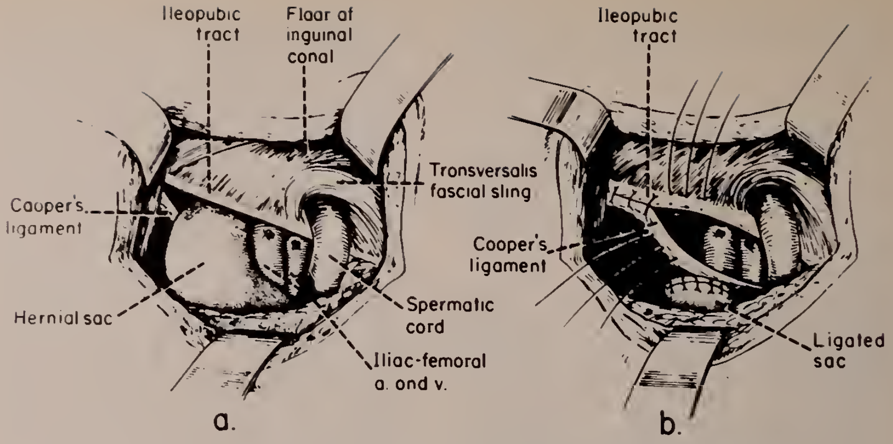

FIGURE 1. a: Anatomy of the femoral hernia; b: Illustration of the preperitoneal repair.

proach ideal for the iliopubic tract repair. Incarcerated and strangulated hernias of the groin can also be easily handled with this operation, which offers several unique advantages over the standard anterior groin approach. Reduction of the hernia is achieved through a single incision, obviating the counter-incision, which is often needed when using the anterior groin repair. Direct inspection of the blood vessels and nerves allows the constricting ring to be cut with least hazard. Early access to compromised bowel allows assessment and if necessary the peritoneum can be opened and ischemic intestine resected. Full control of the involved segment of bowel permits decreased spillage and contamination. The incision is well tolerated after surgery and pulmonary problems from splinting and incisional pain are averted.

A transverse skin incision is made two finger-breadths above the symphysis pubis extending from the border of the rectus abdominus muscle, laterally. After dividing the abdominal muscles, the preperitoneal space is entered, and anatomic landmarks are identified (Fig 1a). The hernia is reduced by traction, and the sac contents are assessed. The peritoneum is then opened and bowel resected if necessary. The defect in the femoral ring or inguinal floor can then be repaired easily and accurately without injury to the femoral vessels (Fig 1b). In two of the three cases presented here, the preperitoneal approach was used. Preoperative findings of incarceration with a high suspicion of strangulation influenced our decision to choose the preperitoneal approach over the standard anterior groin operation for the reasons outlined above. The unexpected findings of an incarcerated femoral hernia and also of perforated appendicitis within the hernia were easily addressed and corrected. An appendectomy and Cooper ligament repair were performed and drains were placed in the most dependent aspect of the preperitoneal space. Removal of a grossly perforated appendix through an anterior groin approach followed by herniorrhaphy generates a much higher risk of wound infection and subsequent recurrence. Many would opt for either drainage or appendectomy with interval herniorrhaphy. The preperitoneal approach minimizes contamination and potentially spares the patient a second procedure. The good results achieved here support the use of this technique in a similar clinical setting.

Acknowledgment. The authors thank Lynne Cooper of Beth Israel Medical Center for drawing the illustrations.

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

The role of hospital medical boards in graduate medical education

TO THE EDITOR: Graduate medical education has been under attack and criticism for many years. In recent issues of the *Journal*, various authors[1-6] have vividly presented a host of cogent suggestions and viable alternatives to the utilization of overworked and undersupervised residents, a situation which can prove dangerous to patients.

The danger of performance which is below par can be catastrophic. (Indeed, we have seen an example of this in the well publicized Libby Zion case.) Residents, in spite of the overwhelming numbers of sicker patients they are seeing in their institutions, must direct themselves to a higher level of attentiveness, exhibit a higher degree of efficiency, and maintain a reservoir of enormous amounts of human energy.

The arguments for a reduction in the number of hours required are valid and would substantially avoid many adverse patient outcomes. However, is this the solution? Will it avoid the need for better supervision on the part of attendings? Will the reduction of resident hours raise the level of care provided to patients? Though the commentaries were excellent, there was a glaring omission in their attempts to trace the source of the problem that may have led to Libby Zion's death. At no point was the role of the medical board identified in the continuum of resident supervision.

The medical board is traditionally given the overall responsibility for quality of care. It is the medical board which has the responsibility to "watch the watchers." It has become increasingly apparent that many hospital medical boards approach their crucial roles in a laissez-faire manner. They often accept the premise pro forma that their bylaws, rules, and regulations are in place, operational, and monitored by means of fail-safe protocols. In the Zion case, we do not know to what degree the medical board monitored the supervision of residents by attending physicians. Did the medical board obtain sufficient data from the directors of service concerning the quality level of resident supervision by attendings? What type of accountability took place?

It is incumbent on all participants in the medical care process, including the governing body, administration, and medical staff, that monitoring and surveillance of high integrity be implemented. Medical boards must be responsible for the organization and administration of the clinical supervision of residents as they function within all clinical departments. There are simply too many forces expecting all of us to function optimally at all times. Medical boards strenuously fought for their distinctive self-regulating style of governance in return for which they accepted an all-important assignment, the overall responsibility for quality of care. The various authors recognize and support the mandate of regulatory agencies and accrediting organizations that "the ultimate responsibility rests with an identifiable attending physician."[6] No one argues this responsibility.

However, I submit that this responsibility goes a step further and must be shared by the medical board. After all, it is the medical board which has been given the "overall responsibility for the quality of the professional services provided by individuals with clinical privileges."[7] Today's complex world of medicine mandates this body to assume roles and responsibilities never before contemplated. Perhaps Pogo was correct. "We have met the enemy and it is us."

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Measuring cancer patients' quality of life

TO THE EDITOR: As a physician who recently underwent chemotherapy for cancer, I think that the study by Walsh and Emrich[1] would have resulted in better insights had the authors chosen to survey physicians who themselves have undergone treatment for cancer.

I do not believe that quality of life can be measured. Philosophically, quality is a concept of the degree of value. Therefore, a statement about quality is a value judgment which, in turn, is a subjective abstract and, as such, impossible to measure.

This can be expressed more poetically. When the feelings of pain, nausea, and general weakness stop defying an exact description, a formula to measure quality of life will be just a matter of prescription.

For now, only matter can be measured, abstract concepts cannot. Not yet, that is.

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Professional medical conduct

TO THE EDITOR: I have been musing for some time now about why my feelings for the practice of medicine have been slowly changing over the past 20 years. Granted, the situation in which I practice has evolved and changed along with my feelings. Technology has exploded and with it new and sophisticated diagnostic and treatment possibilities have arisen, from MRI scanners to delicate cardiopulmonary transplants. However, it is not the knowledge or the hardware changes that have been of concern to me but, almost suddenly, I realize it is the environment of attitudes that has begun first to irk and now to incense me.

Medicine has become a microcosmic reflection of events outside its borders. We have witnessed and experienced the gradually escalating imposition of interminable reviews, monitors, audits, and controls on fees, charges, and, in some states, even the types of patients physicians must treat. We have been party to the shift in nomenclature from "patient" to "market share" with the advent of conglomerate medical practices such as health maintenance organizations. We have all suffered through, and continue to endure, the shame of the "malpractice and liability crisis," in which greed and anger have confused real issues of damaged lives and unrealistic expectations of perfection, on both sides of the judicial adversary system.

Given the slowly developing and enveloping "gestalt," it was my belief that physicians alone were targeted, because of long standing abuses by some, because of failures by others to practice with competence, and because earnings seemed so far out of line. Slowly, my persecutorial alarms have given way to a more general realization. Sometime in the night, stealthily, skillfully, our liberty has been stolen, without any of us stirring in our sleep. While specific for one group, current events and institutions have created a situation with far reaching significance for our communities, states, and society.

At a recent symposium sponsored by the Richmond County Medical Society on Staten Island in New York, I learned a great deal about the New York State Department of Health's Office of Professional Medical Conduct. This office functions under authority of Public Health Law Section 230. The office is charged with the investigation of complaints of medical misconduct (defined by New York State Education Law 6509), and the prosecution of physicians and physicians' assistants found guilty of such misconduct. This office may have been euphemistically named to mislead, as its clear purpose is directed at medical *misconduct*. I had, in fact, been familiar with the Office of Professional Medical Conduct as a result of my past role as president of the Richmond County Medical Society, but had attended this symposium to become more educated and informed as to its functions and mechanisms of action. Instead of becoming more educated, however, I felt threatened, intimidated, and abused

The problem, as I perceive it, is not with the goal of this bureaucratic nightmare, nor with its ends. In fact, most physicians realize, accept, and encourage a winnowing out of those among us who are in need of help to overcome emotional problems or substance dependency, or discipline for incompetent practice. It is the means to these ends that I believe are responsible for the theft of the precious few rights we all have, and we are all members of one group or another in some form. The Office of Professional Medical Conduct received over 3,400 complaints in 1987, many of which originated with individuals; some were anonymous. Once a complaint is received, identified or anonymous, an investigation takes place. The physician in question may never be told, nor is it required that he be told, that an inquiry is in process. The usual rules of due process are suspended. There is no process of "discovery" in which the defendant may seek information and review the charges. There is no procedure to enable one to face one's accuser, certainly not in those cases that are anonymously reported. The state becomes the accuser. Individuals accused of criminal acts, murder, and mayhem have more rights guaranteed them.

Considering the nature of the punishments that can and have been meted out, the suspension of one's rights has far-reaching consequences for the individual and, ultimately, for our society. The revocation or suspension of the license to practice medicine and the publication of the charges and action against physicians eliminates their ability to earn a living in medicine. It may permanently impair their reputations and prevent them from ever becoming practitioners again. Incidentally, this is not to be confused with malpractice issues. The two are very different and only occasionally overlap. However, there is insurance to help protect against the ravages of malpractice claims, just or unjust. There is no insurance coverage to help protect, defray, or dilute the effects of loss of reputation or license, just or unjust. To me, these are serious, overwhelming punishments, with no ability of the accused to defend or to review.

The costs are enormous. Another perhaps paralyzing blow has been delivered to the relationship between the physician and the patient. The mutual trust so necessary for healing and help is again under siege. With loss of license and reputation comes loss of income, home, and possessions. Without insurance to protect oneself, what recourse but to increase fees to safeguard against an anonymous attack from which there is little chance to escape, defend, or counter? It is fair warning that we are all vulnerable and soft-bellied when rights are systematically stripped away. Special groups are not so special since all of us belong to one grouping or another. I am fearful that we will not awaken from our sleep; I do not want them stealing into my home at night.

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Poor legal representation in medical malpractice cases

TO THE EDITOR: Having recently been a defendant for the first time in a malpractice suit, I was struck by the disparity between the legal services afforded the plaintiff as opposed to those afforded the defendant.

The plaintiff's attorney was with him throughout the process. In my case, different attorneys appeared at the deposition and at the trial. The preparation for the examination before trial, which forms a vital part of the trial itself, was confined to a short discussion; the attorney suggested that I come 15 to 30 minutes before the deposition so that we could go over the case. I obviously have no idea how long the plaintiff's attorney spent with his client before the pretrial examination, but judging from the evidence in court, this was a very well rehearsed performance.

The attorney who represented me at this trial informed me that he was at a disadvantage since he had not had enough time to prepare the case, having been assigned to it some ten days before the trial. However, the plaintiff's attorney had had several years to prepare for the case.

The defending attorney and I were both at great disadvantage because two expert witnesses had been engaged on my behalf, neither of whom were expert, and both of whom disagreed with me on certain points. The plaintiff's attorney made good use of this fact in his introductory statement, noting that even my expert witnesses disagreed with me. In actual fact, I believe I was far more of an expert witness than either of them and clearly the witnesses were ill chosen. (One was a practicing general surgeon with no special expertise in the area involved and with no academic background, and the other was a vascular surgeon with no expertise in a general surgical case.) I believe that I should have been consulted about the expert witnesses, as I could have obtained experts in the field with both academic and clinical backgrounds. It was both embarrassing and detrimental to my case to have the plaintiff's attorney point out that my expert witnesses thought I was wrong on several points.

With large settlements being common, and the medical professional paying large sums of money for malpractice insurance, I think that it is a disgrace that the representation for the physician can be so inferior to that of the plaintiff. I am writing this letter in the hope that some concerted action can be taken by others who feel that they have also been poorly represented.

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Recurrent syncope

TO THE EDITOR: We describe a patient with severe and disabling symptoms of several months' duration prior to the time the correct diagnosis was made.

Case Report. A 75-year-old man was admitted to Cabrini Medical Center in August 1986 because of progressive orthostatic dizziness, 15–20 syncopal episodes leading to severe disability, forgetfulness, slurred speech, and difficulty in rising from a recumbent position for two months. A prostatectomy with a bilateral orchiectomy had been done five years earlier for prostatic carcinoma. The patient was impotent following surgery, and urinary incontinence developed six months after the operation. He had been admitted to another hospital for dizziness and syncope 20 days prior to this admission.

Computed tomographic (CT) scan of the head, Holter monitor, echocardiogram, and cardiac enzyme and isoenzyme levels were within normal limits. The ejection fraction was 69%. The patient was discharged but readmitted one day later. On examination he was conscious and alert. A representative sample of physiological response to change in posture was as follows: blood pressure and pulse rate supine, 164/70 mm Hg and 68/min, respectively; standing for 30 seconds, 60/0 mm Hg and 76/min. Upright he was dizzy and light headed, but he felt better when sitting or lying down. His general condition was good.

The neurologic examination revealed normal pupillary reaction to light and accommodation, rigid facies, cogwheel rigidity, persistent glabellar tap, short-stepped festinating gait, normal tendon reflexes, flexor plantar responses, and absent bulbocavernous reflex. Unresponsiveness to a Valsalva maneuver and 0.5 mg atropine intramuscularly suggested autonomic failure.

The following tests were within normal limits: urinalysis, complete blood count, liver function tests, fasting blood sugar, serum calcium, phosphorous, creatinine, serologic tests for VDRL, electrocardiogram, echocardiogram, Holter monitor, electromyography and nerve conduction studies, electroencephalogram, CT scan of head, and bone scan. Serum cortisol and aldosterone levels in recumbent and upright postures were within normal limits. The patient received physiotherapy, elastic stockings, and 9-alpha-fluorocortisone, 0.5 mg orally per day. His blood pressure stabilized to 115/60 mm Hg in the supine position, with a pulse rate of 79/min, and to 110/60 mm Hg upright, with a pulse rate of 80/min. He could walk with the support of a walker. He was followed monthly; pedal edema developed at the end of six months. The 9-alpha-fluorocortisone was reduced to 0.1 mg orally per day. At six months of follow-up no episodes of syncope were reported.

Discussion. This patient's illness—idiopathic autonomic failure—began with incontinence and was followed approximately five years later by dizziness, syncope, and extrapyramidal dysfunction. All these features are characteristic of Shy-Drager syndrome. The range of neurologic abnormalities seen in Shy-Drager syndrome includes parkinsonian features, ataxia, impotence, urinary incontinence, and pyramidal dysfunction. On histologic evaluation, degeneration of cells is seen in the intermediolateral columns, olivo-ponto-cerebellar tracts, and striatonigral tracts, and Lewy bodies are present. There is a variable period of one month to ten years during which signs of degeneration in other systems may precede orthostatic hypotension; however, orthotostatic hypotension may appear before signs appear in other systems.[1,2]

Thomas et al,[3] in their discussion of orthostatic hypotension, have described three major approaches to symptomatic relief: mechanical measures, plasma and extracellular fluid volume expanders, and drug therapy. Elevation of the head and of the bed by 12–15 inches during sleep moderates orthostatic dizziness on rising in the morning. A custom-fitted elasticized garment extending from the metatarsals or ankle to the costal margin offers a gradient of counterpressure. The garment should be donned while the patient is recumbent.[4] Plasma and extracellular fluid expanders, a high sodium intake of 150 mEq/day or more as tolerated, and 9-alpha-fluorocortisone in doses 0.1 mg to 1 mg orally per day are essential. Common side effects such as congestive heart failure, peripheral edema, hypertension, and hypokalemia have to be considered. Pharmacologic agents such as L-dopa to alleviate parkinsonian symptoms are often poorly tolerated, and their effects on extrapyramidal symptoms are often unimpressive.[5] Physiotherapy and rehabilitative measures offer little more. This syndrome leads to disability and death within a few years.[6] This case illustrates that a patient with recurrent orthostatic syncope should be observed for orthostatic hypotension, and those patients with orthostatic hypotension should be followed closely for the development of somatic neurologic manifestations before a definitive diagnosis is made.

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See also pp 570–572.

ANNUAL PROGRESS IN CHILD PSYCHIATRY AND CHILD DEVELOPMENT

Edited by Stella Chess, MD, Alexander Thomas, MD, and Margaret Hertzig, MD. 643 pp, illustrated. New York, Brunner/Mazel Publishers, 1988. $45.00 (hardcover)

The year 1988 marks the 20th year that the *Annual Progress in Child Psychiatry and Child Development* has been published. The fact that this series has such longevity testifies to the need it has filled for all professionals who work with children and to the care and wisdom with which Stella Chess and Alexander Thomas selected the papers included in each volume. The 1988 volume (covering the year 1987), however, has two major changes over the previous volumes. It has, first of all, an author and subject index encompassing the contents of all 20 years of *Annual Progress*. As such, the indexes reflect major contributions of leading theorists, researchers, and clinicians as these contributions have unfolded since 1968. The subject index lists the papers that have appeared in the 20 volumes not only by subject, but also by volume. It is thus possible to trace the change (as reflected by the titles of the papers) within a particular area as it has progressed from 1968 to 1988.

The second major change in the 1988 volume is the addition of Margaret Hertzig to the editorial staff. As Chess and Thomas state, "... each year we ourselves grow a year older. For *Annual Progress* to maintain the standard we have set for it, it becomes necessary to share the editorial burden with a younger colleague." Dr Hertzig is an excellent choice for this task. Not only has she intimate acquaintance with the New York Longitudinal Project and thus is familiar with the thinking of Drs Chess and Thomas, but she is a productive researcher in her own right, a careful reporter who writes with clarity and style.

The 1988 volume, like previous volumes, contains 32 papers categorized in eight general areas: infancy studies (4 papers), developmental issues (6 papers), cognition and learning (3 papers), temperament studies (3 papers), special stress and coping (4 papers), specific clinical syndromes (5 papers), other clinical issues (5 papers), and child abuse (2 papers). The papers themselves are of two general types: review articles and research papers. Nine of the 32 papers may be considered review articles although each research paper generally surveys the literature pertinent to the special research.

In a thoughtful discussion ("Child Psychology: The Future"), H. R. Schaffer points out that the complexity of processes involved in development will make the one-variable type of research less and less common and the lone researcher with limited skills and limited methodology will give way to team research. This trend has implications for more flexible training of professionals in research skills. Research, too, will be concerned with individual variability as well as group trends. Arnold Sameroff ("Environmental Context of Child Development") stresses the importance of the environmental-social parameter in child study and, in parallel with the generally accepted "continuum of reproductive casualty," he describes a "continuum of caretaker casualty." Barbara K. Keogh ("Future of the LD Field: Research and Practice") reemphasizes the heterogeneity of the LD concept. She sees as essential the differentiation of LD from other learning problems, the ways to organize individual variations, the relationship of the variations to treatment, and the recognition of system influences on identification and provision of services. Rachael Gittelman Klein ("Questioning the Clinical Usefulness of Projective Psychological Tests for Children") has serious reservations concerning the use of projective tests for diagnosis and understanding of children. She states, "... one needs to have serious reservations about procedures that promise understanding but fail to deliver." Keith D. McDaniel's three-part paper on "Psychopharmacologic Treatment of Psychiatric and Neurodevelopmental Disorders in Children and Adolescents," which appeared in *Clinical Pediatrics* in 1986, is included here. Unfortunately this paper tries to put too much into too little space. While it lacks the depth and extent of knowledge needed by an expert in the field, the paper may serve as an introductory guide to psychopharmacology.

"The Impact of Child Sexual Abuse: A Review of the Research" (Angela Browne and David Finkelhor) is a significant contribution, not only analyzing the methodological faults in published studies (self-selection of samples, need for control groups, the methods used for measurement of outcome variables, the relationship of sexual abuse in childhood to later adult sexual deviance, the possible effect of molestation) but also describing reactions of anxiety, fear, depression, anger, hostility, oppression, feelings of isolation and stigma, difficulty in object relations, and sexual maladjustment in the children abused.

The research studies encompass a wide spectrum. The clinical issues section, for example, includes, "An Examination of the Borderline Diagnosis in Children" (Greenman et al), "Long Term Follow-up of Anorexia Nervosa" (Toner et al), "Childhood Obsessive Compulsive Disorder" (Rapoport), "Bipolar Affective Disorder" (Pellegrini), "Turner's Syndrome (McCauley et al), and "Right-Hemisphere Deficit" (Voeller). Forensic issues are included in the section on special stress and coping. Pynoos and Eth ("Witness to Violence: The Child Interview"), describe the technique of interviewing the child who has witnessed an extreme act of violence, allowing for exploration, support, and closure in a 90-minute initial interview. Terr ("The Child Psychiatrist and the Child Witness") outlines the responsibility of the child psychiatrist in guiding the child through the stress of appearance in court as a witness. It is clear that in the reviews and research reports psychiatrists and pediatricians will find something of interest.

As has been mentioned in previous reviews of *Annual Progress*, each year there are always questions as to why some of the papers are included. Each of us has our own concept of significant contributions. It would be helpful if the editors were to list the journals surveyed and to outline at least their criteria for inclusion. This comment does not detract from the generally useful *Annual Progress*. We wish Margaret Hertzig many years of rewarding association with it.

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ADOLESCENT SUICIDAL BEHAVIOR

By David K. Curran, PhD. 208 pp. New York, Hemisphere Publishing Corp, 1987. $39.95 (clothcover)

The title of this book aptly expresses its intent, which is to describe not adolescent suicide per se, but adolescent suicidal behavior. The latter includes the attempts which occur several hundred times more often than completed suicides, the after-effects of attempts, and, most especially, the negative attitudes of adults and adolescents alike towards the teenager who survives a suicidal episode.

The book offers a thorough review of current literature, with a particular emphasis on intervention through the school. The author, a counseling psychologist with a suburban Massachusetts school system, convincingly argues that teachers and ultimately other students are most likely to learn of a teenager's suicidal thinking, and are therefore in a position to prevent the act. Since this is probably true, the emphasis is an interesting departure from the usual medical one more familiar to most physicians. As the author shows by example, epidemic adolescent suicidal behavior can be halted through effective intervention by school and community, provided teachers and students alike can stop rejecting potentially vulnerable teenagers. Citing sources as far back as 1841, the author emphasizes that such vulnerability is not universal, but is greatest among those already susceptible for other reasons. An effective prevention program must avoid further insult to a youngster's low self-esteem; it must also undertake effective referrals for care. The author aptly observes that negative attitudes towards adolescent suicidal patients are unfortunately too common among medical caretakers, as observers of any psychiatric emergency room can attest.

The author's nonmedical orientation may have led him to some brevity with respect to diagnosis and treatment. He believes, for example, that depression and suicide in this population are virtually synonymous. While acknowledging the role played by anger and impulsivity in adolescent self-destructive acts, he does not differentiate the latter cases diagnostically. Furthermore, while his descriptions of particular hazards to treatment are astute, he covers family, group and individual therapies too briefly. In particular, since he emphasizes depression, a more complete discussion of therapies for that condition should have been included. The value of greater diagnostic precision, one would hope, is the prospect for more appropriate therapies.

Adolescent Suicidal Behavior is lucid, broad, and particularly valuable in its discussion of attitudinal challenges if school systems are to fight suicidal behavior effectively. It is generally well written, with a current bibliography of over 200 relevant sources, well bound, and attractively presented. (This reviewer's middle-aged eyes would have welcomed larger print.)

The book focuses on adolescents as compared to other sources, such as Cynthia Pfeffer's *The Suicidal Child* (1986). The author's emphasis is on prevention rather than on treatment as in McIntire and Angles' *Suicide* (1980). He provides more emphasis on the educational environment than Peck, Farberow, and Litmans' *Youth Suicide* (1985) or Sudak, Ford, and Rushforth's *Suicide in the Young* (1984). The book has the advantages and disadvantages of single authorship, as compared with Wells and Stuarts' *Self-Destructive Behavior in Children and Adolescents* (1981). While it is not a complete handbook for the physician, it effectively presents a valid hypothesis that suicide in school-aged adolescents should receive attention in the school. It is a suitable addition to a medical collection on suicidology.

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GYNECOLOGY: PRINCIPLES AND PRACTICE

Edited by Zev Rosenwaks, MD, Fred Benjamin, MD, and Martin L. Stone, MD. 668 pp, illustrated. New York, Macmillan Publishing Co, Inc, 1987. $75.00 (hardcover)

The editors of this new volume announce the theme as the study of gynecology approached from the standpoint of the patient and her clinical presentation. Its stated purpose is primarily to serve the overall needs of students of gynecology, in particular medical students and residents in training. Thirty-one outstanding authorities were selected to address the various topics and, in addition to their editorial tasks, the editors have themselves contributed to several chapters.

The book is a significant addition to the medical literature. The fact that two of its three editors are reproductive endocrinologists is reflected in the content, and the material in this field is superb. The pathophysiologic basis of endocrine problems is addressed with current therapy and experimental approaches. The chapters on hirsutism and virilization and recurrent pregnancy loss present the most complete and current material on these subjects available in any text.

The chapter on sexual function, dysfunction, and counseling, topics of major importance in modern gynecologic practice, is outstanding in its range and depth. The sections on determinants of orgasmic function and dysfunction and types of sexual dysfunction are examples of the completeness of the coverage of these subjects.

The overall format of the book is good, with sections devoted to basic concepts; menstrual abnormalities; puberty, development, and the menopause; normal and abnormal reproduction; pelvic inflammatory disease; tumors and other diseases of specific gynecologic organs; and common presenting symptoms in gynecology. The final chapter in that section, entitled "Hirsutism and Virilization," would of itself justify the publication of the book.

The quality and accuracy of the book, however, would have to be described as uneven. As indicated, the sections on reproductive endocrinology are excellent, but the entire field of pelvic infections is accorded less than ten pages. Oncology is addressed in conjunction with other diseases of specific organs. While some of those chapters, particularly the one on the vagina, are excellent, the coverage of this major division of the specialty is disappointing.

This volume compares well with available texts of gynecology. Its strengths include the sections on reproductive endocrinology and its format, focusing on the patient and her clinical presentation. The weaknesses are in fact consequences of its strengths. By a wide, comprehensive, and truly excellent coverage of endocrinology, other areas suffer. If the book were written to serve the overall needs of students and residents in gynecology, then its failure to include any discussion of the breast is a serious omission in view of the stated aims of the Committee on Resident Education in this area. It is also unfortunate, perhaps due to publication deadlines, that two areas already contain out-of-date material. The section on pelvic inflammatory disease contains the 1982, and not the 1986, Centers for Disease Control (CDC) Guidelines, and neither the updated staging of carcinoma of the cervix or ovary is included, although a reference is given in the text to the existence of a new staging for cervical cancer.

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HEART DISEASE

Second Edition. By Earl N. Silber, MD. 1,922 pp, illustrated. New York, Macmillan Publishing Co, Inc, 1987. $120.00 (hardcover)

The first edition of *Heart Disease* by Drs Silber and Katz appeared in 1975. Dr Silber recently undertook the prodigious labor of producing a second edition of what is as close to a comprehensive, single-authored textbook of cardiovascular medicine as exists today. The magnitude of the task is apparent by the length (nearly 2,000 pages) and weight (12 pounds) of the text, which attempts to provide an overall framework from which internists, cardiologists, and family practitioners can approach the day-to-day problems of managing patients with heart disease. Silber is to be commended for the completion of this Herculean effort. A textbook written by one man is more capable of providing a unified approach to diagnosis and management, but it can also be prone to an idiosyncratic analysis out of step with the majority teaching on certain subjects.

The book is divided into six parts which detail normal circulatory function, abnormal function, manifestations of heart disease, diagnosis, specific forms of heart disease, and treatment. Although Silber insists in the preface that his method of organization has met the test of time and approval of the readers, it is awkward to have to consult four or five separate sections of the text to gain an understanding of a given condition. For example, mitral regurgitation is discussed in the chapter on rheumatic heart disease and again in the chapter on nonrheumatic acquired valvular disease, and finally in the chapter on surgery in patients with heart disease. This latter chapter, especially, could have been eliminated and folded into the preceding sections dealing with specific congenital and acquired conditions.

The quality of the writing itself is excellent, with many subjects explained with a clarity too seldom found in scientific writing. In chapter one, the heart is introduced as "more of a biologic engine than a pump." Sections on myocardial energetics and regulation of the performance of the heart are well written and convey a strong sense of the historical evolution of various hypotheses and their subsequent confirmation or disapproval. There are extremely good descriptions of the clinical presentation of angina and an interesting little chapter on the bedside evaluation of arrhythmias. The chapter on congenital heart disease is thorough and easy to read. The bibliographies, in general, are exhaustive and accurate although the referencing of each article with the authors' names rather than numerically makes the body of the text difficult to read at times.

Unfortunately, it would be difficult to recommend this text as a primary source book for internists and family practitioners since many chapters suffer from obsessively detailed discussions of cardiac esoterica while failing to mention state-of-the-art advances of importance. Thus, the chapter on radiologic examination of the heart discusses such rarified techniques as orthodiagraphy and radarkymography while referring to Renografin® as a newer contrast agent. There is no mention, meanwhile, of the advantages now available with truly novel nonionic contrast media. There is much overlap and redundancy between the above-mentioned chapter and a separate chapter on cardiac catheterization. One feels a stronger editor's hand would have helped consolidate the text.

The section on electrocardiography is disorganized and poorly illustrated. There is an inappropriate emphasis on the vector cardiographic appearance of common EKG abnormalities which will be difficult for the nonspecialist to follow and yet, inexplicably, there is minimal explanation of the genesis and instrumentational features of the normal scalar EKG. Even more puzzling is the total lack of discussion about the role of invasive electrophysiologic testing in the diagnosis and management of various arrhythmias.

The practitioner most likely to benefit from reading this text is the cardiologist who, assuming he has kept up with current advances, will be insulated against opinionated treatment strategies put forth by the author and yet enjoy the detailed discussions of circulatory physiology and nuances of physical examination. It is not necessarily wise advice, for example, to place every patient with new-onset angina at bed rest for ten to 14 days, yet this approach is favored by the author. Practicing cardiologists may be able to place such treatment recommendations in a more realistic perspective, whereas nonspecialists might take them literally. The cardiovascular specialist, however, is more likely to enjoy such digressions as a detailed description of the course of Kugel's artery or the value of jugular venous pulse inspection in the diagnosis of arrhythmias.

Most approaches to diagnosis and therapy described in the book, however, are accurate and sensible. The general philosophy regarding treatment strategies appears to utilize common sense and an academic's caution with regard to insisting on rigorous proof of efficacy before embracing new therapies. The flag of caution is flying too high, however, in the discussion of thrombolytic therapy of acute myocardial infarction. The author still regards this important advance as experimental therapy and, in this reviewer's opinion, should have advocated its use more strongly, especially in patients presenting early with anterior wall infarctions.

In other areas the book is also behind the times, with gross underestimates of the number of patients treated by percutaneous transluminal angioplasty and an inaccurate description of the presumed mechanisms of restenosis following this procedure. The lack of discussion of invasive electrophysiologic testing is unfortunate, as is the omission of the use of ergonovine maleate in the diagnosis of coronary artery spasm. The index is too small and not nearly comprehensive enough to aid the reader, especially in a book in which numerous chapters have to be consulted for each topic.

In summary, this book will prove useful ancillary reading for cardiologists of either the clinical or research persuasion, but as a complement rather than replacement for the standard multiauthored textbooks. For the reasons mentioned above, it is probably best avoided by internists and family practitioners as their major reference text in cardiologic disease, although anyone interested in clear, precise scientific writing will find much to admire here.

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DYING: FACING THE FACTS

Second Edition. Edited by Hannelore Wass, PhD, Felix M. Berardo, PhD, and Robert A. Neimeyer, PhD. 474 pp. New York, Hemisphere Publishing Corp, 1988. $49.95 (cloth)

This book, according to the editors, delineates the problems related to death and dying confronting individuals and society as a whole. It presents in a systematic fashion the facts of death as we know them. They see its purpose as a comprehensive basic text for the serious student, whether an undergraduate or a professional in the field.

The book is edited by three prominent people working in this area. Dr Hannelore Wass is a professor of educational psychology at the University of South Florida and the author of many articles and books on children's adaptation to the death of a parent. Dr Robert A. Neimeyer, professor of psychology at Memphis State University, has set up testing protocols and written extensively on the subject of death anxiety. Dr Felix M. Berardo, who is professor and chairperson of the Department of Sociology at the University of Florida, has written several articles on widowhood.

The book is divided into 18 chapters and several valuable appendices. Dr J. Q. Benoliel, professor and chairperson of the Department of Community Health Care Systems at the University of Washington at Seattle, brings a nurse's perspective to her chapter on institutional dying. Robert Fulton, PhD, professor of sociology and director of the Center for Death Education at the University of Minnesota at Minneapolis, adds his expertise to his chapter on the funeral in contemporary society. Robert M. Veatch, professor of medical ethics at Georgetown University, who has written extensively on various areas concerning death, has contributed a detailed chapter on the definition of death.

Other chapters include "Hospice Care for the Dying," "Suicide Prevention," "The Right to Die: Ethical and Medical Issues," and "Bereavement and Mourning," among others. The appendices include good sources for further reading, various audiovisual aids, and documents involved in the issue of death. The bibliographies at the end of the various chapters are also very good. The authors generally present their material in a clear and understandable fashion. The subdivisions of the various chapters help to clarify the book's contents and the questions at the close of each chapter help bring together the various points previously discussed.

Death and dying is often a difficult subject. Nevertheless it is extremely important that it achieve greater prominence in the medical school curriculum. This book serves as a very good basic resource for the topic of death and dying.

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MANSON'S TROPICAL DISEASES

19th Edition. By P. E. C. Manson-Bahr, MD, and D. R. Bell, MB. 1,557 pp, illustrated. Philadelphia, Pa, WB Saunders, 1988. $125.00 (hardcover)

This classic textbook of tropical medicine was first published in 1898. It was then authored by Sir Patrick Manson (1884–1922), the "father of tropical medicine," who wrote a total of six editions of the book. Manson's son-in-law, Sir Philip Manson-Bahr (1881–1966), prepared the next ten editions and now the 19th edition has been produced by his son, P. E. C. Manson-Bahr, and D. R. Bell. Both are eminent authorities in tropical medicine and spent many years working in Third World countries.

This latest edition is as comprehensive as its predecessors but represents a major departure from them in a number of ways. The most important change is in terms of the organization of the contents. The previous zoological classification of diseases used in other editions has been replaced by broader categories such as "diseases commonly presenting as fever" and "diseases commonly presenting as diarrhea."

The emphasis in this edition is on the needs of practicing physicians, a practical approach that will make the book of enormous value to those who are called upon to treat any of a broad range of parasitic and nonparasitic infectious diseases. This goal has been successfully achieved by the editors, the 26 contributors who wrote various chapters, and the ten consultants who helped with specific sections of the book.

The book is divided into 17 sections which in turn are subdivided into 72 chapters. There are also four detailed appendices covering medical protozoology, medical helminthology, entomology, and the laboratory diagnosis of parasitic disease. These appendices cover 273 pages, represent a book in themselves, and obviate the need for a separate textbook of parasitology. In some previous editions of the book the life cycles of parasites were dealt with in depth within the specific chapters themselves. Chapter coverage of these cycles is kept to a minimum, the details being covered in the appendices where they can be consulted as needed. Each chapter contains an excellent list of up to date references from which further information on specific subjects can be obtained.

During the past few years newer problems have appeared in the Third World such as acquired immunodeficiency syndrome (AIDS) and chloroquine-resistant malaria. In addition, newer diagnostic and therapeutic modalities have revolutionized how we manage a number of parasitic diseases such as schistosomiasis, onchocerciasis, clonorchiasis, and Bancroftian filariasis. The authors cover all of these newer developments in appropriate detail. A separate chapter on drugs and an appendix on laboratory diagnosis provide readers with additional information that complements that contained in specific chapters.

Medical texts that become classics often have a tendency to rest on their laurels and reappear every few years as new editions which contain little more than cosmetic changes. This is one classic textbook that does not fall into that category. It retains the best of its predecessors while exhibiting a new and current approach to some very old disease problems. The book is beautifully produced, contains numerous illustrations, charts, and figures, and has a comprehensive index. The authors are to be congratulated for having written a superbly comprehensive and up-to-date volume that retains its place of privilege as the gold standard in the field of tropical medicine.

PASCAL JAMES IMPERATO, MD
Editor

DOCTOR DOCK. TEACHING AND LEARNING MEDICINE AT THE TURN OF THE CENTURY

By Horace W. Davenport. 342 pp, illustrated. New Brunswick, NJ, Rutgers University Press, 1987. $35.00 (cloth)

I enjoyed this book: The author uses

a transcribed account of the daily rounds made at the University Hospital in Michigan by an outstanding clinician and teacher, George Dock, to illustrate the state of the art of medicine at the beginning of this century. Having had the privilege of serving as a resident under his equally famous son, William Dock, I found myself comparing the performance of the two at the bedside. The similarities were remarkable. Both were brilliant diagnosticians and excellent instructors who treated patients with kindness and dignity.

Some of the chapters are difficult to read and portions of the text are dull. However, such characteristics are common to teaching rounds.

Davenport's portrayal of the elder Dock, who was a contemporary and a friend of Osler's, is that of the ideal role model so difficult to find among our present crop of medical educators. George Dock used the knowledge then available to each medical student by demonstration and explanation at the bedside. The verbatim recordings afford the reader an opportunity to review the signs and symptoms of diseases that are now rare such as diphtheria, smallpox, plague, typhoid fever, and the complications of syphilis and tuberculosis. It was interesting to read of the difficulties our predecessors had in diagnosing acute appendicitis and heart disease. It was painful to be reminded of diseases that were fatal in 1900 but which were eliminated by advances made in prevention and treatment after 1940. Read this book slowly. If taken in small doses, it will please you as it did me.

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EXECUTIVE RETIREMENT MANAGEMENT. A MANAGER'S GUIDE TO THE PLANNING AND IMPLEMENTATION OF A SUCCESSFUL RETIREMENT

By Jack J. Leedy, MD, and James Wynbrandt. 260 pp. New York, Facts on File, Inc, 1987. $24.95 (hardcover)

The subject of career planning, whether at the beginning stage of one's work life, or as it draws to a close, often raises more questions than it answers. During our formal education and training, we quickly learn that a step-by-step progression of activities and tasks is essential to achieve personal and organizational objectives. As one's career concludes, a similar approach for retirement planning can produce the same successful results for our postemployment career stage. This volume provides a structure and process that all of us will need as retirement age approaches. Similar to the guidelines and protocols many of us utilize to assist us in the management of our jobs and organizations, Leedy and Wynbrandt have prepared a pre- and post-retirement manual.

The authors' discussion of career and life goals, management personalities, communications, relationships, health promotion, and life styles provides the reader with an easy-to-apply method to evaluate the preparation process for retirement. The book could be used as a quick reference guide on one or more subjects, or as a periodic review guide to determine if the plans one is making towards retirement are satisfactory.

Each chapter offers a self-instructional program for different aspects of retirement planning, highlighting the most important issues to include during the transition into the latter stage of one's career. Subjects such as taking inventory, assessing the impact on office and home, managing your health, and benefit planning are presented with brief definitions, illustrations, examples, vignettes, and/or exercises. Findings from research studies add to the content of each chapter. The use of inventories, lists of questions, and suggestions helps readers apply the subject matter to their personal situations. The format of the book is conducive to sharing ideas with a spouse or with friends who may be dealing with similar issues.

One possible limitation is that the vignettes and most of the examples of career planning are slanted toward large corporate and industrial sectors. Some of the unique needs and life styles of the independent practitioner or the professional who works in health care or academic institutions are not specifically addressed. Even though most of the concepts and suggestions will have meaning to all at the latter stage of their careers, other audiences will need to stretch their imaginations a little.

Executive Retirement Management is a unique text. I am aware of very few, if any, publications that deal with the topic of career transitions and retirement planning in such an in-depth and straightforward manner. I would recommend it to all in the middle to latter stages of their careers. Whether one is employed by a large organization or works independently, the information presented offers many useful guides and suggestions to overcome most of the obstacles of retirement planning. As the authors state in the preface, with the help of this text, you may put your skills and knowledge to work in retirement planning, just as you did when you were planning and managing your career.

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Resource-based relative-value scale

A recent Harvard University study headed by William C. Hsiao, PhD, has produced a resource-based relative-value scale (RBRVS) as an alternative to the system of Medicare payment based on charges for physicians' services (*N Engl J Med* 1988; 319:835–841). The study, which was funded by the Federal Health Care Financing Administration, focuses on the issue of payment to physicians for cognitive services as opposed to procedures. Fee schedules based on an RBRVS would reduce perceived inequities in the current reimbursement system and control costs. The study by Hsiao et al establishes the validity and reliability of the RBRVS system. The RBRVS study will be presented to the Physician Payment Review Commission in November 1988, which in turn is expected to report on it to Congress in March 1989. A formal Department of Health and Human Services report with RVS/fee schedules is expected to be made to Congress in July 1989 for implementation in January 1990. This is the earliest possible date for a new Medicare RVS schedule. If implemented, according to the findings of the Harvard study, the RVS system would result in substantial increases in Medicare reimbursement rates for many categories of primary care practitioners and some substantial reductions for certain surgical subspecialists.

Inspection warrant of the Office for Professional Medical Conduct held to be improper

A recent New York State appellate court decision, *Shankman v Axelrod* [528 NYS2d 937 (App Div, 3d Dept, June 1988)], has held that the Office for Professional Medical Conduct (OPMC) does not have the power to seek an inspection warrant authorizing the removal and copying of a physician's medical records.

In *Shankman*, the OPMC applied to a court and was granted an inspection warrant authorizing the OPMC to enter a physician's office; locate and identify employees present; locate, question, and examine patients present; and locate, remove, and copy patient records. In enforcing the inspection warrant, individuals present in the office, including the physician's attorney, were advised that anyone obstructing the inspection would be subject to a misdemeanor prosecution.

Following the inspection, the physician instituted proceedings to suppress the use of the materials seized by the OPMC. The supreme court in Albany held that the OPMC has implied powers under the Public Health Law to seek an inspection warrant. On appeal, however, the appellate division held that the inspection warrant was improper. In ruling that the OPMC does not have the implied power to seek an inspection warrant, the appellate division cited Public Health Law §230(10)(k,) which provides a prodcedure by which the OPMC may issue subpoenas to gain access to records for the purpose of pursuing professional misconduct investigations. The appellate division stated that in enacting Public Health Law §230(10)(k), the legislature must have intended that a subpoena, as contemplated by the statute, be the exclusive vehicle for obtaining a recalcitrant physician's records. In requiring the OPMC to gain access to a physician's records through a subpoena instead of through an inspection warrant, the physician has an opportunity to seek a motion to quash the subpoena.

Impact of the malpractice crisis on obstetrics

Rising malpractice premiums and increasing numbers of malpractice claims are having a detrimental effect on health care, spawning higher fees, increased and often excessive use of diagnostic tests and procedures, and a general sense of mistrust between patient and physician. In the field of obstetrics, technological advances have encouraged the public to develop unrealistic expectations regarding pregnancy outcomes, observed Firth et al (*Obstet Gynecol* 1988; 72:419–422). However, pregnancy and childbirth still carry large risks, and obstetricians are increasingly faced with costly, time-consuming legal battles. A few years ago, a survey by the American College of Obstetricians and Gynecologists showed that 12% of the 1,400 members surveyed had ceased practicing obstetrics because of the risk of lawsuits. In their study of 218 obstetricians in King County, Washington, Firth and colleagues sought to determine "whether professional liability problems were increasing the cost of obstetric services and threatening the accessibility of care."

The questionnaire completed by each survey participant recorded such factors as the number of deliveries per month, amount of malpractice insurance premiums, and changes in practice habits related to malpractice concerns. Lawsuits were obviously a major concern of King County obstetricians, as 59% had been the subject of a malpractice claim.

The threat of legal action caused 18% of respondents practicing obstetrics to reduce the number of deliveries they performed, and 30% to manage fewer high-risk obstetric cases. Nearly 9% of respondents who practiced obstetrics had stopped delivering babies. The authors detailed the factors that most influenced the respondents' changes in practice habits: fear of lawsuit (39%); rising malpractice premium rates (35%); and difficulty obtaining malpractice insurance (11%). Respondents also cited the threat of a malpractice suit as the main reason that more than half of them ordered more diagnostic tests and procedures in 1986 than they did in 1983. In addition, 71% of the survey participants increased their fees in 1986, primarily to offset the costs of higher malpractice premiums.

"The threat of litigation is altering the pattern of obstetric practice in a manner not always in the patient's best interest," concluded the authors of the study. "The majority of the changes translate simply into higher medical fees and more tests and diagnostic procedures than are necessary." Increasing fees and decreasing numbers of physicians delivering babies can limit the availability of adequate prenatal care. Inadequate prenatal care has serious short term and long term effects that include maternal

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NEWS BRIEFS are compiled and written by Vicki Glaser.

morbidity, stillbirths, and low birth weight. The latter has been associated with increased infant mortality and developmental abnormalities.

Efforts are needed to control malpractice premiums and mediate legal liability in order to ensure access to quality, affordable obstetric care.

Diphtheria toxin developed for use as magic bullets

Researchers are exploiting the potent cell killing ability of diphtheria toxin to develop drugs targeted to destroy specific cells. By modifying the molecular structure of the diphtheria toxin, scientists at the University of Wyoming have eliminated its ability to bind to cells, while preserving its cytotoxic capability and its ability to penetrate cell membranes. Combining the altered toxin with a cell specific agent, such as a monoclonal antibody, hormone, or other cell-binding compound, yields a "magic bullet" that is cell specific and deadly, yet harmless to surrounding tissue.

Such targeted, cytotoxic agents could prove the basis of a new family of pharmaceuticals against a variety of diseases and disorders. Candidates for treatment include colon, rectal, breast, bladder, and kidney cancers, leukemia and Hodgkin disease, a variety of viral and parasitic diseases, and such autoimmune disorders as arthritis, lupus erythematosus, insulin-dependent diabetes, and multiple sclerosis. These magic bullets could also prove effective in treating obesity, facilitating organ transplantation, and developing new contraceptive agents.

Success achieved in retinal cell transplants

A major breakthrough in research to cure degenerative eye diseases is evident in reports from two laboratories of the successful transplantation of retinal cells (*New York Times*, September 25, 1988, p A20). The researchers transplanted cells from one rat into the retina of a rat suffering from an inherited disease that causes the death of retinal cells and eventual blindness. The transplanted cells survived, prevented cell death, and prevented the otherwise inevitable blindness. These dramatic findings represent the first successful attempt to cure degenerative eye disease.

A fairly common affliction, degenerative disease of the retina occurs in an estimated 400,000 people in the US, with irreversible consequences. Macular degeneration, one example of this group of diseases, is the primary cause of blindness in Americans over age 60. The next step in developing retinal cell transplants is to test and perfect the procedure using other species, including monkeys and rabbits, and to try to increase the numbers of transplanted cells.

These exciting reports, expected to be published in the next few months, emerged from laboratories at the Bowman Gray School of Medicine at Wake Forest University (Winston-Salem, NC) and Columbia-Presbyterian Medical Center (New York, NY). The researchers cut slits in the back of the affected rats' eyes and injected fluid that contained pigmented epithelial cells from healthy rats. These cells nourish and control the growth of photoreceptor cells in the eye, which convert light to electrical signals for processing in the brain. Death of pigmented epithelial cells due to the disease state leads to the death of photoreceptor cells and blindness. It is hoped that within five to ten years retinal cell transplants could prevent blindness in many patients with degenerative eye diseases.

Are childhood cholesterol levels predictive of adult levels?

Repeated studies have demonstrated the harmful effects of high cholesterol levels in adults, primarily an increased risk of arteriosclerosis and heart disease. Lauer, Lee, and Clarke (*Pediatrics* 1988; 82:309–318) performed a longitudinal study of cholesterol levels in a group of children first evaluated at 8–18 years of age and then reevaluated when they were 20–30 years of age. The authors observed that increased cholesterol levels in childhood do correlate with a greater risk for high levels in adult life. Other risk factors for elevated adult cholesterol levels included obesity acquired during young adulthood, use of oral contraceptives, and cigarette smoking.

The investigators recorded measurements of height, weight, triceps skinfold thickness, blood pressure, and plasma cholesterol and triglyceride levels. The Quetelet index—weight divided by height squared—served as a measure of relative body weight. Overall, cholesterol levels increased slightly in both boys and girls from age five to puberty, then dropped significantly in boys during puberty, and rose for both sexes following puberty.

"Total cholesterol measurements in childhood are predictors of adult LDL (low density lipoprotein) cholesterol levels and LDL/HDL ratios, but not of adult HDL (high density lipoprotein) cholesterol." In both sexes, as childhood levels rose above the 50th percentile of total cholesterol, the risk of elevated adult levels rose progressively higher.

While the level of cholesterol in childhood is the main predictive factor for determining adult levels, other factors are also important. Smoking at least one pack of cigarettes per day was associated with lower HDL cholesterol levels and higher LDL/HDL ratios. Use of oral contraceptives was associated with significantly higher total cholesterol and LDL cholesterol levels and higher LDL/HDL ratios, but these drugs had no apparent effect on HDL cholesterol levels. A family history of elevated cholesterol produced similar findings, implying the importance of hereditary factors. Conversely, alcohol consumption—at least one drink per week—was associated with higher HDL cholesterol levels and lower LDL/HDL ratios.

Due to the predictive value of childhood cholesterol levels for adult levels and the association between cholesterol levels and heart disease, the authors stressed the need to provide special counseling for such life style choices as diet and exercise for children with elevated cholesterol levels.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Ralph Altman, MD, New York. Died July 27, 1988; age 53. Dr Altman was a 1962 graduate of the Faculte de Medecine de l'Universite de Geneve, Geneva, Switzerland. He was a Fellow of the American College of Emergency Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the the New York County Medical Society and the Medical Society of the State of New York.

Joseph Barnett, MD, Great Neck. Died August 13, 1988; age 62. Dr Barnett was a 1949 graduate of the Chicago Medical School, Chicago, Illinois. He was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Salvatore Joseph Baudo, MD, Massapequa. Died August 20, 1988; age 80. Dr Baudo was a 1936 graduate of the New York Medical College, New York. He was a Diplomate of the American Board of Family Practice. His memberships included the American Academy of Family Physicians, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Harold Birnkrant, MD, Baldwin. Died August 14, 1988; age 84. Dr Birnkrant was a 1932 graduate of the New York Medical College, New York. He was a member of the American Psychiatric Association, the American Society of Clinical Hypnosis, the Nassau County Medical Society, and the Medical Society of the State of New York.

Waldemar Harris Boldt, MD, Binghamton. Died July 24, 1988; age 80. Dr Boldt was a 1934 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the Broome County Medical Society, and the Medical Society of the State of New York.

James Derwin Bradley, MD, Houston, Texas. Died May 31, 1988; age 81. Dr Bradley was a 1935 graduate of Northwestern University Medical School, Chicago, Illinois. He was a Fellow of the American College of Surgeons. His memberships included the American Academy of Compensation Medicine, Inc, the American Occupational Medical Association, the Medical Society of the County of Clinton, and the Medical Society of the State of New York.

Frank E. Ciancimino, MD, Upper Nyack. Died July 27, 1988; age 82. Dr Ciancimino was a 1932 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Angiology, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Surgery. His memberships included the American Society of Abdominal Surgeons, the Medical Society of the County of Rockland, and the Medical Society of the State of New York.

Theodore S. Drachman, MD, Philmont. Died July 13, 1988; age 84. Dr Drachman was a 1938 graduate of the University of Minnesota Medical School, Minneapolis, Minnesota. He was a Fellow of the American College of Preventive Medicine and a Diplomate of the American Board of Preventive Medicine, Inc. His memberships included the American Public Health Association, the Pan American Medical Association, the Medical Society of the County of Columbia, and the Medical Society of the State of New York.

Milton Earle Eisen, MD, New York. Died August 3, 1988; age 81. Dr Eisen was a 1936 graduate of the University of Toronto Faculty of Medicine, Toronto, Canada. He was a member of the New York Cardiological Society, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Donald Mansfield Ervin, MD, Palmetto, Florida. Died June 17, 1988; age 73. Dr Ervin was a 1943 graduate of the University of Rochester School of Medicine-Dentistry, Rochester, New York. He was a member of the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Medical Society of the County of Chautauqua, and the Medical Society of the State of New York.

Sidney Feyder, MD, Rochester. Died July 29, 1988; age 77. Dr Feyder was a 1936 graduate of the University of Rochester School of Medicine-Dentistry, Rochester, New York. He was a Diplomate of the American Board of Urology. His memberships included the New York Academy of Medicine, the American Urological Association, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Constantine D. Generales, MD, New York. Died June 9, 1988; age 80. Dr Generales was a 1934 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. Dr Generales was a Fellow of the American College of Angiology and a Fellow of the American College of Chest Physicians. His memberships included the New York Cardiological Society, the New York Academy of Medicine, the Aerospace Medical Association, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Aaron M. Gold, MD, Bronx. Died May 17, 1988; age 82. Dr Gold was a 1931 graduate of the Harvard Medical School, Boston. He was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. He was a member of the American Academy of Orthopaedic Surgeons, the New York County Medical Society, and the Medical Society of the State of New York.

Henry N. Goldstein, MD, Great Neck. Died July 1, 1988; age 83. Dr Goldstein was a 1919 graduate of the State University of New York at Buffalo School of Medicine, Buffalo, New York. He was a member of the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Leon I. Greenberg, MD, New York. Died July 31, 1988; age 80. Dr Greenberg was a 1932 graduate of the New York Medical College, New York. Dr Greenberg was a Diplomate of the American Board of Otolaryngology. He was a member of the American Academy of Otolaryngology, the New York County Medical Society, and the Medical Society of the State of New York.

A. Richard Hatfield, MD, Utica. Died August 17, 1988; age 84. Dr Hatfield was a 1931 graduate of the McGill University Faculty of Medicine, Montreal, Quebec, Canada. He was a member of the New York Academy of Medicine, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Philip Hust, MD, Sidney. Died August 7, 1988; age 74. Dr Hust was a 1939 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a member of the Delaware County Medical Society and the Medical Society of the State of New York.

John G. Keuhnelian, MD, New York. Died August 2, 1988; age 61. Dr Keuhnelian was a 1951 graduate of the University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology. His memberships included the New York Academy of Medicine, the New York State Urological Society, the American Urological Association, the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Yela Lowenfeld, MD, New York. Died July 31, 1988; age 86. Dr Lowenfeld was a 1930 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Egon E. Lustig, MD, New York. Died July 2, 1988; age 85. Dr Lustig was a 1929 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Henry Richard Maar, MD, Yonkers. Died August 12, 1988; age 87. Dr Maar was a 1928 graduate of the Columbia University College of Physicians and Surgeons, New York. His memberships included the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Kenneth H. MacGregor, MD, Southern Pines, North Carolina. Died August 9, 1988; age 84. Dr MacGregor was a 1928 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the New York Gastroenterological Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Victor Thomas D. H. Major, MD, New York. Died April 1, 1988; age 54. Dr Major was a 1957 graduate of the University of London Faculty of Medicine, London, England. He was a member of the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Albert N. Meyerstein, MD, Farmingdale. Died July 7, 1988; age 96. Dr Meyerstein was a 1919 graduate of the Universitaet Leipzig Medizinische Fakultaet, Saxony, Germany. His memberships included the New York Academy of Medicine, the American Academy of Family Physicians, the American Public Health Association, the American School Health Association, the Suffolk County Medical Society, and the Medical Society of the State of New York.

John Patrick Murphy, MD, Elmira. Died July 16, 1988; age 82. Dr Murphy was a 1933 graduate of the Georgetown University School of Medicine, Washington, DC. He was a member of the Medical Society of the County of Chemung and the Medical Society of the State of New York.

Richard Offenbacher, MD, Brooklyn. Died August 16, 1988; age 99. Dr Offenbacher was a 1914 graduate of the Medizinische Fakultaet der Julius Maximiliams Universitaet, Wurzburg, Bayern, Germany. His memberships included the American Academy of Family Physicians, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Altug Onor, MD, Brooklyn. Died April 24, 1988; age 51. Dr Onor was a 1963 graduate of the Bursa Tip Fakultesi Istanbul Universitesi, Bursa, Turkey. Dr Onor was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Medical Society of County of Kings and the Medical Society of the State of New York.

Henry Ostern, MD, New York. Died July 24, 1988; age 85. Dr Ostern was a 1929 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Padova, Padova, Italy. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Leonard B. Pace, MD, New York. Died March 1988; age 79. Dr Pace was a 1933 graduate of the Vytauta Didziojo University Medical Fakelteto, Kaunas, Lithuania. He was a Qualified Fellow of the International College of Surgeons. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Ely Perlman, MD, Roslyn Heights. Died July 21, 1988; age 75. Dr Perlman was a 1938 graduate of the New York University School of Medicine, New York. He was a Fellow of the American College of Allergists. His memberships included the New York Academy of Medicine, the American Academy of Allergy, the American Association of Immunologists, the Nassau County Medical Society, and the Medical Society of the State of New York.

Harry Perlowitz, MD, Brooklyn. Died June 16, 1988; age 88. Dr Perlowitz was a 1925 graduate of Tufts University School of Medicine, Boston, Massachusetts. His memberships included the Academy of Psychosomatic Medicine, the American Psychiatric Association, the American Association of Psychoanalytic Physicians, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Adrian Jay Pleskow, MD, Buffalo. Died June 30, 1988; age 69. Dr Pleskow was a 1943 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. His memberships included the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Robert Walter Selfe, MD, New York. Died August 12, 1988; age 45. Dr Selfe was a 1968 graduate of the Ohio State University College of Medicine, Columbus, Ohio. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the New York Academy of Medicine, the New York Society of Laryngology and Otology, the American Academy of Otolaryngology, the American Academy of Facial Plastic and Reconstructive Surgery, the American Society of Head and Neck Surgery, the New York County Medical Society, and the Medical Society of the State of New York.

Benjamin Marvyn Shalette, MD, New York. Died April 7, 1988; age 87. Dr Shalette was a 1928 graduate of New York Medical College, New York. He was a member of the New York Cardiological Society, the New York County Medical Society, and the Medical Society of the State of New York.

Yetta Shevell, MD, Baldwin. Died July 22, 1988; age 94. Dr Shevell was a 1922 graduate of the First Leningrad Medicine Institute (I. P. Pavlov Institute), Leningrad, USSR. She was a Fellow of the American College of Chest Physicians. Her memberships included the American Thoracic Society, the Medical Society of the County of Orange, and the Medical Society of the State of New York.

Philip Miles Standish, MD, Bloomington, Illinois. Died June 6, 1988; age 76. Dr Standish was a 1936 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a member of the New York Academy of Medicine, the Ontario County Medical Society, and the Medical Society of the State of New York.

Richard David Sweeney, MD, Mineola. Died August 18, 1988; age 59. Dr Sweeney was a 1955 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Surgery. His memberships included the Nassau County Medical Society and the Medical Society of the State of New York.

Joseph Frank Szoo, MD, Lakewood, New Jersey. Died May 29, 1988; age 91. Dr Szoo was a 1922 graduate of the Orvosi Fakultas Tudomanyegyetem, Budapest, Hungary. He was a member of the American Academy of Family Physicians, the New York County Medical Society, and the Medical Society of the State of New York.

Percy J. Tuthill, MD, Orient. Died August 2, 1988; age 80. Dr Tuthill was a 1935 graduate of the Georgetown University School of Medicine, Washington, DC. He was a Fellow of the American College of Anesthesiologists. His memberships included the New York State Society of Anesthesiologists, the American Academy of Family Physicians, the American Society of Anesthesiologists, Inc, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Imre Weitzner, MD, New York. Died July 29, 1988; age 89. Dr Weitzner was a 1922 graduate of the Institutul de Medicina si Farmacie, Cluj, Romania. He was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Radiology. His memberships included the New York Academy of Gastroenterology, the Radiological Society of New York State, the American Gastro-enterological Association, the Radiological Society of North America, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Samuel J. Werlin, MD, Troy. Died July 22, 1988; age 80. Dr Werlin was a 1935 graduate of Howard University College of Medicine, Washington, DC. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Medical Society of the County of Rensselaer and the Medical Society of the State of New York.

Eugene Zele, MD, Santa Barbara, California. Died January 24, 1988; age 85. Dr Zele was a 1929 graduate of the Deutsche Univerzita Medizinische Fakulta, Praha, Czechoslovakia. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

ERRATUM

Based on information provided by the New York County Medical Society, the September issue of the *Journal* incorrectly reported the death of Dr Morton Chester Gluck of Manhattan.

Attention Delegates
Your Next Meeting

The 183rd Annual Meeting of the Medical Society of the State of New York will be held in Rochester, New York, on April 6–9, 1989

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Nominating Committee, 1989

(The committee nominates Officers, Councilors, and Trustees of MSSNY, and Delegates to the American Medical Association.)

Member-at-Large—Samuel M. Gelfand, M.D., Nassau
Chairman
First District—John A. Finkbeiner, M.D., New York
Second District—Robert E. Fear, M.D., Suffolk
Third District—Albert M. Ellman, M.D., Albany
Fourth District—Kirk R. Panneton, M.D., Schenectady
Fifth District—Theodore C. Max, M.D., Oneida
Sixth District—Antonio F. LaSorte, M.D., Broome
Seventh District—Michael J. Graney, M.D., Monroe
Eighth District—James J. Kropelin, M.D., Niagara
Ninth District—Seymour R. Stall, M.D., Dutchess
Member-at-Large—James M. Flanagan, M.D., Wayne

FOR MY PATIENTS

NUMBER 4/1988

MAKE YOUR *SWEET* HOME A *SAFE* HOME

Dear Patient,

We usually think of our home as a comfortable, safe haven from the dangers that lie in the outside world. But, in truth, more injuries take place in all the homes across our country than in all our automobiles, farms, and workplaces. Some 20 million home injuries occur each year in the United States, and about half a million of them are bad enough to require hospitalization.

Falls and burns account for the most serious cases, and it is known that over 30 percent of adults over age 65 fall down in their homes each year.

Here is the breakdown of where the highest number of these home injuries occur:

- **stairs**
- **bathroom**
- **kitchen**
- **bedroom**
- **playroom (hobbies and toys)**
- **toolshop**

Most injuries that might occur in your own home can be prevented if you will take a few minutes to walk through your home and check for the safety items listed below. If you don't find them, make sure that you write yourself a note to correct the dangerous situation as soon as possible.

ON THE STAIRS

- Well-lighted stairway
- Sturdy handrails fastened tightly on both sides of the stairway
- No frayed or loose carpeting or worn treads
- Nothing stored on stairway
- Toddler gates at top and bottom of stairway if there are small children at home

IN THE BATHROOM

- Bathtubs and showers equipped with grab bars or safety rails
- Bathtubs and showers equipped with non-skid mats
- Hot water temperature set at 110-120° F or less
- A light switch located near entrance
- Electric hair dryer, shaver, curling iron, or radio not near water in sink or tube ***(can be dangerous if plugged into outlet even if they are switched "off")***
- Shower-chair or bath-bench if elderly live at home

IN THE KITCHEN

- During cooking, pot handles turned toward the back of the stove
- Hot liquids (especially coffee pots or cups) not near edges of table or stove
- Extension cords and appliances kept away from sink and stove
- Knives and sharp tools kept out of children's reach
- Strong stepstool for reaching high shelves (standing on chairs is too risky)
- Wipe up spills on floor immediately
- Use non-skid floor wax (only)

IN THE BEDROOM

- Light switch at the entrance door
- Lamp or light switch within the reach of each bed
- Ash trays, heaters, and hot plates kept away from beds and bedding
- Electric blankets not tucked or folded tightly under mattress
- No smoking in bed

IN THE NURSERY

- Crib has slats 2 3/8" (or less) apart
- Crib mattress fits snugly between mattress and railing (no more than a two-finger space)
- Toy chest has lightweight lid or no lid or a safe-closing hinge
- Diaper-changing table has safety straps

THROUGHOUT THE HOUSE

- All electrical and telephone cords are placed out of the flow of traffic
- Electrical cords do not run beneath furniture or rugs
- Electrical wires are not nailed or stapled to walls
- Electrical cords are not frayed or cracked
- If toddlers are in the house, plastic "shock-stop" outlet plugs are inserted in unused outlets
- All electrical outlets have cover plates
- Electrical cords are out of reach of small children
- Hallways, basement, and garage are well-lighted and free of small boxes or clutter on floor
- Small rugs and runners are tacked down or slip-resistant
- Emergency numbers (including Poison Control Center number) posted on or near each telephone

Now is the right time to practice injury prevention in your own "castle". Make this safety tour today. You can truly prevent serious injuries to yourself and your family and make your home **sweet** home a ***safe*** home.

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

Office Handouts for Your Patients

This issue of the *New York State Journal of Medicine* contains a special additional item, "For My Patients." This feature will be run in future issues as well.

Use this new feature to keep your patients informed about timely issues concerning diseases, treatments and health maintenance. Simply make photocopies of the page and distribute them to your patients with your newsletter or other correspondence, or place them in your reception area for your patients to read and take home.

MSSNY and the *New York State Journal of Medicine* have waived the copyright for these handouts, so you may duplicate them without permission.

As an added feature, MSSNY will personalize this form and make copies for you, at a nominal charge. See the example below for how these forms would look with your name and address professionally printed on them. Use the order form below, and we'll ship the personalized forms to you as soon as possible.

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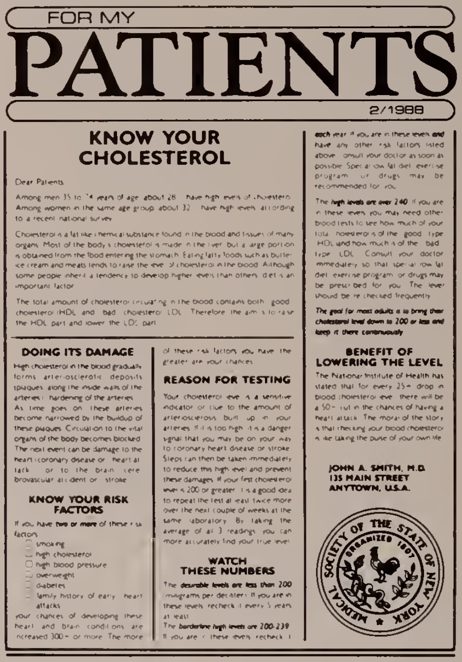

FOR MY
PATIENTS
2/1988

KNOW YOUR CHOLESTEROL

Dear Patients

Among men 35 to 74 years of age about 28 have high levels of cholesterol. Among women in the same age group about 32 have high levels according to a recent national survey.

Cholesterol is a fat-like chemical substance found in the blood and tissues of many organs. Most of the body's cholesterol is made in the liver but a large portion is obtained from the food entering the stomach. Eating fatty foods such as butter, ice cream and meats tends to raise the level of cholesterol in the blood. Although some people inherit a tendency to develop higher levels than others, diet is an important factor.

The total amount of cholesterol circulating in the blood contains both "good" cholesterol (HDL) and "bad" cholesterol (LDL). Therefore the aim is to raise the HDL part and lower the LDL part.

DOING ITS DAMAGE

High cholesterol in the blood gradually forms arteriosclerotic deposits (plaques) along the inside walls of the arteries (hardening of the arteries). As time goes on, these arteries become narrowed by the buildup of these plaques. Circulation to the vital organs of the body becomes blocked. The next event can be damage to the heart (coronary disease or "heart attack") or to the brain (cerebrovascular accident or "stroke").

KNOW YOUR RISK FACTORS

If you have **two or more** of these risk factors:

- ☐ smoking
- ☐ high cholesterol
- ☐ high blood pressure
- ☐ overweight
- ☐ diabetes
- ☐ family history of early heart attacks

your chances of developing these heart and brain conditions are increased 300% or more. The more of these risk factors you have, the greater are your chances.

REASON FOR TESTING

Your cholesterol level is a sensitive indicator or clue to the amount of arteriosclerosis built up in your arteries. If it is too high it is a danger signal that you may be on your way to coronary heart disease or stroke. Steps can then be taken immediately to reduce this high level and prevent these damages. If your first cholesterol level is 200 or greater, it is a good idea to repeat the test at least twice more over the next couple of weeks at the same laboratory. By taking the average of all 3 readings, you can more accurately find your true level.

WATCH THESE NUMBERS

The **desirable levels are less than 200** (milligrams per deciliter). If you are in these levels, recheck it every 5 years at least.

The ***borderline high levels are 200-239***. If you are in these levels, recheck it each year. If you are in these levels **and** have any other risk factors listed above, consult your doctor as soon as possible. Special low fat diet, exercise program, or drugs may be recommended for you.

The ***high levels are over 240***. If you are in these levels, you may need other blood tests to see how much of your total cholesterol is of the "good" type (HDL) and how much is of the "bad" type (LDL). Consult your doctor immediately so that special low fat diet, exercise program or drugs may be prescribed for you. The level should be re-checked frequently.

The goal for most adults is to bring their cholesterol level down to 200 or less and keep it there continuously.

BENEFIT OF LOWERING THE LEVEL

The National Institute of Health has stated that for every 25% drop in blood cholesterol level, there will be a 50% cut in the chances of having a heart attack. The moral of the story is that checking your blood cholesterol is like taking the pulse of your own life.

JOHN A. SMITH, M.D.
135 MAIN STREET
ANYTOWN, U.S.A.

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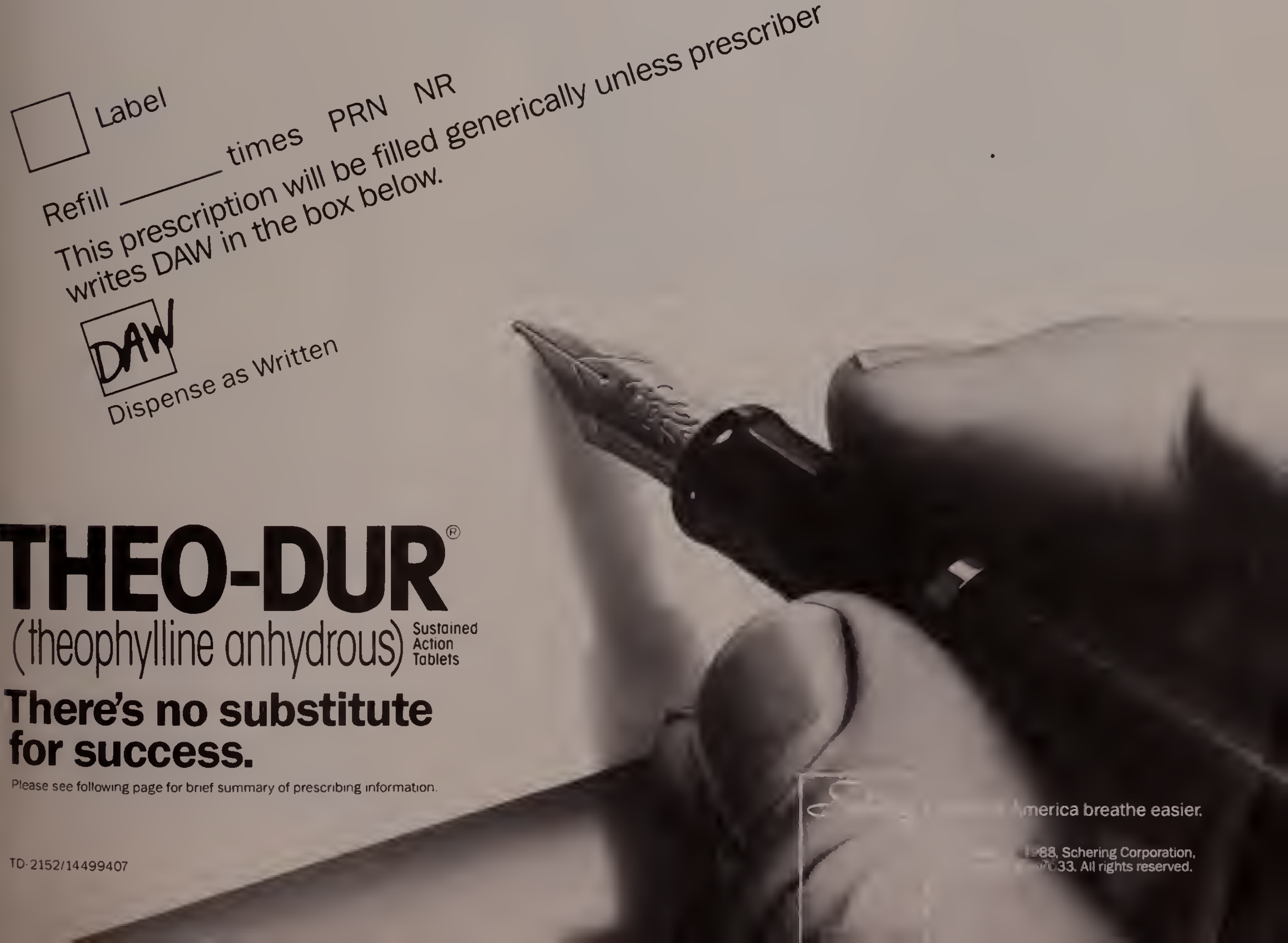

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There's no substitute for success.

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THEO-DUR®

THEOPHYLLINE (Anhydrous)
Sustained Action Tablets

INDICATIONS. THED-DUR is indicated for relief and/or prevention of symptoms of asthma and for reversible bronchospasm associated with chronic bronchitis and emphysema

CONTRAINDICATIONS: THEO-DUR is contraindicated in individuals who have shown hypersensitivity to theophylline or any of the tablet components

WARNINGS: Status asthmaticus should be considered a medical emergency and is defined as that degree of bronchospasm which is not rapidly responsive to usual doses of conventional bronchodilators Dptimal therapy for such patients frequently requires both *additional medication*, parenterally administered, and *close monitoring*, preferably in an intensive care setting

Although increasing the dose of theophylline may bring about relief, such treatment may be associated with toxicity The likelihood of such toxicity developing increases significantly when the serum theophylline concentration exceeds 20 mcg/ml Therefore, determination of serum theophylline levels is recommended to assure maximal benefit without excessive risk

Serum levels above 20 mcg/ml are rarely found after appropriate administration of recommended doses However, in individuals in whom theophylline plasma clearance is reduced *for any reason*, even conventional doses may result in increased serum levels and potential toxicity Reduced theophylline clearance has been documented in the following readily identifiable groups 1) patients with impaired renal or liver function, 2) patients over 55 years of age, particularly males and those with chronic lung disease; 3) those with cardiac failure from any cause, 4) neonates, and 5) those patients taking certain drugs (macrolide antibiotics and cimetidine). Decreased clearance of theophylline may be associated with either influenza immunization or active infection with influenza

Reduction of dosage and laboratory monitoring is especially appropriate in the above individuals Less serious signs of theophylline toxicity (i e nausea and restlessness) may occur frequently when initiating therapy, but are usually transient when such signs are persistent during maintenance therapy, they are often associated with serum concentrations above 20 mcg/ml Unfortunately, however, serious side effects such as ventricular arrhythmias, convulsions or even death may appear as the first sign of toxicity without any previous warning Stated differently *serious toxicity is not reliably preceded by less severe side effects*

Many patients who require theophylline may exhibit tachycardia cue to their underlying disease process so that the cause/effect relationship to elevated serum theophylline concentrations may not be appreciated

Theophylline products may cause dysrhythmia and/or worsen pre-existing arrhythmias and any significant change in rate and/or rhythm warrants monitoring and further investigation

The occurrence of arrhythmias and sudden death (with histological evidence of necrosis of the myocardium) has been recorded in laboratory animals (minipigs, rodents and dogs) when theophylline and beta agonists were administered concomitantly, although not when either was administered alone The significance of these findings when applied to human usage is currently unknown

PRECAUTIONS: THED-DUR TABLETS SHOULD NDT BE CHEWED OR CRUSHED

General: Theophylline half-life is shorter in smokers than in non-smokers Therefore, smokers may require larger or more frequent doses Morphine and curare should be used with caution in patients with airway obstruction as they may suppress respiration and stimulate histamine release Alternative drugs should be used when possible Theophylline should not be administered concurrently with other xanthine medications Use with caution in patients with severe cardiac disease, severe hypoxemia, hypertension, hyperthyroidism, acute myocardial injury, cor pulmonale, congestive heart failure, liver disease, in the elderly (especially males) and in neonates. In particular, great caution should be used in giving theophylline to patients with congestive heart failure Frequently, such patients have markedly prolonged theophylline serum levels with theophylline persisting in serum for long periods following discontinuation of the drug Individuals who are rapid metabolizers of theophylline, such as the young, smokers, and some non-smoking adults, may not be suitable candidates for once-daily dosing These individuals will generally need to be dosed at 12 hour or sometimes 8 hour intervals Such patients may exhibit symptoms of bronchospasm near the end of a dosing interval, or may have wider peak-to-trough differences than desired

Use theophylline cautiously in patients with history of peptic ulcer Theophylline may occasionally act as a local irritant to the G I tract although gastrointestinal symptoms are more commonly centrally mediated and associated with serum drug concentrations over 20 mcg/ml

Information for Patients: The physician should reinforce the importance of taking only the prescribed dose and time interval between doses THED-DUR tablets should not be chewed or crushed When dosing THEO-DUR on a once daily (q24h) basis, tablets should be taken whole and not split. As with any controlled-release theophylline product, the patient should alert the physician if symptoms occur repeatedly, especially near the end of the dosing interval

ORUG INTERACTIONS: **Drug-Orug**: Toxic synergism with ephedrine has been documented and may occur with some other sympathomimetic bronchodilators In addition, the following drug interactions have been demonstrated

Orug	Effect
Theophylline with lithium carbonate	Increased excretion of lithium carbonate
Theophylline with propranolol	Antagonism of propranolol effect
Theophylline with cimetidine	Increased theophylline blood levels
Theophylline with troleandomycin, erythromycin	Increased theophylline blood levels

Drug-Food. *THEO-DUR 100 mg Sustained Action Tablets* have not been adequately studied to determine whether their bioavailability is altered when given with food Available data suggest that drug administration at the time of food ingestion may influence the absorption characteristics of theophylline controlled-release products resulting in serum values different from those found after administration in the fasting state

A drug-food effect, if any, would likely have its greatest clinical significance when high theophylline serum levels are being maintained and/or when large single doses (greater than 13 mg/kg or 900 mg) of a controlled-release theophylline product are given

THED-DUR (200, 300, and 450 mg) Sustained Action Tablets The rate and extent of absorption of theophylline from THEO-DUR 200 mg, 300 mg, and 450 mg tablets when administered fasting or immediately after a moderately high fat content breakfast is similar

Drug-Laboratory Test Interactions: When plasma levels of theophylline are measured by spectrophotometric methods, coffee, tea, cola beverages, chocolate, and acetaminophen contribute falsely high values

Carcinogenesis, Mutagenesis, and Impairment of Fertility: Long-term animal studies have not been performed to evaluate the carcinogenic potential, mutagenic potential, or the effect on fertility of xanthine compounds

Pregnancy: Category C—Animal reproduction studies have not been conducted with theophylline It is not known whether theophylline can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Xanthines should be given to a pregnant woman only if clearly needed

Nursing Mothers: It has been reported that theophylline distributes readily into breast milk and may cause adverse effects in the infant Caution must be used if prescribing xanthine to a mother who is nursing, taking into account the risk-benefit of this therapy

Pediatric Use: Safety and effectiveness of THED-DUR administered
1 Every 24 hours in children under 12 years of age, have not been established
2 Every 12 hours in children under 6 years of age, have not been established

AOVERSE REACTIONS: The most consistent adverse reactions are usually due to overdose and are
1 *Gastrointestinal* nausea, vomiting, epigastric pain, hematemesis, diarrhea
2 *Central nervous system* headaches, irritability, restlessness, insomnia, reflex hyperexcitability, muscle twitching, clonic and tonic generalized convulsions
3 *Cardiovascular* palpitation tachycardia extrasystoles, flushing, hypotension, circulatory failure, ventricular arrhythmias
4 *Respiratory* tachypnea
5 *Renal* albuminuria, increased excretion of renal tubular and red blood cells, potentiation of diuresis
6 *Other* rash, hyperglycemia and inappropriate ADH syndrome

OVERDOSAGE: **Management**. If potential oral overdose is established and seizure has not occurred
A Induce vomiting
B Aominister a cathartic (this is particularly important if sustained-release preparations have been taken)
C Administer activated charcoal
If patient is having a seizure
A Establish an airway.
B Administer oxygen
C Treat the seizure with intravenous diazepam, 0 1 to 0 3 mg/kg up to 10 mg
D Monitor vital signs maintain blood pressure and provide adequate hydration

Post Seizure Coma:
A Maintain airway and oxygenation
B If a result of oral medication, follow above recommendations to prevent absorption of the drug but intubation and lavage will have to be performed instead of inducing emesis, and the cathartic and charcoal will need to be introduced via a large bore gastric lavage tube
C Continue to provide full supportive care and adequate hydration while waiting for drug to be metabolized In general the drug is metabolized sufficiently rapid so as not to warrant consideration of dialysis, however, if serum levels exceed 50 mcg/ml charcoal hemoperfusion may be indicated

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Revised 6/87

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MISCELLANEOUS

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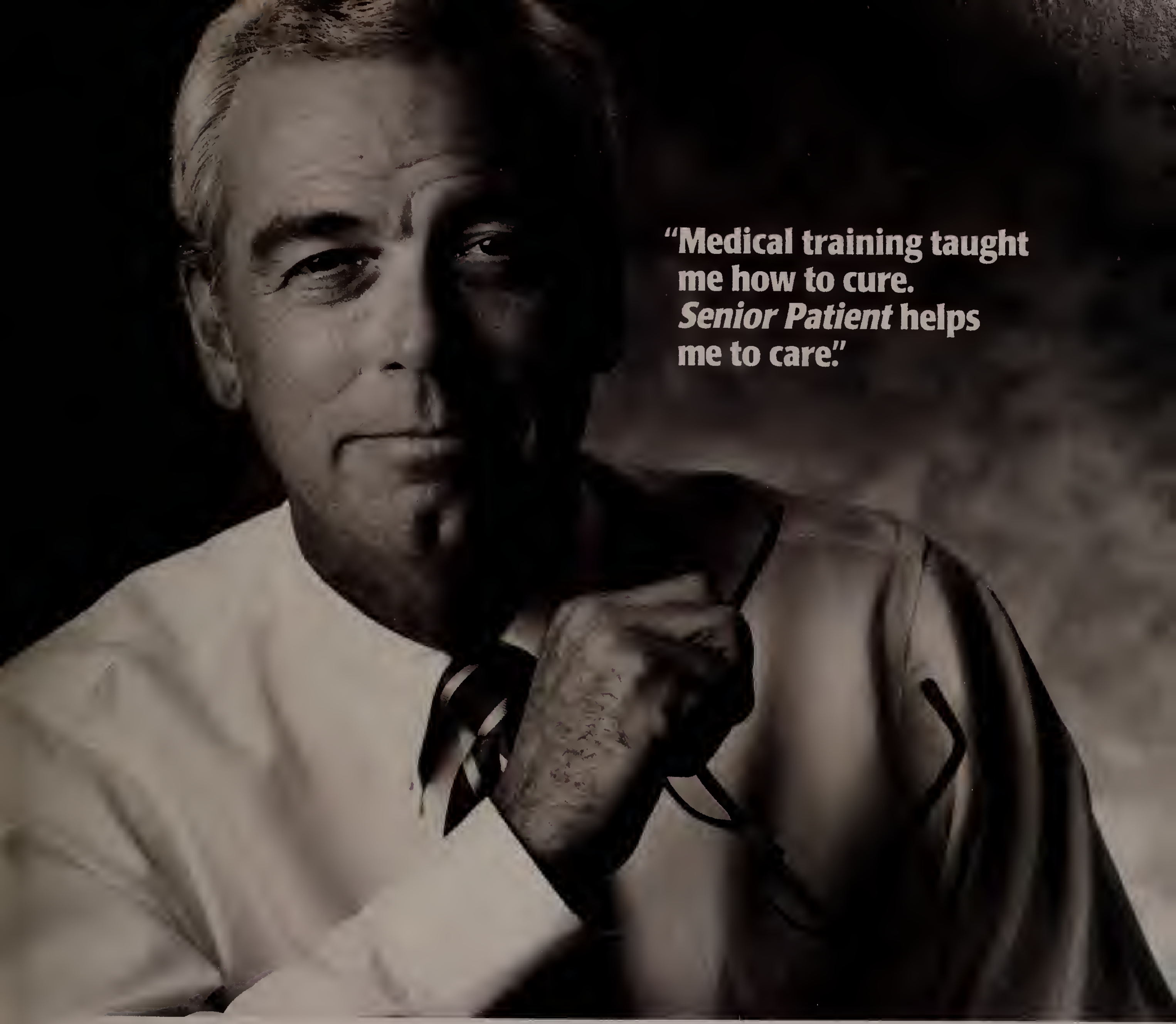
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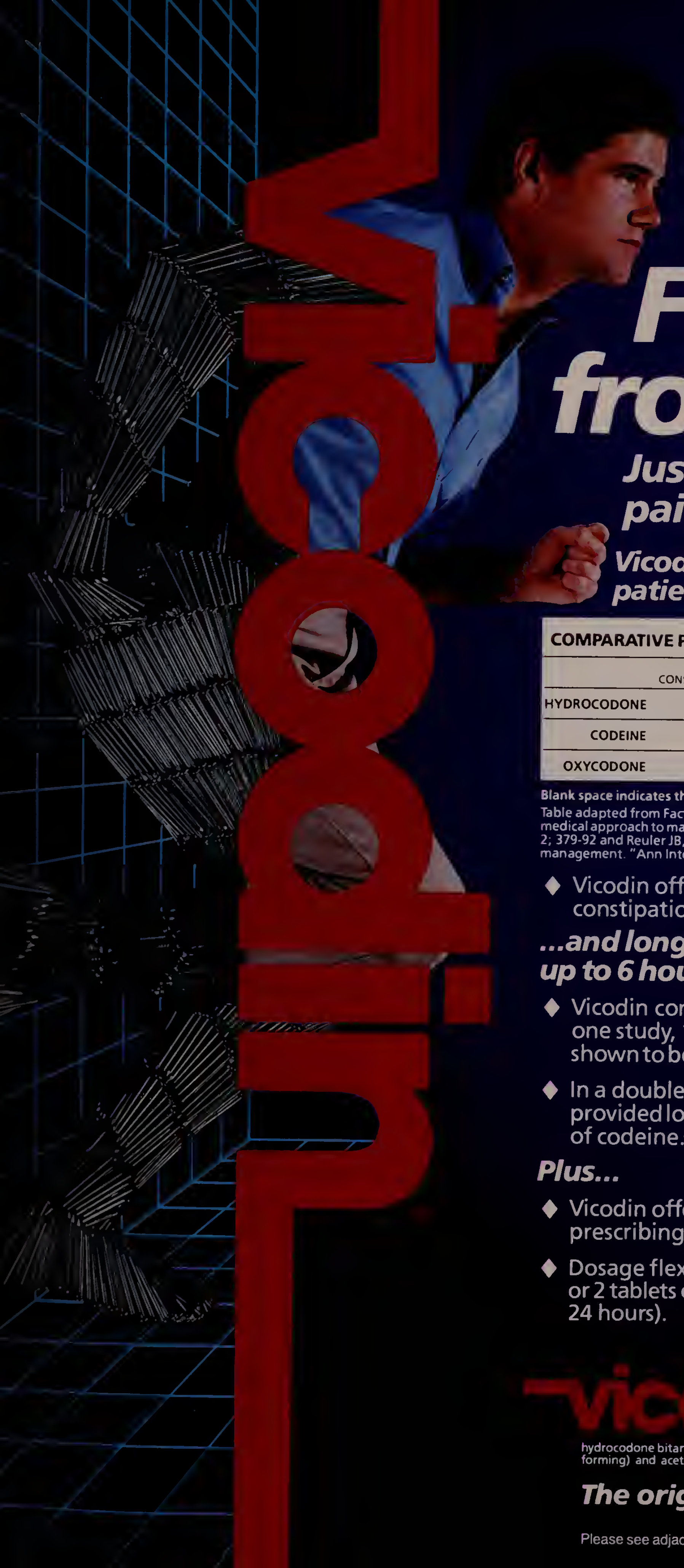

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1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
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The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone.
The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
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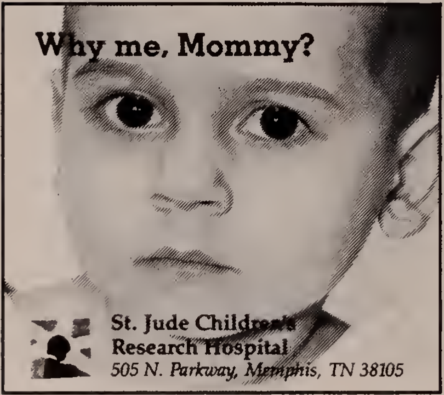

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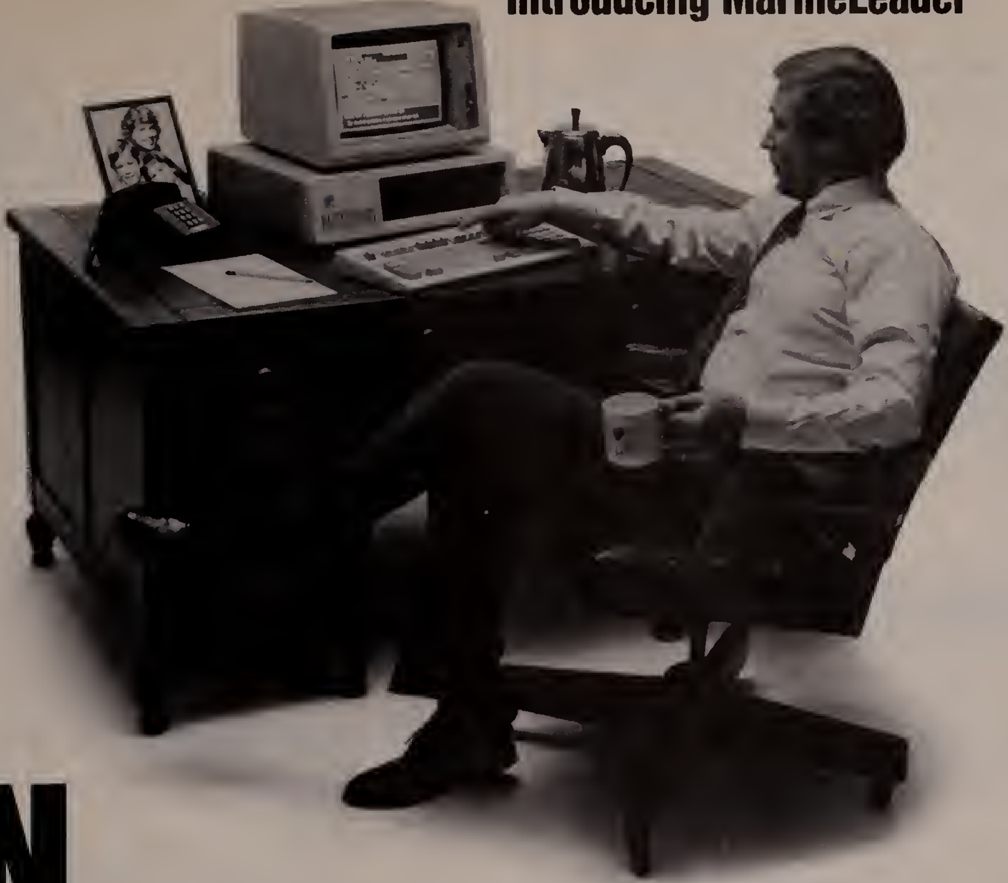

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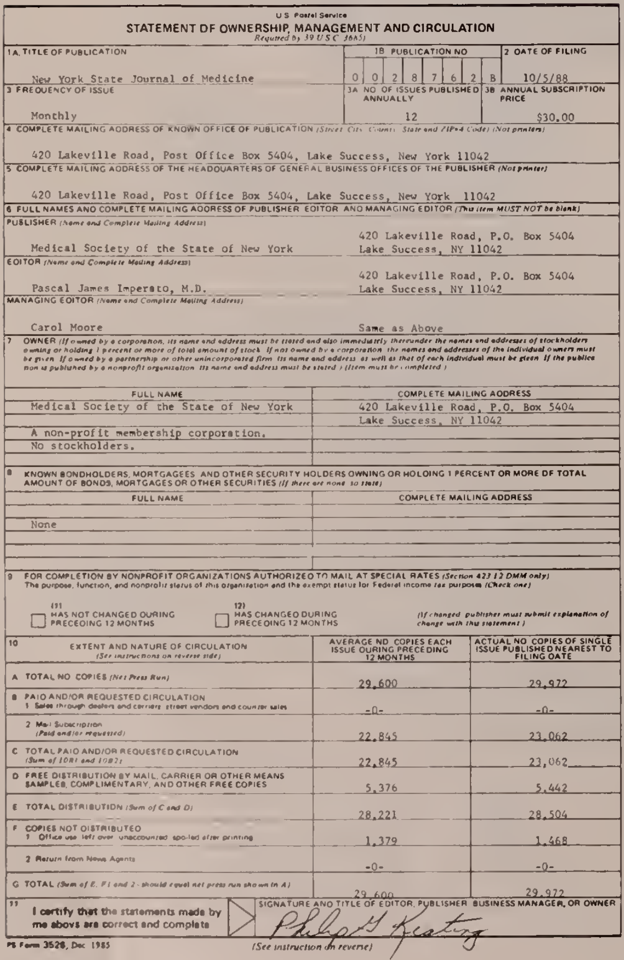

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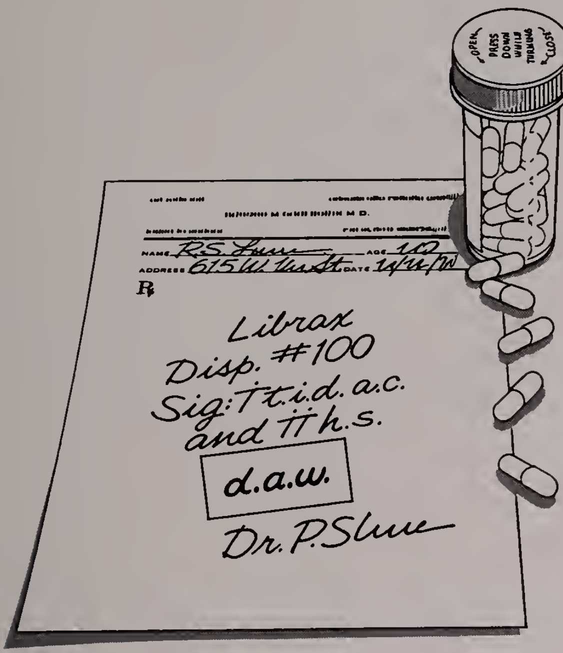

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Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

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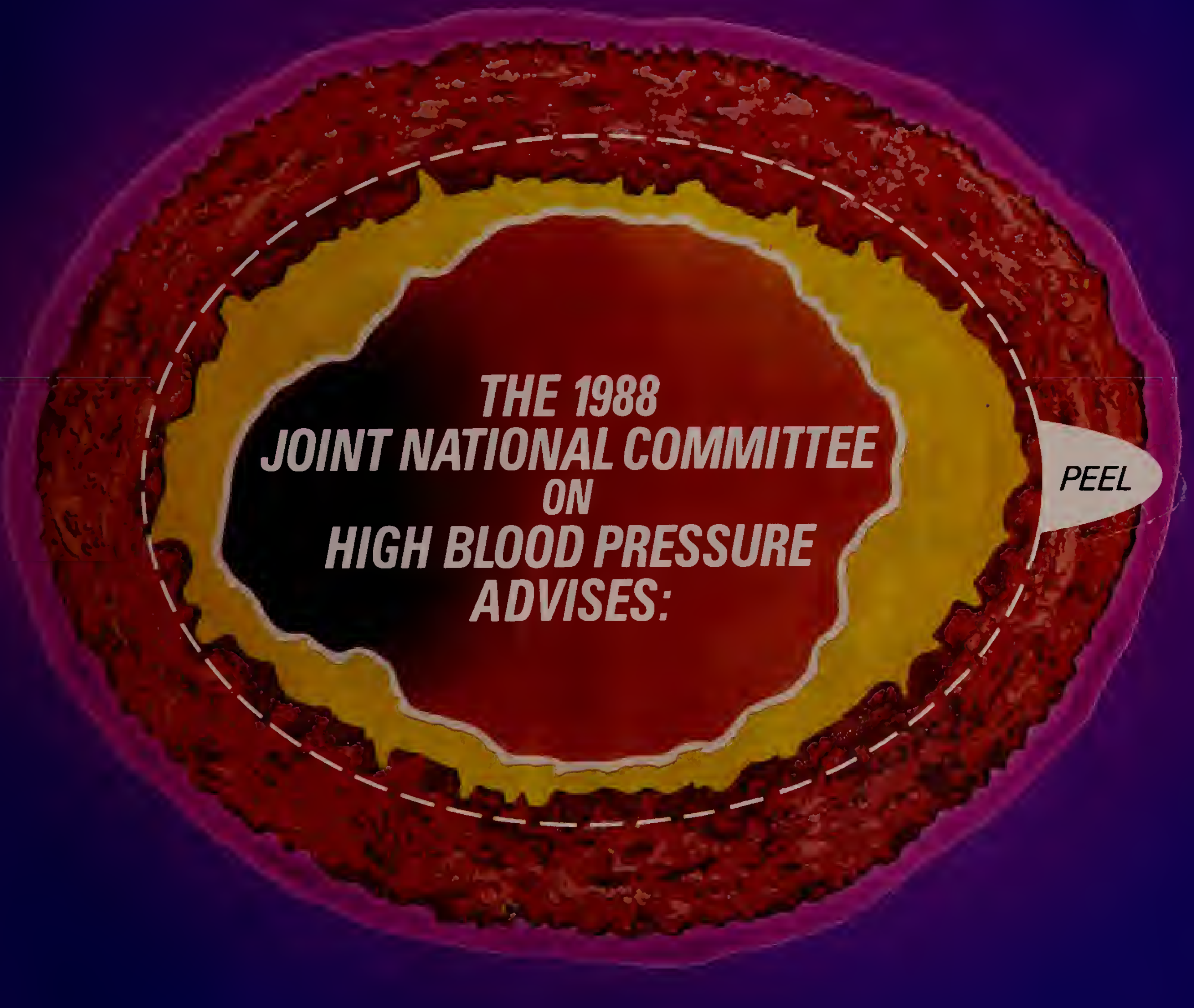
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Superior lipids performance in hypertensive patients with elevated cholesterol[1,2]*

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Brief Summary

CLINICAL PHARMACOLOGY: Pharmacodynamics: Clinical studies of terazosin used in once-a-day (majority) and b.i.d regimens with total doses usually in the range of 5-20mg/day, in patients with mild or moderate hypertension. Because terazosin, like all alpha antagonists, can cause large falls in blood pressure after the first dose or first few doses, the initial dose was 1mg in virtually all studies, with subsequent titration to a specified fixed dose or titration to a specified blood pressure end point.

Blood pressure responses were measured at the end of the dosing interval (usually 24 hrs.) and effects were shown to persist throughout the interval, with usual supine responses 5-10mmHg systolic and 3.5-8mmHg diastolic greater than placebo. The responses in the standing position tended to be somewhat larger, although this was not true in all studies. The magnitude of blood pressure responses was similar to prazosin and less than hydrochlorothiazide (in a single study). In measurements 24 hrs. after dosing, heart rate was unchanged.

Limited measurements of peak response (2-3 hrs. after dosing) during chronic terazosin administration indicate that it is more than twice the trough (24 hr.) response, suggesting some attenuation of response at 24 hrs., presumably due to a fall in blood terazosin concentrations at the end of the dose interval. This explanation is not established with certainty and is not consistent with the similarity of blood pressure response to once-a-day and b.i.d. dosing. With the absence of an observed dose-response relationship over a range of 5-20mg, i.e., if blood concentrations fall to the point of providing less than full effect at 24 hrs., a shorter dosing interval or larger dose should lead to increased response. Measure blood pressure (BP) at the end of the dose interval; if response is not satisfactory, patients may be tried on a larger dose or b.i.d. regimen. The latter should be considered if side effects, such as dizziness, palpitations, or orthostatic complaints, are seen within a few hours after dosing.

The greater BP effect associated with peak plasma concentrations (first few hours after dosing) appears somewhat more position-dependent (greater in the erect position) than the effect of terazosin at 24 hrs. In the erect position there is a 6-10 bpm increase in heart rate in the first few hours after dosing. During the first 3 hrs. after dosing 12.5% of patients had a systolic pressure fall of 30mmHg or more from supine to standing, or standing systolic pressure below 90mmHg with a fall of at least 20mmHg, compared to 4% of a placebo group.

INDICATIONS AND USAGE: Indicated for the treatment of hypertension.

CONTRAINDICATIONS: None known.

WARNINGS: Syncope and "First-dose" Effect: Terazosin, like other alpha-adrenergic blocking agents, can cause marked hypotension, especially postural hypotension, and syncope in association with the first dose or first few doses. A similar effect may occur if therapy is interrupted for more than a few doses. Syncope has been reported with other alpha-adrenergic blocking agents in association with rapid dosage increases or introduction of another antihypertensive drug. Syncope may be due to an excessive postural hypotensive effect, although occasionally the syncopal episode has been preceded by severe supraventricular tachycardia with heart rates of 120-160 bpm.

To decrease the likelihood of syncope or excessive hypotension, always initiate treatment with a 1mg dose at bedtime. The 2mg, 5mg and 10mg tablets are not indicated as initial therapy. Increase dosage slowly, and add additional antihypertensive agents with caution. Caution patients to avoid situations where injury could result if syncope occurs during initiation of therapy.

In early studies, where increasing single doses up to 7.5mg were given at 3 day intervals, tolerance to the first dose phenomenon did not necessarily develop and the "first dose" effect was observed at all doses. Syncopal episodes occurred in 3 of 14 subjects given doses of 2.5, 5, and 7.5mg, which are higher than the recommended initial dose. Severe orthostatic hypotension (BP 50/0mmHg) was seen in two others and dizziness, tachycardia, and lightheadedness occurred in most subjects. These adverse effects all occurred within 90 min. of dosing.

In multiple dose clinical trials involving nearly 2000 patients, syncope was reported in about 1% of patients, in no case severe or prolonged, and was not necessarily associated with early doses.

If syncope occurs, place patient in recumbent position and treat supportively. There is evidence that the orthostatic effect of terazosin is greater, even in chronic use, shortly after dosing.

PRECAUTIONS ***General Orthostatic Hypotension:*** While syncope is the most severe orthostatic effect of terazosin, other symptoms of lowered BP, such as dizziness, lightheadedness and palpitations, are more common, occurring in 2[illegible] of patients in clinical trials. Patients with occupations in which such events represent potential problems should be treated with particular caution.

Information for Patients: Make aware of possibility of syncopal and orthostatic symptoms, especially at initiation of therapy, and to avoid driving or hazardous tasks for 12 hrs. after the first dose, after a dosage increase, and after interruption of therapy when treatment is resumed. Caution to avoid situations where injury could result should syncope occur during initial therapy. Advise to sit or lie down when symptoms of lowered BP occur and to rise carefully from a sitting or lying position. Bothersome dizziness, lightheadedness, or palpitations should be reported to physician.

Tell patients that drowsiness or somnolence can occur, requiring caution in people who must drive or operate heavy machinery.

Laboratory Tests: Small but statistically significant decreases in hematocrit, hemoglobin, WBC, total protein and albumin were observed in clinical trials. The magnitude of decreases did not worsen with time. These findings suggest the possibility of hemodilution.

Drug Interactions: In controlled trials, terazosin was added to diuretics, and several beta-adrenergic blockers; no unexpected interactions were observed. Terazosin has also been used concomitantly without interaction in at least 50 patients on the following: 1) analgesic/anti-inflammatory (acetaminophen, aspirin, codeine, ibuprofen, indomethacin), 2) antibiotics (erythromycin, trimethoprim and sulfamethoxazole), 3) anticholinergic/sympathomimetics (phenylephrine HCl, phenylpropanolamine HCl, pseudoephedrine HCl), 4) antigout (allopurinol), 5) antihistamines (chlorpheniramine); 6) cardiovascular agents (atenolol, hydrochlorothiazide, methyclothiazide, propranolol), 7) corticosteroids, 8) gastrointestinal agents (antacids); 9) hypoglycemics, 10) sedatives and tranquilizers (diazepam).

Carcinogenesis, Mutagenesis, Impairment of Fertility: HYTRIN was devoid of mutagenic potential when evaluated *in vivo* and *in vitro*.

HYTRIN, administered in feed to rats at doses of 8, 40, and 250mg/kg/day for 2 yrs., was associated with a statistically significant increase in benign adrenal medullary tumors of male rats exposed to the 250mg/kg dose. This dose is 695 X max. recommended human dose (20mg/55kg). Female rats were unaffected. HYTRIN was not oncogenic in mice when administered in feed for 2 yrs. at a maximum tolerated dose of 32mg/kg/day.

The absence of mutagenicity in a battery of tests, of tumorigenicity of any cell type in the mouse carcinogenicity assay, of increased total tumor incidence in either species, and of proliferative adrenal lesions in female rats, suggests a male rat species-specific event. Numerous other diverse pharmaceutical and chemical compounds have been associated with these tumors in male rats without supporting evidence for carcinogenicity in man.

Effects on fertility were assessed in a standard fertility/reproductive performance study in which male and female rats were administered oral doses of 8, 30 and 120mg/kg/day. Four of 20 male rats given 30mg/kg and 5 of 19 male rats given 120mg/kg failed to sire a litter. Testicular weights and morphology were unaffected. Vaginal smears at 30 and 120mg/kg/day appeared to contain less sperm than smears from control matings and good correlation was reported between sperm count and subsequent pregnancy.

Oral use for 1 or 2 yrs. elicited a statistically significant increase in testicular atrophy in rats exposed to 40 and 250mg/kg/day, but not in rats exposed to 8mg/kg/day (> 20 X max. recommended human dose). Testicular atrophy was observed in dogs dosed with 300mg/kg/day (> 800 X max. recommended human dose) for 3 months but not after 1 yr. when dosed with 20mg/kg/day. This lesion has also been seen with Minipress®.

Pregnancy: Teratogenic effects: Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women and the safety of terazosin in pregnancy has not been established. HYTRIN is not recommended during pregnancy unless potential benefit justifies potential risk to mother and fetus.

Nonteratogenic effects: In a peri- and post-natal development study in rats, significantly more pups died in the group dosed with 120mg/kg/day (> 300 X max. recommended human dose) than in the control group during the 3-week post-partum period.

Nursing Mothers: It is not known whether terazosin is excreted in breast milk, therefore, exercise caution when administering terazosin to a nursing woman.

Pediatric Use: Safety and effectiveness have not been determined.

ADVERSE REACTIONS: The prevalence of adverse reactions has been ascertained from 14 placebo-controlled studies conducted primarily in the U.S. The studies involved once-a-day administration of terazosin as monotherapy or in combination with other antihypertensive agents, at doses ranging from 1 to 40mg. All adverse events reported during these studies were recorded as adverse reactions. Adverse events where the prevalence rate in the terazosin group was at least 5%, where the prevalence rate for the terazosin group was at least 2% and was greater than the prevalence rate for the placebo group, or where the reaction is of particular interest are summarized below. Only asthenia, blurred vision, dizziness, nasal congestion, nausea, peripheral edema, palpitations and somnolence were significantly ($p < 0.05$) more common in patients receiving terazosin than in patients receiving placebo. Other events include **[%TERAZOSIN-%PLACEBO]**: asthenia (11.3%-4.3%), back pain (2.4%-1.2%), blurred vision (1.6%-0%), depression (0.3%-0.2%), dizziness (19.3%-7.5%), dyspnea (3.1%-2.4%), edema (0.9%-0.6%), headache (18.2%-15.8%), impotence (1.2%-1.4%), libido decreased (0.6%-0.2%), nasal congestion (5.9%-3.4%), nausea (4.4%-1.4%), nervousness (2.3%-1.8%), pain-extremities (3.5%-3%), palpitations (4.3%-1.2%), paresthesia (2.9%-1.4%), peripheral edema (5.5%-2.4%), postural hypotension (1.3%-0.4%), sinusitis (2.6%-1.4%), somnolence (5.4%-2.6%), tachycardia (1.9%-1.2%), weight gain (0.5%-0.2%).

Adverse reactions were usually mild or moderate in intensity but sometimes were serious enough to interrupt treatment. Adverse reactions that were most bothersome as judged by being reported as reasons for discontinuation of therapy by at least 0.5% of the terazosin group and being reported more often than in the placebo group **[%TERAZOSIN-%PLACEBO]** are: asthenia (1.6%-0%), blurred vision (0.6%-0%), dizziness (3.1%-0.4%), dyspnea (0.9%-0.6%), headache (1.3%-1%), nasal congestion (0.6%-0%), nausea (0.8%-0%), palpitations (1.4%-0.2%), paresthesia (0.8%-0.2%), peripheral edema (0.6%-0%), postural hypotension (0.5%-0%), somnolence (0.6%-0.2%), syncope (0.5%-0.2%), tachycardia (0.6%-0%).

Additional adverse reactions have been reported, but these are not distinguishable from symptoms that might have occurred in the absence of exposure to terazosin. The following additional adverse reactions were reported by at least 1% of 1987 patients who received terazosin in clinical studies or during marketing experience: abdominal pain, abnormal vision, anxiety, arrhythmia, arthralgia, arthritis, bronchitis, chest pain, cold symptoms, conjunctivitis, constipation, diarrhea, dry mouth, dyspepsia, epistaxis, facial edema, fever, flatulence, flu symptoms, gout, increased cough, insomnia, joint disorder, myalgia, neck pain, pharyngitis, pruritus, rash, rhinitis, shoulder pain, sweating, tinnitus, urinary frequency, urinary tract infection, vasodilation, vomiting.

DOSAGE AND ADMINISTRATION: Dose and dose interval (12 or 24 hrs.) should be adjusted according to BP response.

Initial Dose: 1mg at bedtime. Observe the initial dosing regimen strictly to minimize potential for severe hypotensive effects.

Subsequent Doses: Slowly increase dose to achieve desired BP response. Usual dose range is 1mg to 5mg once a day. Some patients may benefit from doses up to 20mg/day. Doses over 20mg do not appear to provide further BP effect. Doses over 40mg have not been studied. Monitor BP at the end of dosing interval to assure control is maintained. It may be helpful to measure BP 2-3 hrs. after dosing to see if maximum and minimum responses are similar, and to evaluate symptoms which can result from excessive hypotensive response. If response is substantially diminished at 24 hrs. consider an increased dose or b.i.d. regimen. **If administration is discontinued for several days or longer, reinstitute therapy using initial dosing regimen.** In clinical trials, except for the initial dose, the dose was given in the morning.

Use With Other Drugs: Caution should be observed when terazosin is administered concomitantly with other antihypertensive agents (e.g., calcium antagonists) to avoid the possibility of significant hypotension. When adding a diuretic or other antihypertensive agent, dosage reduction and retitration may be necessary.

Revised, Sept., 1988 **Abbott Health Care Products, Inc.** North Chicago, IL 60064 B083B73

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AROUND THE STATE

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Jan 30–Feb 2. **International Pediatric Update Conference.** Trapp Family Lodge, Stowe, Vermont. Contact: Lynne Wechsler, Office of Continuing Medical Education, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

MANHATTAN

Jan 19–21. **Bicarbonate Chloride and Proton Transport Systems.** Sheraton Centre. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

AROUND THE NATION

ARIZONA

Jan 27–29. **Mammography and the Search for Breast Cancer.** 20 Cat I Credits. Scottsdale. Contact: Theresa Wade, 1351 Mt Hope Ave, Rochester, NY 14620. Tel: (716) 442-8432.

CALIFORNIA

Jan 13–15. **Advanced Seminars in Diagnostic Imaging.** 18 Cat I Credits. Ritz Carlton Hotel, Laguna Niguel. *Jan 19–21.* **Duplex Imaging.** *Jan 21–22.* **Workshop on Percutaneous Discectomy.** *Jan 23–26.* **Neuroradiology Update.** *Jan 24–27.* **Magnetic Resonance Imaging.** *Jan 24–27.* **MRI for Technologists: Symposium and Workshops.** San Diego Marriott Hotel and Marina. Contact: R. Dawne Ryals, Ryals and Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

Jan 21–22. **Breast Imaging Update.** Hyatt Regency Hotel, San Francisco. Contact: Radiology Postgraduate Education, Registration Office, University of California, Room 569U, San Francisco, CA 94142-0766. Tel: (415) 476-5731.

Jan 21–28. **32nd Tutorial on Clinical Cytology for Pathologists, Clinicians and Cytotechnologists.** 80 Cat I Credits. Sheraton-Universal Hotel, Universal City. Contact: 32nd Tutorial on Clinical Cytology, University of Chicago, 5841 S Maryland Ave, HM #449, Chicago, IL 60637. Tel: (312) 702-6569.

Jan 27–29. **New Drugs and New Indications.** 15 Cat I Credits. Granlibakken Resort, Lake Tahoe. Contact: Continuing Education S/P, University of California, 3333 California St, Room 218, San Francisco, CA 94118. Tel: (415) 476-8469.

COLORADO

Jan 12–15. **Current Concepts in Pediatrics.** 16 Cat I Credits. Marriott's Mark

(*continued on p 18A*)

ARMY RESERVE MEDICAL PROFILE NO.3

DALE L. TIPTON, M.D.

Associate Clinical Professor, Department of Otolaryngology, Head and Neck Surgery, University of California School of Medicine, San Francisco, California.
Chairman, Division of Otolaryngology, Franklin Hospital, San Francisco, California.
Lieutenant Colonel, U.S. Army Reserve.

EDUCATION University of California at Berkeley, A.B. Physiology; University of California School of Medicine, San Francisco, M.D. and Master of Science, Pharmacology.

RESIDENCY University of California School of Medicine, San Francisco: General Surgery—2 years; Otolaryngology—3 years.

FELLOWSHIPS National Institute of Health Fellow; Cancer Research Institute, University of California, San Francisco.

OUTSTANDING ACHIEVEMENTS Freshman Medical Student Research Award; Class President—2nd year medical school; Student Body President—senior year medical school; Special Award by National Institute of Health to attend and present paper at International Congress of Otolaryngology in Tokyo, Japan; Chairman, Department of Otolaryngology, San Francisco General Hospital 1970-76; Chief of Medical Staff, Franklin Hospital 1982-84.

Dr. Tipton and residents examining post-operative patient in recovery room.

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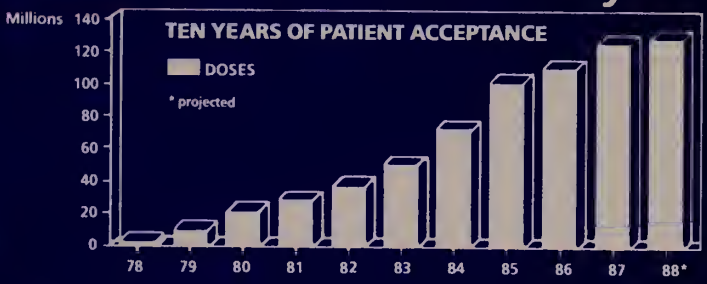

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2. Hopkinson JH III. *Curr Ther Res* 24:503-516, 1978
3. Beaver WT. *Arch Intern Med*, 141:293-300, 1981.

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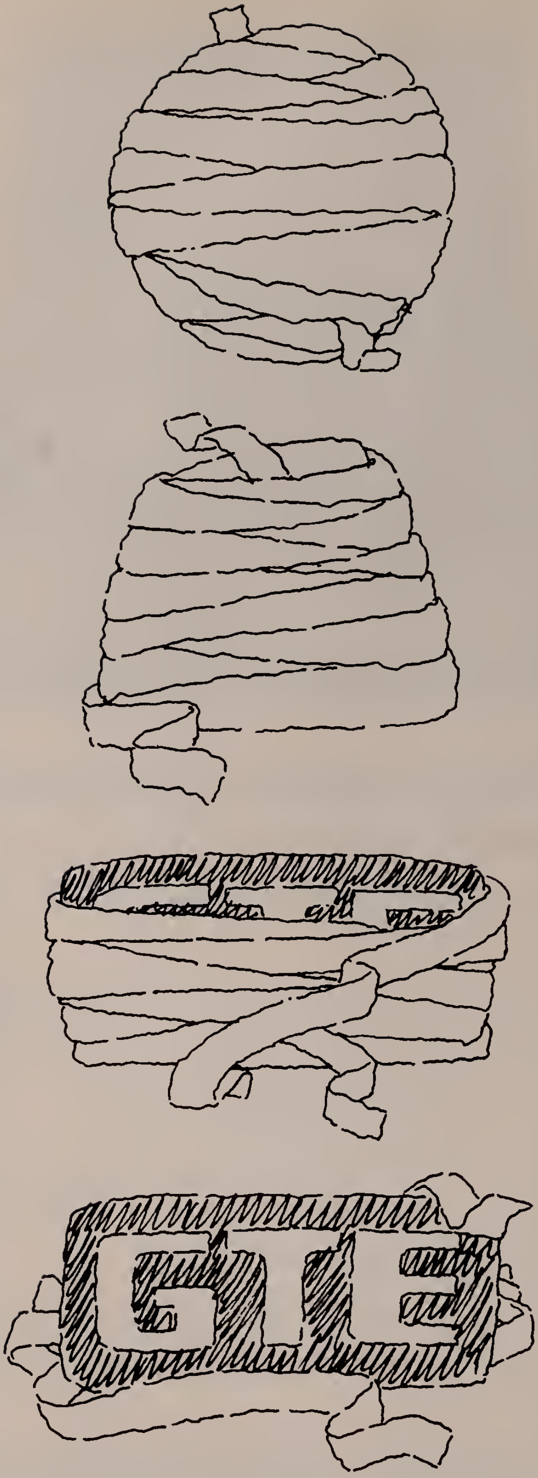

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- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

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Therapy-related adverse reactions are uncommon. Those reported include:
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NEW YORK STATE
JOURNAL OF MEDICINE

December 1988 • Volume 88, Number 12

COMMENTARIES

The new hospital code and the supervision of residents

The Ad Hoc Committee on Emergency Services was appointed in March of 1987. This committee, which extensively studied the working conditions and supervision of residents, was appointed by the New York State Commissioner of Health, Dr David Axelrod, in an effort to improve quality assurance. The report of the Committee[1] has led to a series of recommendations, some of which are now a part of the State Hospital Code. The recommendations in regard to the working conditions of residents initially sparked a great deal of controversy. It is now apparent that there is a need to change the working conditions and particularly the scheduled time commitments of residents (which originally were developed over the first 50 years of this century) to fit the radically different and complex hospital practice of 1989. A variety of national medical organizations such as the Accreditation Council for Graduate Medical Education, the Association of American Medical Colleges, the American College of Physicians, and other organizations are addressing these issues. It can be expected that the past practice of scheduling residents to work for time periods which have promoted sleep deprivation and disruption in their psychosocial conditions will be changed. The intent of these regulations is to improve quality assurance. They will also improve postgraduate medical education by permitting residents to have the time and the psychic energy to be in a condition to learn medicine and to make the kind of commitment to patients which is the essence of doctoring. Physicians who are sleep-deprived and overworked cannot make a commitment to continuity of care. The new regulations should lead to a reduction in the work load of residents and in their scheduled work time which will allow for improved patient care and resident education.

The recommendations regarding the working conditions of residents in emergency departments were virtually universally endorsed. Emergency medicine was recognized as a separate and distinctive discipline of medicine in the committee report. This should lead to the establishment of academic departments of emergency medicine by the medical schools of New York State.

The need for additional ancillary help, such as messenger transporters, phlebotomists, and nurses' aides, is also recognized and will be resolved. While addressing this need may be complicated in the beginning by manpower shortages, the manpower should be available if training programs are quickly developed. These occupations must also be upgraded and the people recruited for these important positions paid appropriately.

The issue that has provoked the most controversy is in regard to the supervision of residents. Nevertheless, it has become clear to the public and to some medical educators that supervision of residents also does not fit the realities of today's hospital practice. The intent of the new regulations with regard to supervision is to ensure that house officers do not make decisions regarding patient care without "on-site and in-person"[1(p3)] consultation with a physician who has at least finished residency training. The responsibility for patient care has been confounded in modern times by new practice patterns and different teaching attitudes that have evolved since World War II. This has led to a situation in which many private practitioners depend upon residents to care for their hospitalized patients, particularly during off hours. The idea that house officers can make important decisions, without clear-cut documentation that they have sufficient experience and wisdom to make such decisions, may be based on the thinking exhibited in testimony presented by the American College of Physicians (ACP) to the ad hoc committee. The ACP said:

> In the first place, while it is true that experience teaches senior attendings maturity that can help keep residents out of trouble, the simple truth is that, over time, many clinical skills of senior attendings—particularly the complex, technological skills with which residents need the most help—also suffer from disuse atrophy. In consequence, a PGY3 or PGY4 medical resident is, therefore, in many instances, better equipped than a senior attending to help an intern or PGY2 resident in trouble.[2]

In order for residents to learn how to assume responsibility and to make appropriate decisions, they must be supervised by attending physicians who have graduated from residencies and who have appropriate wisdom and experience to be comfortable with the responsibility of allowing residents to make decisions and assume responsibility. Resident education and supervision requires the input of experienced clinicians who, while they may not know how to use a new respirator or how to place a Swan-Ganz catheter, do know when specific procedures and therapies are indicated for specific patients and who should carry them out. Residents are in graduate medical education programs to learn how to make decisions and how to take on increasing responsibility for patients. For this to happen safely they must be taught and supervised by individuals with wisdom and experience. The need for such supervision is what led to the ad hoc committee's recommendations in regard to supervising physicians and attending physicians.

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* The views expressed in this article are those of the author and do not necessarily represent those of the New York State Department of Health.

The meaning of these recommendations is quite clear and was extensively discussed by the committee. As noted in the report, "The modern practice of in-hospital medicine requires that residents have available on-site and in-person supervising physicians."[1(p3)] The report goes on to say that:

> In hospitals that can document that the patients' attending physicians are readily available in person, the in-house supervising physicians may be in their final year of board preparation or have completed a minimum of four post graduate years of residency training as defined by specific hospital policy.[1(p3)]

This provision was based on conditions as they exist at the Strong Memorial Hospital in Rochester, where attending physicians are "readily available" within 20-30 minutes of being called. ("Readily available" as written in the hospital code means within 20-30 minutes.[3])

The 20-30-minute stipulation recognizes that individual practitioners cannot be present in the hospital 24 hours a day, seven days a week. This clearly means that alternative arrangements will be necessary when attending physicians or their equivalent surrogates cannot be readily available to respond to a resident who understands and has accepted the need for mature help in the care of a patient. In voluntary hospitals where private patients are cared for there is an obvious solution to the code requirement for "on-site and in-person supervision." Private patients have in reality contracted with their physicians or their surrogate equivalents for this kind of supervision. It is the private physicians who will arrange for equivalent surrogates if they cannot fulfill this responsibility. This responsibility can be discharged through an arrangement already in place among physicians who care for patients in hospitals that do not have house staff. In this arrangement physicians in solo, group, or other practice systems see to it that they or their equivalents are present for medical decisions, except in true emergency situations where others can be expected to deal safely with the unexpected condition. This should not pose any additional cost to the state, since patients are already paying for this service.

The care of patients who are "service" patients in both private hospitals or in large urban public hospitals is entirely different. These facilities are "resident run" and patients depend on residents for their care. In these hospitals additional funds will be necessary if the recommendations on supervision of residents are to become a reality.

Perhaps the fashion in which the New York City Health and Hospitals Corporation (NYCHHC) has tried to address the issue of supervision of residents in their institutions can be used to illustrate how care is different in the large urban public hospital. Particularly over the past five years, the NYCHHC has recognized that there is insufficient funding for attending supervision of residents. Through its 1982 Women's Health Initiative it has succeeded in providing 24-hour attending "on-site and in-person supervision" by obstetricians on those obstetrical services that have participated in the Women's Health Initiative. This indicates that, with reasonable resources, obstetricians and other personnel will commit themselves to care for patients in large urban public hospitals even when they realize that such positions include night and weekend duty. However, on the acute care specialties of medicine and pediatrics the NYCHHC has not been given the resources to pay for attending supervision. The attendings who are employed in these specialties, for the most part, do not have a contract which makes them available at night or on weekends. Furthermore, most full-time physicians in Health and Hospitals Corporation facilities have other responsibilities above and beyond those they may have for supervising residents. In some NYCHHC hospitals, in addition to "full-time" physicians, voluntary physicians (eg, those whose incomes come from private practice, research grants, or other non-HHC sources) function as unpaid attending physicians and they have even less time available for supervision of residents. The tradition and the reality on these services is that supervision is confined to morning teaching rounds which are scheduled for two hours a day. In testimony before the committee it was stated that there is virtually no supervision of residents by physicians who have finished their residency training.[4] This statement merely placed on the public record a fact that is well known to medical students, residents, and medical educators. If this system is to be changed as suggested in the committee report, then new monies must be made available to provide for supervising and attending physicians in the large urban public hospitals and those caring for "service" patients in voluntary hospitals.

The recommendations of the ad hoc committee are changing the working conditions of residents nationally. Supervision of residents in voluntary hospitals by private attending physicians does not require additional funding. Supervision of residents in large urban public hospitals and on "service cases" in voluntary hospitals will require new funding. The recommendations with regard to supervision are of equal if not greater importance. Both on-site/in-person supervision and improved working conditions are now recognized as important components for improving quality assurance. It is unfortunate that it was the public that recognized the problem and demanded that government lead the way in attempting to improve the care of hospitalized patients. It is now up to our profession to make certain that these reforms are implemented in a fashion which satisfies the public and also enhances the practice of medicine.

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Meeting the psychosocial and legal needs of women with AIDS and their families

The epidemiologic shift of newly diagnosed cases of human immunodeficiency virus (HIV) infection and frank acquired immunodeficiency syndrome (AIDS) has been clearly reported in scientific journals and the popular press. In fact, data from the New York City area recently indicated that more newly reported cases of AIDS are attributable to drug use than to homosexual or bisexual activity in men.[1] Additionally, statistics from the past year also show that the number of deaths attributable to HIV infection among drug users has begun to surpass that among gay men in New York City.[1] Increasingly, it is intravenous drug users, their sexual partners (primarily heterosexual), and even their children who are joining the swelling ranks of the over 50,000 Americans diagnosed with AIDS.

The faces of many of these "new" persons with AIDS differ drastically from those of previous years. One particular consequence of this epidemiologic shift of infection has been the increasing appearance of AIDS among young women of black or Hispanic background, the majority of whom appear to be single heads of households, with young children dependent on them for emotional and financial support.[2] Recent statistics from the Bronx indicate that females constitute 20% of those diagnosed with AIDS; 87% of these women are black or Hispanic.[3]

The debate continues in the bioethical and legal communities about the moral appropriateness of counseling women at high risk against procreation. Meanwhile, however, many of these women, already infected with the virus and overwhelmed with survival issues, are desperately in need of concrete legal and social services to help them make sense of the time that remains and to plan for the future, particularly the future care and custody of their young children. These women cannot wait for, nor will they benefit from, the results of an as yet unresolved philosophical and ideological debate. What they need is immediate, comprehensive assistance. It is our premise that such help can be uniquely provided through, and linked with, their inevitable contact with the medical community, and that caregivers have the moral obligation to try to arrange for such help.

As members of the AIDS team at Montefiore Medical Center in the Bronx, we have become all too familiar with the range of significant psychosocial and legal problems in this patient population. The team, composed of physicians, nurses, social workers, attorneys, hospice personnel, substance abuse staff, and other specialized caregivers, has come to recognize that in this population, an AIDS diagnosis is not only a medical crisis for the individual but also a social, psychological, legal, and financial crisis which affects the patient's entire family.

Many of these individual patients and their families are disempowered, disenfranchised, and alienated from traditional sources of help and support. Due to urban isolation, impoverishment, homelessness, societal bias, and other social ills that afflict urban, minority communities, they are unaware of or, more likely, unable to gain access to existing avenues of support. Because of this social isolation, we consider psychosocial and legal services to be essential components of the "package of care" available to persons with AIDS and their families, both in the acute care setting as well as in outpatient clinics. Their unavoidable and often regular contact with medical caregivers permits us to offer them these additional services which might otherwise be unavailable. At the same time, their medical caregivers have become sensitized to these "nonmedical" yet equally relevant sequellae of AIDS, with the consequent bonus of more compassionate, humane care and understanding for these patients and their families.

Recognizing the special problems facing these women, and crafting workable, expeditious solutions, requires a heightened sensitivity and a peculiar expertise as yet largely undeveloped among otherwise caring members of the AIDS service community. Our clinical exposure and experience with this patient population have enabled us to identify several recurring characteristics, some unique to this group, others generalizable to the larger AIDS population, which affect and constrain the process of planning for these women and their families.[2] The most important of these characteristics are described below.

Time Limits Imposed by the Illness. There are data to suggest that women with AIDS, particularly black or Hispanic women, tend to survive for shorter periods of time than other risk groups once the diagnosis is made, leaving less time available for future planning.[4] The frequent accompaniment of AIDS-related dementia often presents further barriers to patient participation in decision making, as the patient's health deteriorates.

Diagnosis-Precipitated Emotional Chaos and Shock. A frenzy of emotions often overwhelms both patient and family upon diagnosis. The substantive effects of blame and denial often necessitate extensive psychosocial counseling.

Existence of Dependent, Minor Children. Numerous psychosocial and legal issues arise when the parent of a young child develops AIDS. Prominent among these is the need for early legal planning for the future care and custody of minor children once the patient has died. The dilemma of how to maintain some semblance of "normalcy" for these children while their parents are ill and hospitalized is also paramount. Arrangements for child care during a single parent's hospitalization, and the provision of crisis and support counseling for survivors devastated by the illness are also necessary.

Nature and Patterns of Familial Relationships. Nontraditional but culturally specific and accepted patterns of extended family arrangements at times produce strong support systems. However, they may also cause confusion and conflict regarding the legal or de facto responsibility for both the patient's affairs and those of her children. For many affected women, the fathers of their children are absent or uninvolved, which diminishes the utility of traditional legal methods of planning, as such methods often spring from outdated, stereotypical views of what constitutes a "family" in our society.

Because of the varied living situations of these patients, we have created an array of diverse and eclectic services. Psychosocial counseling and assistance are available for issues ranging from denied entitlements to education about safer sexual practices, to mediation of conflicts among angry and frustrated family members, to counseling women about options available for child care arrangements upon their death. Legal advice and consultations, in addition to concrete legal services, address such distinct issues as arranging for durable powers of attorney, assisting the patient with financial transactions or health care decisions, drafting both living wills and traditional wills, and petitioning courts of law to appoint guardians for children whose mothers are imminently dying of AIDS.

What is perhaps most remarkable and, we believe, most appropriate, is the coordination and integration of these services with the provision of traditional diagnostic and therapeutic interventions in the medical setting. Such a method of service delivery stands as demonstrable recognition that AIDS is not merely a medical illness but a social crisis as well. It also symbolizes that these individuals are not merely "walking medical problems," whose opportunistic infections and illnesses can be separated from other aspects of their lives. They are in fact suffering human beings, many of them mothers with children, in need of help beyond the often inadequate medical care available to them. Yet they are historically tangled in a web of poverty, illness, oppression, and bureaucracy through which little hope or light can shine. Physicians have the ability, and arguably the responsibility, to assist these parents and their dependent children by working to broaden the societal definition of what is considered necessary and appropriate care for these persons with AIDS, and by arranging this linkage with the "ancillary services" described above. Other service providers, including members of the legal profession, should also be encouraged to familiarize themselves with the specific problems of these patients, and to offer their services as part of an AIDS team "package." This coordination of services is crucial and necessary for women with AIDS and their children, particularly if the latter are to survive an otherwise stormy sea of illness and decline on which they are afloat.

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Cerebral aspergillosis

Cerebral aspergillosis is a disease associated with medical progress. Its incidence has increased dramatically in the era of transplantation and improved therapy for hematologic malignancies. In this issue of the *Journal*, Barrios et al[1] present a case of cerebral aspergillosis in a leukemic child. Most of the salient features and some of the diagnostic and therapeutic dilemmas are discussed.

Prior to the introduction of antibiotics and immunosuppressive drugs, neurologic aspergillosis usually was an isolated infection that started in the paranasal sinuses or orbits and extended to the anterior fossa. The first case of aspergillosis involving the brain was reported by Oppe[2] in 1897; granulomatous meningitis started in the sphenoid sinus and extended to the optic chiasm and right internal carotid artery. Numerous subsequent reports have noted involvement of the sinuses or the orbit, sometimes resulting in a syndrome mimicking painful ophthalmoplegia (Tolosa-Hunt syndrome).[3] Rarely, isolated cerebral abscesses occur in intravenous narcotic abusers. Isolated spinal cord lesions or meningitis have also been reported in nonimmunocompromised hosts.

The first case of disseminated aspergillosis with cerebral involvement was reported in 1947.[3] This has become the most common setting in which cerebral involvement is encountered.[4-6] Central nervous system symptoms are the presenting feature in one third of these patients, and involvement eventually occurs in half. Some cases have occurred in apparently healthy infants. Most cases, however, have been associated with acute leukemia, lymphoma, or transplantation. Both renal and cardiac transplantation patients are particularly susceptible, and cerebral aspergillosis should be strongly suspected when these patients develop neurologic deficits. Most of these patients are being treated with broad spectrum antibiotics. Other predisposing factors include cardiac surgery with fungal endocarditis, hepatic failure, and Cushing disease.[4,5] Cerebral aspergillosis can accompany Cushing syndrome from a variety of sources including ectopic adrenocorticotrophic hormone production. The importance of corticosteroids as a predisposing factor in aspergillosis is well known.

The clinical picture may be nonspecific, with lethargy and obtundation. However, there are characteristic focal deficits. These often develop rapidly with stroke-like deficits or seizures. This is due to the pathogenesis of the illness, which involves hematogenous dissemination and vascular invasion. The illness usually originates in the lungs. Most patients therefore show progressive pulmonary infiltrates or cavitary lesions prior to or coincident with the onset of neurologic symptoms. This was the case in all 11 patients we reported.[3] Rarely, however, as in the case reported in this issue of the *Journal* by Barrios et al,[1] pulmonary findings may be minimal or absent.

The fungus shows a striking predilection to invade blood vessels. This results in vascular thromboses and, more rarely, mycotic aneurysms and subarachnoid hemorrhage. A recent clinicopathologic conference at Massachusetts General Hospital described a 27-year-old man with treated myelomonocytic leukemia in whom a mycotic aneurysm of the basilar artery developed associated with recurrent episodes of subarachnoid hemorrhage.[6] The involvement of large arteries is characteristic of this type of mycotic aneurysm. Stroke-like deficits or seizures are the result of vascular thrombosis, due to compromise of the vascular lumen by the fungus. Patients often show sudden and rapid deterioration.

A few other clinical features are helpful in establishing a diagnosis. The patients are almost invariably febrile, despite frequently being on corticosteroids and immunosuppressive drugs. The present case was notable for brainstem involvement. This feature was noted initially by Young et al.[6] Of the 11 cases we reported, brainstem or cerebellar signs were a presenting feature in three cases and were eventually seen in five cases.[4] These features consisted of nystagmus, dysarthria, ataxia, cranial nerve palsies, and intranuclear ophthalmoplegia.

As noted in the present case report, ancillary laboratory studies are of limited usefulness. The cerebrospinal fluid is frequently abnormal with increased white cell count and protein, however, the fungus usually cannot be cultured. Bronchoscopy with endobronchial biopsy or open lung biopsy is the most reliable means of establishing the diagnosis. The computed tomographic (CT) findings generally show poorly circumscribed, low density areas with minimal enhancement and little mass effect. The findings in the present case are typical. The CT findings do not adequately reflect the severity of the disease, even when obtained in close proximity to death. It is likely that magnetic resonance imaging (MRI) scans will prove much more sensitive in defining the extent and severity of cerebral involvement. Initial experience has shown this to be the case. They will undoubtedly be more sensitive in the posterior fossa.

The differential diagnosis of cerebral aspergillosis in the immunocompromised host is complex. The leading considerations are other opportunistic infections such as toxoplasmosis, nocardia, phycomycoses, candida, cryptococcus, and listeria. Transplant patients on chronic immunosuppressive therapy are also particularly prone to develop cerebral lymphoma.

Experience with treating cerebral aspergillosis has been uniformly disappointing.[4,5] Despite full-dose treatment with amphotericin and 5-fluorocytosine, lesions progress rapidly, and most patients die within a few weeks of onset. The major objective of treatment therefore should be prevention in high-risk groups such as patients with leukemia or transplants. The usefulness of nasal cultures in this regard has been suggested. Improved serologic tests may also become available. Early recognition and treatment of pulmonary aspergillosis is probably the most successful way to prevent central nervous system dissemination.

An interesting aspect of cerebral aspergillosis is that it is virtually never encountered as a central nervous system complication of acquired immunodeficiency syndrome (AIDS).[8-10] The most common central nervous system infection in these patients is *Toxoplasma gondii*. The presence of a severe disturbance in cell-mediated immunity alone is therefore not sufficient to result in aspergillus infections. It is likely that the use of broad spectrum antibiotics alters normal bacterial flora, allowing fungal invasion, or that the use of corticosteroids uniquely predisposes to aspergillus infection. In the present case, both factors were present. The case reported in this issue of the *Journal* is illustrative of the difficulties in making a diagnosis of cerebral aspergillosis during life. The sudden onset of stroke-like deficits and brainstem findings in a febrile immunocompromised host with pulmonary infiltrates, however, should suggest the diagnosis of cerebral aspergillosis.

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UNDERGRADUATE DEFICIENCIES AND POSTGRADUATE REQUIREMENTS

In his "Oath" Hippocrates has given us a professional standard which has held as the ideal of the relations of the physician to himself and his fellow man for almost twenty-five centuries. In his "Law" we have a statement of the essentials for the making of a physician which put beside all our writings on this subject, in this present day, easily takes first rank. In this "Law" we learn that who ever is to acquire a competent knowledge of medicine should first of all have a natural talent, for when Nature leads the way to what is most excellent, instruction in the art takes place, which the student must try to appropriate to himself by reflection, becoming an early pupil in a place well adapted for instruction. He must also bring to the task perseverance and a love of labor. Instruction in medicine is like the culture of the products of the earth. For our natural disposition is, as it were, the soil; the tenets of our teachers are, as it were, the seed; instruction in youth is like the planting of the seed in the ground at the proper season; diligent study is like the cultivation of the fields and it is *time* which imparts strength to all things and brings them to maturity. From this we shall see that Hippocrates clearly valued the early training on the part of our teachers as only the seed in the ground, which would spring from the soil following diligent study, but that it was time alone which would give rise to the true physician. Again he tells us that certain persons have the shape and dress and personal appearance of the actor, but are not actors, so also physicians are many in title, but few in reality. . . .

E. R. STITT, MD
(*NY State J Med* 1925; 25:650–653)

HIV antibody testing in a family planning clinic setting

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ABSTRACT. Self-reported risk factors and human immunodeficiency virus serostatus were determined for 433 women attending two family planning clinics in western Pennsylvania between April 1987 and April 1988. Only one of the 433 women had a positive ELISA test, which was negative by Western blot analysis. However, a substantial number reported high risk behaviors. These findings suggest that family planning clinics are important locations for conducting AIDS prevention education and for identifying women who are infected with HIV and in need of medical evaluation.

(NY State J Med 1988; 88:623–625)

Retrospective analysis of acquired immunodeficiency syndrome (AIDS) cases diagnosed in American women between 1981 and 1986 has shown that heterosexual contact with a partner at risk for AIDS has increased significantly as a risk category, and that in this country, a heterosexual woman is currently at greater risk of acquiring AIDS through sexual intercourse than is a heterosexual man.[1] For women who develop AIDS, the major "risk behavior" is intravenous drug use or sex with a partner who uses intravenous drugs.[1]

However, little is known about the seroprevalence rate of human immunodeficiency virus (HIV) in the female population. Screening of military recruits has revealed a crude HIV seroprevalence rate of 0.07% for female applicants.[2] While the overall prevalence of confirmed HIV seropositivity among Red Cross blood donors who have not been previously tested has averaged 0.04%,[3] female blood donors in Atlanta, Georgia, demonstrated a confirmed seroprevalence rate of 0.01%.[4] Recent studies of women seeking abortions in New York City,[5] and of parturients at an inner-city municipal hospital in New York City,[6] have documented HIV seroprevalence rates of 2.6% and 2%, respectively. While sobering, these rates are not entirely unexpected, given the prevalence of HIV infection among intravenous drug users in New York City.[7]

The current study was undertaken to estimate the prevalence of HIV infection and to determine the frequency of risk factors among a sample of regular users of two family planning clinics located in metropolitan Pittsburgh, an area with a relatively low incidence of AIDS.

METHODS

Data were collected from female clients of two clinic sites owned and operated by the Family Health Council, Inc. This nonprofit organization provides reproductive health services to approximately 72,000 women annually, through a series of 53 clinics operating in 23 counties of western Pennsylvania. This project was implemented at two clinic sites as a prelude to a more extensive program funded by the Pennsylvania State Department of Health.

Between April 1986 and April 1987, women who were scheduled for a routine appointment were given a flyer providing basic information about HIV transmission and inviting them to participate in a research project designed to obtain information about the prevalence of HIV infection among women from Pittsburgh. Individuals who were interested were given detailed information about the study and about the HIV antibody test by one of the two nurse-practitioners working within the clinic. Participation was not limited to individuals who had a historic risk factor for HIV infection. When a woman agreed to participate, she signed a consent form, completed a three-page questionnaire, and had 5 cc of whole blood drawn for subsequent HIV antibody testing. The questionnaire was self-administered and was reviewed by the clinic nurse on completion; it asked questions about age, race, current method of contraception, and behaviors within the past five years that might have placed the respondent at risk for HIV infection. HIV antibody testing was performed using the enzyme-linked immunosorbent assay (ELISA) procedure[8] by the Central Blood Bank of Pittsburgh, which performs a high volume of HIV testing on a daily basis. Specimens that were HIV seropositive by ELISA were subsequently confirmed by Western blot analysis.

RESULTS

Four hundred thirty-three women agreed to participate in the study, completed a questionnaire, and had HIV serologic testing performed. This represented approximately 20% of the women who were approached to participate. The women ranged in age from 18 to 45 years, although the majority (62%) were between the ages of 18 and 24 years, and the average age was 24 (Table I). Seventy-four percent of the sample was white, and 26% was nonwhite. Based on the percentage of individuals on medical assistance and the distribution of methods of birth control, this sample does not appear to differ greatly from the typical clientele at these two clinic sites.

Table II shows the risk factors reported by the participants. The majority of questions inquired about these factors within the context of the past five years; the question dealing with blood transfusion was posed for a ten-year frame of reference, and the question concerning sexual intercourse with an individual who had or went on to

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TABLE I. Selected Demographic Characteristics of the Participants (N = 433*)

Age	
18-24 years	268 (62%)
25-30 years	111 (26%)
31 or older	54 (12%)
Race	
White	320 (74%)
Nonwhite	113 (26%)
Current contraceptive method	
Oral contraceptives	284 (66%)
Condoms, or condoms and foam	67 (15%)
Diaphragm	18 (4%)
Intrauterine device	8 (2%)
Other	18 (4%)
None	36 (8%)
Percentage on medical assistance	89 (21%)

* Sums less than 433 are because of missing values.

TABLE III. White Compared to Nonwhite Participants

	White (N = 320)	Nonwhite (N = 113)
Average age (years)	24	24
Medical assistance (%)*	17%	32%
Sex with intravenous drug user	6%	4%
Sex with bisexual	3%	4%
Sex with African/Haitian	1%	3%
History of blood transfusion	2%	3%
History of prostitution	2%	3%
More than 26 sex partners in last five years	3%	2%
Current/past intravenous drug use	2%	0%
Past history sexually transmitted diseases		
Herpes	5%	4%
Gonorrhea*	3%	17%
Pelvic inflammatory disease*	3%	11%
Syphilis	0%	2%

* $P < 0.01$, chi-square test.

develop AIDS was not bracketed by a time frame. Many women reported more than one risk factor, although the data are not presented in this manner. The most frequently reported risk factor was a past history of sexually transmitted disease, with gonorrhea being the most commonly reported sexually transmitted infection. Only two of the respondents reported a past history of syphilis. Sexual intercourse with an intravenous drug user (5.5%) was the next most common risk factor, followed in frequency by a history of sexual intercourse with a bisexual male (3.5%). Three percent of the women reported 26 or more different sexual partners within the past five years, 2% had a history of blood transfusion within the past ten years, and 2% reported either a current or past history of intravenous drug use. Only 1% of the sample reported engaging in sex in exchange for gifts or money, and 1% reported a history of sexual relations with a man from a country where the rate of heterosexual transmission of HIV is high. None of the women in this sample reported sexual relations with a hemophiliac partner, an HIV antibody positive partner, a man with AIDS-related complex (ARC), or a man with AIDS. Of note is the fact that 9% of the women were not sure if a past sexual partner had been an intravenous drug user, and 11% were not sure if a past partner had been bisexual.

When the women were stratified on the basis of race (Table III), it was noted that nonwhite participants were similar to the white participants, except for a substantially higher percentage of nonwhite persons on medical assistance and a more frequent medical history of gonorrhea and pelvic inflammatory disease (PID) among that group.

Of the 433 women who agreed to participate in this project, only one had a positive ELISA test. When this sample was subjected to confirmatory testing using the Western blot analysis, the results were negative (ie, false positive).

DISCUSSION

The failure to detect any cases of HIV infection among this group of self-selected women attending two family planning clinics is not surprising, especially given the size of the sample. Also, the cumulative incidence of AIDS in Allegheny County (metropolitan Pittsburgh) is relatively low; approximately 150 cases had been diagnosed between 1981 and April 1987. Although the HIV seroprevalence rate among a large cohort of homosexual and bisexual men, recruited from the Pittsburgh area to participate in the MultiCenter AIDS Cohort Study, is approximately 20%,[9] rates of infection among heterosexuals in Allegheny county are substantially lower—consistent with the low rate of infection among intravenous drug users in this community. According to unpublished data from the Allegheny County Health Department, the seroprevalence rate of infection among intravenous drug users is less than 5%.

TABLE II. Self-reported "Risk Factors," in Decreasing Order of Rank*

	Yes	Not Sure	No
History of sexually transmitted diseases			
Gonorrhea, past five years	27 (6%)	3 (0.7%)	398 (93%)
Pelvic inflammatory disease, past five years	22 (5%)	10 (2%)	394 (92%)
Genital herpes, past five years	19 (4%)	5 (1%)	406 (94%)
Syphilis, past five years	2 (0.5%)	1 (0.2%)	423 (99%)
Sex with an intravenous drug user	24 (5.5%)	39 (9%)	369 (85%)
Sex with a bisexual man	15 (3.5%)	48 (11%)	369 (85%)
Sex with 26 or more partners in the last five years	12 (3%)	—	—
History of blood transfusion	10 (2%)	0 (0%)	420 (98%)
History of intravenous drug use, current or past	7 (2%)	0 (0%)	403 (98%)
History of sex in exchange for gifts or money	6 (1%)	0 (0%)	425 (99%)
Sex with a man from Haiti or Africa	6 (1%)	5 (1%)	420 (97%)
Sex with a hemophiliac	0 (0%)	16 (11%)	415 (96%)
Sex with a person with AIDS	0 (0%)	28 (6%)	405 (94%)
Sex with a person with ARC	0 (0%)	25 (6%)	408 (94%)
Sex with an HIV-positive partner	0 (0%)	42 (10%)	391 (90%)

* Sums less than 433 are because of missing values.

The seroprevalence rate of zero must be considered as a crude estimate since this was a self-selected sample, and more than half of the women who were approached to participate declined. It is possible that those who declined did so because they did not perceive themselves to be at risk for HIV infection, or did not want to participate in a research project. Perhaps it was because some were concerned that they may have already been infected with HIV and were unwilling to submit to testing. This remains supposition without additional information. Also, it is important to note that women were not recruited for this study on the basis of their past behaviors, but volunteered this information after they had consented to participate in the study. Therefore, the frequency of self-reported risk behaviors among this sample may not represent the true frequency among the total population of women who attend these two clinics.

The utility of this survey is not so much in its failure to document infection among the women tested as it is in demonstrating the significant proportion of women who report behaviors that could place them at risk for HIV infection, especially sexual intercourse with partners who use intravenous drugs, and a past or current history of intravenous drug use. Although there are those who view heterosexual intercourse, per se, as a risk factor for HIV infection, a history of past or current sexual intercourse with a partner who uses intravenous drugs should be considered an even greater risk for HIV infection. This survey documents that family planning organizations do provide services to women who have used intravenous drugs themselves, and who report sexual intercourse with partners who use intravenous drugs. Also, because of their ability to reach the female partners of male intravenous drug users (a notoriously hard to identify at-risk population), family planning organizations offer an important means of interrupting the transmission of HIV from infected male drug users to their female partners. Because the vast majority of their clients are of reproductive age, family planning organizations can also play an important role in the early identification of pregnancy among women who are at increased risk for HIV infection.

Although the HIV testing we performed was initiated for the purpose of research, it was preceded by adequate counseling, provided by clinical staff who were specifically trained to explain the test to our volunteers, to provide them with basic information about preventing HIV infection, and to answer their questions about AIDS. Therefore, this study also demonstrates the feasibility of integrating AIDS prevention education into the nationwide system of family planning providers.[10]

Nationwide, these agencies routinely provide care for millions of sexually active women each year, and based on the results of this survey, it is likely that other urban agencies are seeing clients who are at risk for infection on the basis of intravenous drug use or sexual intercourse with men who are themselves at risk for HIV infection. It is also likely that similar programs conducted in areas of the country where the prevalence of HIV infection among intravenous drug users is higher[11] would identify women who are already infected with HIV. These women could then be referred for medical evaluation, including a determination of the status of their cellular immune systems, especially since decreased T helper cell counts have been shown to be significant predictors of progression to AIDS among HIV seropositive persons.[9]

Finally, it should be noted that the use of the term "risk factor" is relative in regard to HIV infection, since there is no sexual or needle sharing activity that is capable of resulting in infection if both partners are uninfected. Other variables may also modulate the risk of infection in a susceptible individual who is exposed (through sexual intercourse or needle sharing) to an infected partner. Obviously, historic factors such as prior sexually transmitted disease do not in themselves predispose to HIV infection, but may serve as surrogate markers for sexual activity with many different partners.

As this survey documents, the predictive value of these risk factors for heterosexual women depends on the prevalence of infection within the community, especially among intravenous drug users. Referring women for counseling and testing based on the presence of a risk factor is unlikely to result in a high yield of infected cases in communities in which the level of HIV infection among intravenous drug users is low. However, it is important to stress that all sexually active women should receive specific information about HIV prevention, regardless of the presence of risk factors in their clinical history.

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Personality factors that differentiate homosexual men with positive and negative attitudes toward condom use

MICHAEL W. ROSS, PHD

ABSTRACT. **The Homosexual Attitudes Toward Condoms scale was administered to 148 homosexually active men along with two personality and mood inventories, the Adjective Check List (ACL) and the Profile of Mood States (POMS). For each of the five subscales of the Homosexual Attitudes Toward Condoms scale and for the total scale, groups were split at the median score and compared by t-tests on the scales of the ACL and POMS. Results showed that high scorers on the ACL scales for dominance and aggression, and less frequently for autonomy, exhibition, and self-confidence, had consistently positive attitudes toward condoms; and that high scorers on the ACL scales for abasement, deference, and the POMS scale for tension-anxiety had consistently negative attitudes toward condoms. These data suggest that the most important variables associated with positive attitudes toward condom use are an assertive personality style and the ability to raise the issue of condom use in sexual encounters without fear of rebuff. Future educative efforts should be directed toward assertiveness training in sexual encounters rather than information on the efficacy of condoms for prevention of HIV transmission.**

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In the absence of a vaccine or cure for human immunodeficiency virus (HIV) infection, preventive measures are the only effective means of reducing viral spread. Among preventive measures recommended is the use of condoms to prevent transfer of infected secretions during sexual contact. While condoms have been used for some time in heterosexual intercourse as a contraceptive and as a prophylactic,[1] they were rarely used by homosexual men until the advent of acquired immunodeficiency syndrome (AIDS). While problems with condom use do occur among homosexual men,[2] evidence suggests that about one third of nonmonogamous homosexual men use condoms consistently, another third use them occasionally, and the remainder use them not at all.[3-5]

Clearly, increased use of condoms by homosexual men at risk of HIV infection is important if some degree of control of the epidemic is to be achieved. Such increased use will depend, in part, on changing attitudes toward condoms among homosexual men. Recent research has identified the major dimensions of attitudes toward condom use among homosexual men. Brown's[7] attitudes toward condoms scale has been adapted[6] for homosexual men. It was found that there are five dimensions of homosexual men's attitudes toward condom use. These dimensions are: seeing condoms as unreliable and unerotic; seeing condoms as not providing protection from infection; seeing condoms as unavailable when needed; seeing condoms as interrupting sex; and seeing no need to assume responsibility for partners' safety and comfort with condom use.

There is no information to date on the characteristics of homosexual men who have positive or negative attitudes toward condom use. Such information is necessary if we are to successfully attempt to increase the use of condoms among homosexual men at risk of HIV infection. This study reports on the personality characteristics that differentiate homosexual men with positive and negative attitudes toward condom use as measured by the Homosexual Attitudes Toward Condoms scale.[6]

METHODS

The 46-item Homosexual Attitudes Toward Condoms scale[6] was administered to 148 homosexually active men. Subjects were recruited in late 1986 from among homosexual men undergoing testing for sexually transmitted diseases and HIV; and those responding to advertisements placed in local gay magazines and announcements made at meetings of local gay organizations. Mean age (± standard deviation) was 32.8 ± 6.3 years (median age, 32 yrs; range, 16 to 60 yrs). The scale was recoded so that all the items were scored in a negative direction (high scores indicating negative attitudes toward condoms for all items) and the five subscales were scored as described elsewhere.[6]

In addition, the Adjective Check List (ACL) of Gough and Heilbrun[8] and the Profile of Mood States of McNair et al[9] were administered and scored according to the standard method described in the respective test manuals. The five subscales of the Homosexual Attitudes Toward Condoms scale were split at the median and two-tailed t-tests computed between those respondents who scored high and those who scored low on the five subscales (and the total scale score) for the scales of the ACL and POMS.

RESULTS

Results are presented in Table I (mean ± SD given). A number of differences were significant beyond the 5% level for each of the five subscales and for the total scale. These revealed consistent patterns across the subscales, with the same personality and mood state vari-

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TABLE I. Variables Differentiating High and Low Scorers on Homosexual Attitudes Toward Condoms Scale

Variable	Low	High
Subscale 1: Condoms as Unreliable and Unerotic		
Adjective Check List		
Aggression	−2.5 ± 4.6	−4.5 ± 4.8*
Profile of Mood States		
Tension-Anxiety	9.6 ± 6.9	13.1 ± 8.1*
Subscale 2: Rejection of Condoms as Protection from Infection		
Adjective Check List		
Unfavorable items checked	9.1 ± 9.4	14.7 ± 14.8*
Dominance	5.0 ± 5.8	1.4 ± 5.5†
Exhibition	2.1 ± 5.0	0.0 ± 4.8*
Abasement	1.1 ± 4.7	3.6 ± 4.8*
Counseling readiness	−7.2 ± 5.2	−4.5 ± 6.0*
Self-confidence	8.9 ± 5.2	5.9 ± 5.7*
Ideal self	6.2 ± 6.3	3.3 ± 7.0*
Military leadership	8.9 ± 5.4	6.7 ± 6.1*
Profile of Mood States		
Tension-Anxiety	9.7 ± 6.7	13.3 ± 8.5*
Fatigue-Inertia	7.5 ± 5.4	10.1 ± 7.4*
Confusion-Bewilderment	6.6 ± 5.2	8.8 ± 6.3*
Total mood disturbance	55.4 ± 32.9	71.4 ± 42.9*
Subscale 3: Unavailability		
Adjective Check List		
Unfavorable items checked	9.5 ± 8.7	14.5 ± 15.7*
Dominance	5.1 ± 5.4	1.1 ± 5.7†
Exhibition	2.5 ± 4.8	−0.2 ± 4.7†
Aggression	−2.0 ± 4.3	−4.8 ± 4.5†
Succorance	0.3 ± 2.9	2.8 ± 4.0†
Abasement	0.7 ± 4.5	4.1 ± 4.8†
Deference	0.8 ± 4.0	2.9 ± 5.3*
Counseling readiness	−7.4 ± 4.4	−4.4 ± 6.6*
Self-control	−0.9 ± 3.3	0.9 ± 3.7*
Self-confidence	8.9 ± 5.2	6.0 ± 5.9*
Profile of Mood States		
Tension-Anxiety	9.6 ± 7.6	13.8 ± 7.9*
Subscale 4: Interruption of Sex		
Adjective Check List		
Favorable items checked	51.1 ± 14.8	44.3 ± 18.7*
Communality	12.3 ± 3.2	10.3 ± 4.9*
Achievement	11.3 ± 6.3	8.8 ± 6.2*
Dominance	4.3 ± 6.2	1.8 ± 5.2*
Autonomy	3.8 ± 4.5	0.9 ± 4.9†
Change	4.9 ± 3.5	3.1 ± 3.5*
Deference	0.6 ± 4.4	3.4 ± 4.9†
Ideal self	6.0 ± 6.7	3.2 ± 6.4*
Creative personality	4.6 ± 3.8	2.8 ± 3.9*
Subscale 5: Absence of Responsibility and Comfort with Condom Use		
Adjective Check List		
Nurturance	10.8 ± 6.5	14.1 ± 8.0*
Heterosexuality	7.7 ± 4.5	9.5 ± 4.4*
Autonomy	3.5 ± 5.2	1.6 ± 4.2*
Aggression	−2.1 ± 4.4	−4.8 ± 4.5†
Deference	0.3 ± 4.8	3.4 ± 4.4†
Personal adjustment	6.6 ± 3.8	8.6 ± 5.1*
Total Scale Score: Negative Attitudes Toward Condom Use		
Adjective Check List		
Dominance	4.4 ± 6.1	2.0 ± 5.6*
Aggression	−2.1 ± 4.6	−4.7 ± 4.6†
Abasement	1.2 ± 5.2	3.6 ± 4.4*
Deference	0.5 ± 4.4	3.2 ± 5.2†
Profile of Mood States		
Tension-Anxiety	9.3 ± 7.2	13.5 ± 7.6*

* $P < 0.05$.
† $P < 0.01$.

ables recurring among those variables which differentiated the high and low scorers. Those most consistently occurring are illustrated in Table I as the variables differentiating respondents with high and low scores on the total scale score of Negative Attitudes Toward Condom Use.

Variables which characterized those with most positive attitudes toward condoms (low scorers) included high scores on the ACL scales for dominance (described as strong-willed, ambitious, determined, and forceful individuals); and aggression (described as competitive and aggressive individuals who view others as rivals to be vanquished).

Variables that characterized those with the most negative attitudes toward condoms (high scorers) included high scorers on the ACL scales for abasement (described as asking for little, submitting to the wishes and demands of others, and avoiding conflict at all costs) and deference (described as conscientious, unassuming, and patient); and on the POMS scale for tension-anxiety (described as a generalized state of discomfort).

Other ACL variables significantly associated with at least two subscales which characterized those with positive attitudes toward condoms included high scorers on exhibition (described as forceful, intrusive, and even bombastic); autonomy (described as independent, assertive, and self-willed); self-confidence (described as confident of ability to initiate and achieve goals); and ideal self (described as being characterized by interpersonal effectiveness and goal-attaining abilities). A further variable characterizing those with negative attitudes toward condoms was the number of unfavorable ACL items checked, with high scorers being characterized as disbelieving, pessimistic about the future, and changeable.

DISCUSSION

These results are remarkably consistent. Without exception, those homosexual men who have positive attitudes toward condoms score highly on the ACL and POMS scales which describe individuals as being assertive, instrumental, and determined. In contrast, those who have negative attitudes toward condoms are consistently described as submissive, passive, and conflict-avoiding.

Good scale validity has been reported,[6] with significant relationships between having positive attitudes toward condoms (as measured by the Homosexual Attitudes Toward Condoms scale) and actual use of condoms. These data consistently suggest that more positive attitudes toward condoms, which are themselves associated with a significantly increased use of condoms for both oral and, to a lesser extent, anal sex,[6] are associated with a greater degree of assertiveness. It appears that personality variables associated with the ability to raise the issue of condom use in a sexual encounter without fearing rebuff is the single most significant variable associated with positive attitudes toward their use. This is true even though health-related attitudes are often not associated with actual behaviors. It is probable that positive attitudes toward condom use in this population are thus associated with sufficient strength of personality and social skills to raise the subject prior to or during sexual encounters, and the consequent higher rate of condom use.

These data are of considerable significance for attempts to increase condom use among homosexual men at risk of HIV infection. They suggest that increasing the availability of information alone will not increase the frequency of condom use. In a study by Ross and Herbert,[3] it was noted that while over 96% of the sample knew why condoms should be used, only 28% used them consistently. A change of preventive education towards assertiveness and social skills training to enable individuals to insist on condom use is probably extremely important to increase the rate. Where there is knowledge of the risks of failure to use condoms, the inability to put that knowledge into effect makes it useless in reducing the spread of HIV infection.

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Cremation patterns for patients dying of AIDS in New York City

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ABSTRACT. This paper traces the historical development of cremation. In this century, cremation has increased due to a number of factors, such as changes in religious attitudes toward cremation, concerns about funeral costs, and public health concerns. Recently, it has been argued that persons with HIV infection and AIDS face discrimination while alive and also after death. This paper analyzes major causes of death in New York City for the period 1982–1986 for persons aged 25–44 years old. The major causes of death are categorized into major infectious and noninfectious causes to determine if there is a difference in the cremation rate. We present evidence that persons dying of infectious diseases have higher cremation rates than those dying of noninfectious diseases. This pattern is especially true for persons dying of AIDS.

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At the turn of the century, less than 1% of all persons who died were cremated in the United States.[1] By 1986, about 14.3% of the 2.1 million who died were cremated.[2] There are several possible explanations for this increase in the cremation rate over the past 80 years. These include an increase in personal or religious tolerance toward cremation and concerns about cemetery space, sanitation or public health, and the costs of conventional funerals. The last three considerations were the major points used in the crusade of the "Cremation Movement" at the turn of the century.[1,3]

In the late 19th century, Sir Henry Thompson, an English physician, and others proposed that incineration of the dead was an efficient solution to the graveyard problem plaguing Europe.[4] At that time, graveyards in many locations were overcrowded and were sources of contagion to the living. For example, in England, one such graveyard that was an acre in size had had more than 80,000 burials in a period of 50 years.[5]

In the years that followed, physicians throughout Europe and the United States argued for cremation for public health and economic reasons. In 1907, Knopf[6] argued that cremation provided a more reliable manner of destroying contagious pathogens. In addition, he felt that the cremation of persons who died of tuberculosis and other infectious diseases would be an economically sound way to relieve the contagious disease problem among the poorer populations of New York City. However, in the same year, Harrington[7] was convinced that cremating the bodies of patients with infectious diseases was ill advised, for it would drive people to hide their illnesses to prevent being cremated.

Although cremation gained support among academics and public health officials, public support was not quick to follow. While many Protestant leaders even came forward to support cremation as an ethical alternative to burial, the public's view on death seems to have been grounded in more traditional thinking.[3]

By the middle of the 20th century, with major reductions in the incidence of infectious diseases, concerns about contagion from the disposal of the dead had declined. At the same time, there was an increase in cremations. This increase was correlated with a number of changes going on in society that favored cremation: increased standard of living, increased levels of education, and increased tolerance.[8] Most of the increase in cremations in this century occurred in the last few decades. From 1966 to 1986, the cremation rate for all deaths increased from 3.76% to 14.32%.[9]

In 1986, there were about 879 crematories in the United States, with approximately 10% of them located in

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New Jersey, New York, and Pennsylvania.[2] In 1986, the crude cremation rate was 12.0% for that region.[2] The highest crude cremation rate (36.6%) was reported for California, Oregon, and Washington,[2] which also had the most crematories; the lowest rates were reported in the South.[2] These regional differences may be explained largely by a number of underlying cultural factors.

While studies have examined death and funeral practices, little is known about cremation practices. One study that focused specifically on cremation practices in a white population was done in 1985.[8] This study found that the type of person whose body is cremated tends to be better educated, more likely to be a professional, and less likely to be religious. Protestants had higher cremation rates than any other group. These findings were similar to those of another study done almost 25 years ago.[10]

The decision to cremate is a personal one that is influenced by personal attitudes, traditions, and the influence of people close to the dying person. In making final arrangements, cremation is usually decided on jointly by the individual and the funeral director before death, or by the family (or proxy) and the funeral director after death. While the wishes of the deceased are usually taken into account, the family (or proxy) has great influence over the final decision to cremate, especially in situations of unexpected or early death. Other important mediators include clergy and funeral personnel. The influence of these persons may often go unnoticed; Mitford[11] has cited instances in which funeral directors may subtly influence the decision making process (eg, the selection of a more expensive casket over a cheaper alternative).[11]

Since the acquired immunodeficiency syndrome (AIDS) epidemic began in the late 1970s, numerous reports have discussed the medical obligation to treat those infected with the AIDS virus. To date there are no reports of the AIDS virus having been transmitted to workers through funeral preparation procedures including embalming. Nonetheless, according to several accounts, AIDS patients are regularly discriminated against by funeral homes that refuse to provide service or that overcharge families of AIDS patients[12,13] (K. Glidden, unpublished data, Gay Men's Health Crisis, New York, 1987).

The purpose of this paper is to determine whether persons with infectious diseases, and in particular AIDS, are cremated more frequently than persons with noninfectious conditions.

METHODS

New York City is one of the few cities that maintains its own registry of vital statistics. Underlying causes of death listed on death certificates are coded according to ICD-9-CM codes. Using death certificates, a cross-sectional study was performed to explore the association between cause of death and cremation outcome. The cause of death was taken to be the independent variable with cremation outcome being the dependent variable. All available death certificates were initially included to minimize sampling bias. All analyses reported herein are restricted to those deaths that occurred in New York City among persons aged 25–44 during the years 1982 to 1986, the most recent five-year period for which data are complete.

Death certificate burial codes were compared with cremation codes. In this study, the proportion cremated (cremation rate) was defined as the number of cremations divided by the total number of deaths. Because of the legal requirement to report deaths and to dispose of the dead almost immediately,[14] assessment of cremation patterns can use cumulative incidence measures, rather than the less desirable prevalence measures.

Seven categories were created for disease status; this categorization includes most of the major causes of death for 25–44-year-olds.[15] These categories, with their corresponding ICD-9-CM codes in brackets, are as follows: acquired immunodeficiency syndrome (AIDS) [136.3, 173.9, 279.1]; cirrhosis of the liver [571]; drug (narcotism) dependence [304]; influenza and pneumonia [480-487]; total tuberculosis (TB) [010-018]; violent deaths [800-949, 950-959, 960-975, 990-999]; and other conditions [all other ICD-9-CM codes].

Though total tuberculosis is not a leading cause of death for this age group, patients dying of this illness often come from lower socioeconomic groups and will serve as proxy for this group. Also, it is not grouped with AIDS since it is not possible (at this writing) to determine which tuberculosis cases actually would be reclassified as AIDS cases.[16] The category of "violent deaths" is a composite group of deaths due to accident, suicide, and homicide. The "other conditions" category is composed primarily of chronic diseases (eg, neoplasms, heart disease, cerebrovascular diseases, and diabetes).

Some variables that may be associated with cremation are not coded for on the death certificate or are not coded in a useful manner. For instance, education and usual occupation could not be assessed, since the computerized categories were too limited to allow meaningful analysis. Personal preferences, family traditions, and religious beliefs cannot be analyzed from death certificates. Nevertheless, correlated with these personal preference variables are ethnic and socioeconomic differences. These factors are related to cause of death.[17] It has been shown that 25–44-year-olds have excess morbidity and mortality due to causes of death that typically do not affect an older, wealthier population. This may be due in part to lack of access to quality health care and to lack of preventive health behaviors for those in this age group who do die. The possible exception to this observation may be AIDS, which affects persons in all socioeconomic strata. It is assumed that some ethnic differences can be controlled for by stratification by race, which does appear on the death certificate. Data were also stratified by location at time of death, race, and sex. No other demographic variables available could be used to enhance this study.

Proprietary and voluntary hospitals were combined to form the "private hospitals" category, and municipal hospitals formed a second category of "public hospitals" for location at time of death. It is assumed that private hospitals tend to cater to a wealthier, better insured population than public hospitals, even though poorer people may enter private hospitals through various routes. State and federal hospitals formed a third group; they typically cater to special beneficiaries and to patients in need of long term psychiatric and chronic care. The fourth category, "other sites," included persons who died at home, in hospices, in nursing homes, or in other locations.

Data on race were categorized into three groups: whites; nonwhites, which included blacks and Hispanics; and the "other racial" group, which included Asians, Native Americans, and persons whose race was unknown.

A comparison of demographic variables in the sample and in the population from which the sample arose was done without any statistical analysis. A chi-square test for comparison of m proportions was used to determine if the proportion cremated [P(Crem)] for each cause of death differed significantly over the time period studied.[18] Each year was assumed to be an independent sample. Values were considered significant at alpha = 0.05.

Using the "other conditions" category as the standard group for each racial/location of death stratum, rate ratios were calculated. Due to the small number of persons who had died and who fell in the stratas for "other racial" and "state and federal hospitals," these groups were excluded from the rate ratio analyses in this paper. These rate ratios were internally standardized and were considered similar to standardized mortality ratios (SMRs)[19]; therefore, across-stratum comparisons are not done,

TABLE I. Comparison of Characteristics of Persons Dying in 25–44-Year-Old Cohort and in the General Population, 1982–1986

Characteristic	Sample (N = 32,019)	General Population (N = 362,053)
% Cremated	12.7	9.7
% Female	28.6	48.1
% Black and Hispanic	60.0	29.2
% Private hospitals	39.6	51.9
% Public hospitals	25.4	14.4
% Other sites	33.6	33.7

as each racial/location of death stratum has a different standard. Since the ratio uses a noninfectious condition as a standard, values equal to or less than one correspond to a similar (or less frequent) cremation rate as the standard noninfectious condition. Values greater than one represent cremation rates greater than the standard condition. Mantel-Haenszel[20] rate ratios were used to pool data across sex strata. Subsequent test-based limits were calculated at the 95% confidence level.[19]

RESULTS

The study group of 25–44-year-olds differed from the general population of New York City (Table I). More persons in the age group studied were cremated (12.7%) than in the general population (9.7%). This sample also had twice as many nonwhites (60% vs 29%) and fewer females (29% vs 48%) than the general population. People in the sample also died more frequently in public hospitals (25% vs 14%), whereas the general population tended to die more frequently in private hospitals (40% vs 52%). The two groups did not differ in terms of other locations of death. The percentage that died in state and federal institutions was less than 2% in both groups; thus, the decision to exclude this location of death group was valid.

The five-year trends for most of the seven different causes of death investigated showed stable rates with small secular changes (Table II). The categories that did show significant changes in cremation over the five-year period were AIDS, tuberculosis, violent deaths, and the other conditions category. For AIDS, violent deaths, and the other conditions category, the rates have increased during this time period. This was especially noted for the other conditions category, which forms the denominator of the rate ratios. This significance is due to the magnitude associated with even small fluctuations in a group with such large size. For AIDS, there was an almost two-fold jump in the cremation rate from 1982 to 1983, at which point the rate became stable. In any given year, AIDS patients were cremated twice as often as patients who died of other diseases, including both infectious and noninfectious conditions. The only exception to the stability previously noted was in the tuberculosis rates. The small number of deaths due to this entity resulted in unstable rates. This affected later point estimates and confidence intervals for this cause of death.

Table III shows cause of death-specific cremation rate ratios and the associated confidence intervals for site of death and racial type.

TABLE III. Cause of Death-Specific Cremation Rate Ratios By Site and Race, 1982–1986

	Nonwhite		White	
	RR_{mh}	(CI)*	RR_{mh}	(CI)
Public hospitals				
AIDS	2.59	(2.14, 3.14)	2.76	(2.08, 3.67)
Cirrhosis	0.98	(0.69, 1.38)	1.33	(0.86, 2.05)
Drug dependence	1.35	(0.98, 1.86)	0.93	(0.49, 1.76)
Influenza/pneumonia	1.65	(1.22, 2.23)	1.45	(1.14, 2.40)
Total tuberculosis	1.41	(0.42, 4.75)	—	
Violent deaths	0.61	(0.17, 2.14)	0.85	(0.28, 2.61)
Private hospitals				
AIDS	3.09	(2.61, 3.65)	3.13	(2.75, 3.56)
Cirrhosis	0.86	(0.62, 1.12)	1.88	(1.41, 2.51)
Drug dependence	1.02	(0.72, 1.44)	1.25	(0.75, 2.08)
Influenza/pneumonia	1.22	(0.86, 1.74)	1.30	(0.92, 1.85)
Total tuberculosis	1.61	(0.93, 2.78)	—	
Violent deaths	0.81	(0.61, 1.08)	0.71	(0.51, 0.98)
Other sites				
AIDS	4.30	(3.28, 5.63)	3.58	(3.02, 4.24)
Cirrhosis	1.09	(0.82, 1.44)	1.06	(0.82, 1.35)
Drug dependence	1.07	(0.82, 1.39)	0.98	(0.77, 1.24)
Influenza/pneumonia	1.54	(1.21, 1.95)	0.77	(0.40, 1.48)
Total tuberculosis	—		2.47	(0.84, 7.24)
Violent deaths	0.92	(0.75, 1.13)	1.10	(0.94, 1.29)

* RR_{mh} (CI) = rate ratios (confidence interval).

Based on the point estimates, both whites and nonwhites who died of infectious causes in public hospitals appeared to be cremated more frequently than persons who died of noninfectious causes. Point estimates for persons dying of AIDS and influenza/pneumonia are significantly different from 1.0. While the point estimate for tuberculosis for nonwhites supports this association, the confidence interval is large enough to contain one. The tuberculosis-related values for whites could not be calculated, since there were no tuberculosis deaths in this group.

Although not as strong as the association seen in public hospitals, the point estimates for nonwhites dying in private hospitals suggest a similar pattern between cause of death and cremation outcome. Furthermore, more persons dying of AIDS appear to be cremated than those dying of noninfectious causes. For whites dying in private hospitals, no association could be found between cause of death and cremation outcome. Two causes of death were significantly different from the noninfectious standard: cirrhosis and AIDS. Two other causes also had point estimates greater than 1.0 but had confidence intervals that contained the null value: drug dependence and influenza/pneumonia.

For nonwhites dying in other sites, the point estimates and confidence intervals for two of the three infectious causes of death are significantly different from the noninfectious standard. Since the point estimate of the rate ratio and the test-based confidence intervals could not be calculated for the tuberculosis-related strata, no comparison could be made for this disease. However, for non-hospi-

TABLE II. Cremation Trends by Cause of Death, 1982–1986

Condition	1982 % Crem	1982 Deaths	1983 % Crem	1983 Deaths	1984 % Crem	1984 Deaths	1985 % Crem	1985 Deaths	1986 % Crem	1986 Deaths	Chi Square
AIDS	15.9	44	25.1	358	27.1	749	30.0	1,311	30.4	2,021	10.09*
Cirrhosis	9.0	580	9.0	524	10.5	580	11.3	630	11.2	534	2.89
Drug dependence	8.9	459	10.1	475	10.1	407	11.6	545	11.8	604	3.06
Influenza/pneumonia	13.7	146	8.2	194	11.2	303	12.4	338	13.9	352	4.46
Total tuberculosis	0.0	47	13.3	45	4.8	62	16.9	59	17.3	75	13.52*
Violent deaths	8.1	1,517	7.8	1,381	9.4	1,286	11.1	1,243	10.1	1,391	12.22*
Other conditions	9.5	2,488	10.5	2,630	11.9	2,766	13.1	2,837	12.5	3,038	22.67*

* Significant at $p = 0.05$ or less.

tal locations of death, most of the confidence intervals for whites contained 1.0, and hence were not different from cremation due to the noninfectious standard. No conclusions can be made regarding the association between cause of death and cremation outcome for whites dying in other sites.

DISCUSSION

This study demonstrates that black and Hispanic persons are cremated more frequently if they have died of infectious, rather than noninfectious causes. Whites who die in public hospitals and other sites also exhibit a similar pattern, although this is not the case for whites who die in private hospitals. Patients who die of AIDS are cremated more frequently regardless of race or location of death.

The major limitation with using death certificates in this study is the inflexibility of these records. Limited information is available, and much of this information is not highly specific. Hence, it is not wise to attribute all of these differences in cremation to cause of death. The major findings focus on minorities. Nevertheless, no previous data exist on cremation patterns among blacks and Hispanics. Furthermore, while controlling for location of death and race may reflect some educational and socioeconomic differences, conclusions based on these findings are prone to fallacy. Specifically, education and related factors were not directly controlled. The significant associations seen with AIDS may be due to these or other factors; this also applies to the other conditions with even weaker associations.

It is impossible to determine from these data whether the families and friends of AIDS patients are seeking cremation as an alternative to burial or are being strongly urged to accept cremation as an alternative. In the past, funeral directors have refused to care for the dead with contagious diseases. With greater public tolerance for cremation, funeral directors might influence the decision of the bereaved to opt for cremation. Others have suggested that there may be discrimination against HIV-infected patients even after death (K. Glidden, unpublished data, Gay Men's Health Crisis, 1987),[21,22] although the evidence has been difficult to analyze systematically.

The risk of infection from HIV and other infectious agents to funeral directors should be similar to that of pathologists doing autopsies. It has been documented that transmission of HIV occurs infrequently under conditions such as those experienced by funeral directors.[23] Infection control guidelines for performing autopsies have been created to minimize the risk of HIV transmission.[24] A certain amount of risk, though, is expected among pathologists who do autopsies. This is true for other areas of the medical profession as well.[25] Refusing to care for infected patients is currently considered professionally unacceptable by the medical profession.[25-27] Still, there are many gray areas. For instance, physicians are currently debating whether it is professionally acceptable to refuse unnecessary medical treatment.[28] Ethical standards concerning refusal to care for the deceased are less debatable. Funeral directors have a duty to care for these dead as for any other dead. Also, while there is elective surgery in medicine, there are no unnecessary funerals.

It is important to understand how funeral directors are dealing with HIV infection so that health education efforts can be targeted appropriately. In reviewing the literature, no efforts have been made to understand the extent of AIDS knowledge and fear of AIDS among funeral directors. Fear of AIDS is a problem even among health care professionals,[29] although one study has shown that it does not affect care.[30] Currently, studies are being done to assess the effects of caring for patients with HIV infection on health care professionals in hospitals.

The stigmatization of AIDS,[31] coupled with this fear among health care professionals, is a potent source of discrimination. Future research must focus on funeral directors, since they are an important part of the health care system and are subject to the same prejudice and ignorance that afflict other groups. One of the problems that exists in New York City is that there are a limited number of funeral homes that will care for HIV-infected patients.[14] Further, an informal study has shown that funeral homes often provide families of AIDS patients with misleading or incorrect information. Similar to the problem of abandonment by physicians, this forces a few funeral directors to handle the bulk of deaths from this epidemic. Just as such behavior cannot be tolerated from physicians, it cannot be tolerated from funeral directors.

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An AIDS education program for Vietnamese women

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ABSTRACT. The purpose of this study was to determine the effect of an AIDS education program for Vietnamese women on the knowledge, attitudes, and practices of the participants. The study used a variation on the nonequivalent control group design. The sample (N = 369) consisted of Vietnamese clients who attended the Public Health Foundation Women, Infants, and Children (WIC) program in Los Angeles. Knowledge, attitudes, and practices were measured using a structured pretest and post-test questionnaire. The educational intervention consisted of a didactic slide-tape program delivered by a Vietnamese nurse educator and accompanied by informational and resource brochures. Data were analyzed using the Statistical Package for the Social Sciences. Differences between experimental and control groups on individual items on the questionnaire, and differences in pretest/post-test scores for the experimental group were examined by way of two-tailed t-tests. The Pearson product moment correlation coefficient (r) and chi-square analysis were used to examine the relationship between sociodemographic variables and individual knowledge, attitudes, and practice items. Control and experimental groups did not differ significantly. Significant differences occurred in the experimental group in pretest/post-test items measuring knowledge, attitudes, and practices. Significant gains occurred in knowledge and positive changes occurred in attitudes and intended practices. The most dramatic changes occurred in attitudes and intended changes in practice. The findings demonstrate that a didactic program can result in changes in knowledge levels and also in attitudes and intended behavior.

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Since the collapse of the South Vietnam regime in 1975, more than 860,000 Southeast Asian refugees have been resettled in the United States.[1] Of these, by far the largest group (40%) lives in California. The number of refugees in California continues to grow because of new arrivals and an internal migration of Southeast Asians within the United States from small towns where they were originally resettled to large metropolitan areas.[2] Los Angeles County is one of the metropolitan areas in which refugees were originally resettled and to which they continue to migrate and to immigrate.

The countries from which the immigrants come do not have high rates of acquired immunodeficiency syndrome (AIDS). However, World Health Organization officials consider the spread of AIDS a threat in what they call the "sentinel" countries of Asia, such as the Philippines, Sri Lanka, and Thailand. These countries all have large and well developed male and female prostitution industries that cater to travelers.[3,4]

Los Angeles has the third largest number of cases of AIDS in the United States, consistently around 8–9% of the total AIDS cases since 1981.[5] Reported cases of AIDS among Asians, however, are extremely low. The Centers for Disease Control (CDC) reports cases of AIDS among Asians under the category of "unknown/other." This category has accounted for only 1% of total cases. In Los Angeles, this category has accounted for 2% of all cases.[6] New York City keeps records linking its AIDS surveillance registry to death certificates. Mortality rates are categorized by age, sex, and ethnicity (black, Hispanic, Asian, and white). Asians had the lowest risk of AIDS mortality, with a relative risk compared with whites of 0.43. No deaths from AIDS had occurred among Asian women.[7]

Despite the extremely low risk of AIDS in the Asian population, the California Department of Health Services Office of AIDS funded projects to reach minority populations, including Southeast Asians, with educational and prevention programs on AIDS. This paper reports the process and outcome of an AIDS education program for Vietnamese women in Los Angeles County.

Program Design and Vietnamese Culture. A decision was made at the outset to focus the AIDS education program on only one ethnic group among the Southeast Asian refugees. Differences in interest, language, experiences, and education make it difficult to design and implement multiethnic programs. The Vietnamese were chosen for several reasons: They make up the largest percentage of Southeast Asian refugees (62.5%); they are generally more urban in background, more educated, and quicker to

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adapt to Western life than other Southeast Asian groups[8]; the Vietnamese have a large and well developed system of mutual assistance associations in California[2]; and the investigators had close working relationships with Vietnamese nurses who could act as consultants to the project and educators in the program. It was thought that all of these characteristics would facilitate an AIDS education prevention program among Vietnamese refugees. An ethnic-specific program was considered the approach of choice within the constraints of funding, population patterns, and cultural differences.

Certain aspects of Vietnamese culture had an impact on development of the program, and these deserve attention and explanation. The first of these is the traditional support systems of the refugees. The Vietnamese come from backgrounds in which the family provides a comprehensive support system, with many persons living in extended family situations. Many refugees have not only lost their network of extended families but have also lost their network of former friends. Therefore, most Southeast Asian groups in cities where the refugee population density is high have formed mutual assistance associations.[2,8] These associations are reflections of the community's effort to provide substitutes for former social networks and support systems. In California, in 1984, there were 206 of these associations, 141 of which were Vietnamese.[2] The mutual assistance associations appear to be one of the best vehicles through which to launch a prevention effort. They provide network services linking, referring, and directing refugees to community service centers.

In Los Angeles, a community-based service center which provides a wide range of services for Southeast Asians is the Indochinese Refugee Service Center (IRSC). Among the programs housed in the IRSC are the Public Health Foundation Nutrition Program for Women, Infants, and Children (WIC); the Asian Women's Health Project; the Southeast Asian Mental Health Training Project; and the county Indochinese units of the health services and mental health departments. Other county services include legal assistance, economic assistance, social services, and English as a Second Language (ESL) classes.[9] All mutual assistance associations in Los Angeles in proximity to the IRSC refer refugees to this center for services and programs.

The Women, Infants, and Children (WIC) program in the IRSC was chosen as the vehicle for an AIDS educational program because of its natural fit with educational programs and its access to large numbers of program participants. Clients come to the WIC program on a monthly basis for a counseling and educational program and nutritional supplements. Clients are usually women (pregnant and nursing mothers) and their high-risk infants and children. A woman-to-woman AIDS education program was decided on because of its potential for cultural acceptability and its potential impact on a wider range of persons than just the participants themselves. It has been recommended that workshops for Southeast Asian women be presented whenever possible by other women.[2] In the case of AIDS, cultural attitudes toward sexuality and modesty might make a woman-to-woman program more acceptable. Women are an important target group for AIDS education because they are involved in the transmission of AIDS via all three major transmission routes: perinatal, intravenous, and sexual. Women are also wives and mothers and can disperse information about AIDS to husbands, boyfriends, and teenaged children.

The traditional roles of Vietnamese men and women were another aspect of culture that had an impact on the development of the AIDS educational program. Southeast Asian societies were completely male dominated societies. The male head of household handled all monetary transactions, made every major family decision, and dealt with the outside world.[2] Women and children were not supposed to socialize as males did. Women were expected to stay at home, run the house, raise the children, and support their husbands' work. In the United States, Vietnamese women often feel that they cannot cope with their new environment. Some find the public assistance application process, cashing checks, or negotiating the food stamp process beyond their comprehension.[2]

These role characteristics became apparent during the design of the program and the pilot testing of the instrument. The WIC program is a program for women and children and is attended by women of all ethnic groups. However, among the Southeast Asians, and in this case the Vietnamese, men attended the WIC program sometimes along with their partners and sometimes alone. The WIC program staff permitted this as long as the men also attended the educational program. During the pilot testing of the instrument, it became apparent that a male version of the questionnaire was necessary. Some women participants in the program handed the questionnaire to their partners to complete, others turned to their partners for assistance, and in some cases only the male was present for the program. In response to these occurrences, both male and female versions of the questionnaire were developed.

Cultural beliefs about sexuality and language differences superimposed on role differences presented problems in the development of the program and the questionnaire. There is a cultural bias against discussing sex with strangers. The subject is practically taboo for women, although females are more candid and open with other females.[10] Allusions and circumlocutions are used when sex life and sexual problems are discussed. In Asia, topics that include discussions of sexuality, illegitimacy, and homosexuality are not subjects for public presentation. In many parts of Asia, homosexuality is illegal.[11] Some countries deny that illegitimacy occurs, and illegitimate children and orphans are not acknowledged and are ignored. In addition to the prohibition against discussing sexual matters, the language often does not have words that will directly translate from English into Vietnamese.

Two topics presented problems in developing the AIDS education program: sexual topics and intravenous drug use. The use of allusions and circumlocutions was one problem. This practice became evident in trying to translate the word "condom." An early translation, "pocket of love," was not considered direct enough and was replaced with a demonstration of a condom and the introduction of the English word "condom." AIDS itself is referred to as the "sickness stemming from love," but in the program was referred to by the English acronym "AIDS."

Translation of two items on the questionnaire presented major difficulties. An item inquiring whether the respondent or her sexual partner had multiple sexual partners was translated, "my friend and I have sex with different people." The back-translation did not reveal this inconsistency. The pilot test, however, signaled a clear warning that something was wrong with the item, when 64.4% of respondents answered "yes" to this question. Similarly, the item that asked whether the person or her partner used illegal intravenous drugs was translated, "my friend and I use injected medication"; 86% of the pilot group responded "yes." The translators, a Vietnamese man and an older woman, were questioned about how these inconsistencies in translation could have occurred. They responded that it would have been insulting to the women in the study to have translated the items in the way the investigators intended. The same sort of reaction occurred in a young Vietnamese man who served as a data collector. He was appalled that the women in the study were being asked these questions. Each time, the investigators had to request that translations be accurate and that data collection proceed.

Despite these difficulties, there was widespread community support and participation in the study. As an example, the first day that the research project team arrived at the study site to begin planning the program, the word had spread that there was to be an AIDS educational program. Many persons attended the WIC clinic that day in anticipation of being included in the program. Interest was stimulated by reports that a young Vietnamese man had AIDS and had attended the mental health clinic. The mental health counselor was reported to be afraid of him and had refused to see him.

Although there was initial reticence or shyness on the part of some of the subjects prior to participation in the program, after the program was completed, participants expressed their satisfaction with it. One of the data collectors, an older Vietnamese man, became so convinced of the worth of the program that he wanted it repeated in sites other than just the WIC program.

The process of designing and developing the AIDS educational program was influenced greatly by the cultural beliefs of the Vietnamese and by the study site. The hypotheses were that there would be significant differences in pretest/post-test measures of knowledge, attitudes, and intended changes in practices as a result of a culture-specific AIDS educational program for Vietnamese clients of the WIC program.

METHODS

Setting. An agreement was reached with the Public Health Foundation Women, Infants, and Children (WIC) program in Los Angeles County to provide AIDS education for their Vietnamese clients. The WIC program has more than 70,000 clients enrolled; about 3,000 of these are Southeast Asian, and 60% of these are Vietnamese. Clients attend the WIC program on a monthly basis to receive food coupons. Prior to receiving the coupons, they participate in an educational and counseling program. In the spring of 1988, one month was set aside for AIDS education, and all Vietnamese clients of WIC were invited to attend the AIDS educational program as their educational program for the month.

Design. The study used a variation on the nonequivalent control group design.[2] The experimental group (N = 316) constituted a naturally assembled collective of Vietnamese WIC clients at three WIC program community sites who consented to be involved in the study. This group received a pretest, the AIDS educational intervention, and a post-test. The control group (N = 53) consisted of Vietnamese WIC clients at a fourth site who received a pretest and then attended an educational program on nutrition during pregnancy. This group received the AIDS educational program at a later date.

Procedure. Subjects in the study were approached when they came for their regularly scheduled visits. They were invited to participate in an AIDS educational program and research project; informed consent was obtained at that time. There were 369 subjects who participated in the study, and an additional 984 persons who attended the program but did not complete the pretest and post-test. Subjects read and completed the pretest on their own. Vietnamese data collectors were available to assist the respondents by answering questions on how to complete the pretest and by reading it to the respondents if they had difficulty. They were also available to receive comments regarding the questionnaire. Completion of the pretest took about five minutes.

Following the pretest, subjects attended a 12-minute Vietnamese language slide-tape educational program on AIDS, which was presented by a Vietnamese nurse educator. Subjects also received a Vietnamese language brochure on AIDS and a community resource brochure which focused on AIDS service organizations and resources specific and available to the Vietnamese population. The educator was available for questions following the program. After participation in the educational program, subjects completed the post-test. The same procedures using Vietnamese data collectors were followed for the post-test as for the pretest. Completion of the post-test took less than five minutes.

Sample. The experimental group consisted of 242 women (76.5%) and 74 men (23.5%), and the control group consisted of 53 women. Sample characteristics are presented in Table I.

Relationships were examined between sociodemographic characteristics and group membership (experimental, control), between sociodemographic characteristics and sex, and between sociodemographic characteristics and program sites. There were

TABLE I. Sociodemographic Characteristics of Study Participants

	Experimental Group		Control Group
	Female	Male	Female
	(N = 242)	(N = 74)	(N = 53)
Age (years)			
Range	17–51	17–58	16–48
Mean	30.4	31.3	30.1
Education (year)			
Range	0–16	1–16	3–17
Mean	8.7	8.9	9.2
Marital status (%)			
Married	79.1	81.5	78.3
Never Married	14.1	11.9	12.7
Ever Married (divorced, single, widowed)	6.8	6.6	9.0
Monthly income ($)			
0–832	80.6	74.1	76.4
833–1666	19.4	25.9	23.6
Children under 18 years (%)			
0–2	50.3	47.7	49.2
3–5	46.3	47.4	45.1
6 and over	3.5	5.0	5.7
Religion (%)			
Protestant	2.5	2.4	1.9
Catholic	19.8	22.2	19.2
Buddhist	61.9	60.8	61.2
Other/none	15.7	14.5	17.7
Country of Birth (%)			
Vietnam	98.1	98.3	98.2
US	1.9	1.7	1.8

no significant relationships between sample characteristics and group membership. Furthermore, there were no significant relationships between sociodemographic characteristics and sex or program site. In the total sample, the number of children was significantly related to age ($r = 0.24, p = 0.000$), years of school ($r = -0.18, p = 0.004$), and income ($r = 0.36, p = 0.000$); income and age were also significantly related ($r = 0.22, p = 0.000$).

Instrument. A questionnaire was developed in both English and Vietnamese that collected sociodemographic data and assessed the attitudes, knowledge, and practices of respondents as they related to AIDS. The questionnaire was developed with the assumption that most respondents were literate. Various literature and resources were surveyed to develop items on the questionnaire: questionnaires currently in use to measure knowledge and attitudes toward AIDS; AIDS educational materials and literature; literature and educational materials about AIDS that focused on women; and general AIDS literature and research. Knowledge items assessed knowledge of signs and symptoms of AIDS, transmission through sexual contact and intravenous drug abuse, prevention of AIDS, and community resources for persons with AIDS and their families. Attitude items assessed attitudes toward homosexuality and bisexuality, drug use, sexual promiscuity, and fears such as homophobia, AIDS transmission, and blood donations. Practice items assessed current sexual practices and drug use practices. Responses to the knowledge, attitudes, and practice items were dichotomized into "Yes" and "No" categories. Ordinal scales were not considered because of previous experiences that suggested a possible cultural bias in the Likert-type scale format with refugee populations.[13] Sociodemographic items assessed age, education, income, marital status, religion, number of children, race/ethnicity, and country of birth. The pretest and post-test were the same, except that on the post-test the order of items was changed, subjects were asked to project their intended changes in practice, and the sociodemographic items were deleted.

WIC administrators and educational staff (Vietnamese women and other Asian women), Asian health professionals, and Asian researchers acted as an advisory group in preparation of the pretest/post-test questionnaires. Initially, a long questionnaire (43 items) measuring knowledge, attitudes, and practices was developed in English. This questionnaire was submitted to the advisory group, which made changes in length, wording, and question order. A shortened version (16 items) of the questionnaire resulted. Items that were deleted were judged by the advisory group as being redundant, offensive, or too complex. Both the long and short versions of the questionnaire were submitted to experts in AIDS research and questionnaire construction at UCLA Schools of Nursing, Medicine, and Public Health. These experts judged the questionnaires for content validity. Modifications were made in wording and order until 100% agreement was reached with the panel of judges. The Human Subject Protection Committee (HSPC), UCLA, and the California Department of Health Services Office of AIDS also reviewed and approved the questionnaire for content. The questionnaire was then translated into Vietnamese by the Vietnamese nurse educator. It was next back-translated into English by a different bilingual Vietnamese translator to establish semantic equivalence between the English and Vietnamese versions. The questionnaire was pilot tested on one group of Vietnamese women participants in the WIC program. The questionnaire underwent its final revision based on results of the pilot testing.

The 16-item questionnaire was considered to contain items measuring knowledge (ten items), attitudes (three items), and practices (three items). Scales were created for each group of items. Reliabilities for these scales were determined using Cronbach's alpha, but did not reach acceptable levels. An exploratory factor analysis of the dimensions of the instrument using a principal components analysis with a quartimax orthogonal rotation identified three factors representing knowledge, attitudes, and practices. The knowledge factor consisted of five of the ten knowledge items with an eigen value of 2.73 and accounted for 17.1% of the variance. The attitudes factor consisted of two of the three attitude items plus an additional item with an eigen value of 1.72 and accounted for another 10.7% of the variance. The practices factor consisted of two of the three practice items with an eigen value of 1.15 and accounted for an additional 7.2% of the variance. Reliabilities for these factors were calculated using Cronbach's alpha and reached moderate levels. For the knowledge factor, alpha equaled 0.65; for the attitudes factor, 0.58; and for the practices factor, 0.59.

Educational Intervention. The educational program consisted of a slide-tape presentation, an AIDS educational brochure, and a community resource brochure. The script of the slide-tape program was written following a procedure similar to that used to develop the questionnaire. Content was based on review of numerous audiovisual presentations currently available and on literature review. The advisory group reviewed and revised the content of the script. Also reviewing the script were the California Office of AIDS, UCLA, and the HSPC. Revisions were made in wording and content of the script. The script was then translated into Vietnamese and back-translated into English to establish semantic equivalence. Slides to accompany the videotapes featured Vietnamese subjects and a Vietnamese nurse educator who also read the script for the slide-tape production. A Vietnamese language AIDS educational brochure was developed to enhance and enforce information provided in the educational program. Content and format of brochures was based on review of numerous brochures currently available. Special consideration was given to educational level and information specific to the Vietnamese population.

Community resources and AIDS service organizations in the Los Angeles area were identified with an emphasis on those specific and available to the Vietnamese population. These community resources were reproduced in a brochure format for distribution during the educational program. The HSPC, UCLA, and the California Office of AIDS reviewed and approved the community resource brochure and the AIDS educational brochure prior to their production.

Data Analysis. Data were analyzed using the Statistical Package for the Social Sciences (SPSS-X). Differences between experimental and control groups on individual items on the questionnaire, and differences in pretest/post-test scores for the experimental group were examined by way of two-tailed t-tests. To decrease the chance of inflating the alpha level when multiple t-tests are used, significance was set at 0.0026. The Pearson product moment correlation coefficient (r) and chi-square analysis were used to examine the relationship between sociodemographic variables and individual knowledge, attitude, and practice items. Relationships were examined as well between sociodemographic variables and a single knowledge factor, attitude factor, and practice factor.

RESULTS

There were no significant differences in knowledge, attitudes, and practices between experimental and control groups in response to individual items on the questionnaire. Significant relationships between sociodemographic variables and individual items on the questionnaire were examined for the total sample. Marital status and income were significantly related to having less knowledge of community resources ($\chi^2 = 12.39$, degrees of freedom (df) $= 4, p = 0.01$; $\chi^2 = 7.77$, df $= 1$, $p = 0.005$, respectively). Those who were ever married (divorced, separated, or widowed) and those with more income had less knowledge. Religion was significantly related to being uncomfortable talking about AIDS ($\chi^2 = 12.45$, df $= 4$, $p = 0.01$). Catholics were more uncomfortable than other religious groups.

Age was inversely related to believing that AIDS is caused by a virus ($r = -0.12, p = 0.03$). Years of education were directly related to believing one could not get AIDS from donating blood ($r = 0.23, p = 0.00$), to not having multiple sexual partners ($r = 0.20, p = 0.001$), to not taking one's child out of a school attended by a child with AIDS ($r = 0.25, p = 0.000$), and to not being uncomfortable talking about AIDS ($r = 0.41, p = 0.000$). Education was inversely related to use of condoms ($r = -0.18, p = 0.005$) and knowledge of AIDS agencies in the community (r = −0.14, p = 0.016). Number of children was inversely related to being uncomfortable talking about AIDS (r = −0.21, p = 0.001), to having multiple sexual partners (r = −0.15, p = 0.01), and to using injected drugs (r = −0.18, p = 0.005).

There were significant differences in pretest/post-test scores for the experimental group on most items. These are presented in Table II. Pretest measures of items that assessed knowledge of AIDS demonstrated that respondents had generally high levels of knowledge about the symptoms of AIDS and the transmission of AIDS. Responses to items that assessed attitudes revealed that the majority of respondents were uncomfortable with homosexuals and children with AIDS attending school with their children. Pretest measures of practices revealed that a surprisingly large minority of respondents indicated that they had multiple sexual partners (24.7% of males and 22.9% of females), and that they used intravenous drugs (32.6% of males and 33.2% of females).

As can be noted in Table II, there were significant post-test changes in knowledge, attitudes, and practices items. There were modest gains in knowledge; these were significant on all but two items. The most dramatic changes in knowledge related to donating blood. There were major changes in attitudes toward homosexual/bisexual men, the subject of AIDS, and children with AIDS. There were also major changes indicated in intended practices, including use of condoms, use of injected drugs, and multiple sexual partners.

From the factor analysis, items 2, 3, 6, 8, and 9 (Table II) combined to form a knowledge factor; items 5, 12, and 13 combined to form an attitudes factor; and items 15 and 16 combined to form a practices factor. There were significant differences in pretest/post-test scores for all of these factors. Again, the attitudes factor showed the greatest change; there was also a noteworthy change in the practices factor.

Relationships were examined between sociodemographic variables and weighted factor scores. The knowledge factor was not significantly related to any of the sociodemographic variables. The attitudes factor was significantly related to years of schooling (r = 0.40, p = 0.000). The practices factor was significantly related to age (r = 0.16, p = 0.02) and number of children (r = −0.20, p = 0.006).

TABLE II. Pretest/Post-test Differences in Knowledge, Attitudes, and Practices

	t Value	Degrees of Freedom	2-tail Probability
Individual Items			
1. AIDS caused by virus	−3.51	291	0.001
2. People with AIDS usually die	−3.72	294	0.000
3. Get AIDS from sex with carrier	−4.27	275	0.000
4. Get AIDS from sharing needles	−3.65	291	0.000
5. Get AIDS from donating blood	−57.92	260	0.000
6. Get AIDS from mother to baby	−3.04	293	0.003*
7. Can protect self by using condoms	−6.80	256	0.000
8. Persons with AIDS have fevers	−4.67	252	0.000
9. Persons with AIDS lose weight	−3.38	266	0.001
10. AIDS agencies available in community	−2.93	277	0.004*
11. Uncomfortable talking to homosexual/bisexual men	−10.72	257	0.000
12. Uncomfortable talking about AIDS/sex	−24.41	254	0.000
13. Take child from school attended by child with AIDS	−53.10	273	0.000
14. Self or partner uses condoms	−9.93	244	0.000
15. Self or partner uses injected drugs	−20.23	256	0.000
16. Self or partner has sex with others	−10.64	239	0.000
Factors			
1. Knowledge (items 2,3,6,8,9)	−5.06	245	0.000
2. Attitudes (items 5,12,13)	−43.25	222	0.000
3. Practices (items 15,16)	−17.93	230	0.000

* Significance was set at 0.0026.

DISCUSSION

The findings of this study supported the hypotheses. There were significant pretest/post-test differences in knowledge, attitudes, and intended changes in practices in the Vietnamese participants in the program. The study site and attention to the culture of the participants influenced the success of the program. Educational programs are an integral part of the study setting (WIC), and clients of WIC expect an educational program before receiving their food coupons. Clients feel positive and comfortable with the WIC program and familiar with its routines. The AIDS educational program duplicated these routines. The culture of the participants influenced the language, content, and portrayals in the slide-tape program, the questionnaires, and the brochures. It also governed the ethnicity of the nurse educators, the data collectors, the translators, and the advisory board. Cultural roles, social support systems, and beliefs were taken into account in allowing participation in the study, selecting the study site, and using women to reach and teach other women.

The massive educational program being undertaken in the United States against AIDS has gone ahead with relatively little data to support the effectiveness of educational programs in preventing disease. This has been especially true of programs for minority populations. While this study cannot answer the question of whether educational programs will prevent disease, it does support the idea that knowledge, attitudes, and practices can be changed by educational programs in a minority population, at least on a short term basis. Program planners have been fairly confident that educational programs can effect changes in knowledge. This study revealed that although knowledge level of AIDS was generally high in the sample, a significant increase in knowledge did occur as the result of the educational program.

Program planners have been less confident that educational programs can effect changes in attitudes. The findings of this study suggest that information may change attitudes, at least in the short term. Attitudes showed the greatest change as a result of the educational program.

Several questions still remain about the items on the questionnaire that attempted to measure drug use and sexual practices. During the pilot study, it was revealed that these items were incorrectly translated to avoid offending the subjects. The items were re-translated and then back-translated again for the actual study. Still, a sizable minority of both male and female subjects indicated that they had multiple sexual partners and used illegal intravenous drugs. This was difficult to believe, and yet another translation was requested of a Vietnamese university student. She translated the items as "My lover or I use illegal drugs pumped into the vein" and "My lover or I have intercourse with other people." The translator, who was bilingual, believed that both of these items were understandable, but that some participants with less education might not be familiar with the word "intercourse."

On the post-test, however, these same terms were used to measure intended changes in practices. Respondents indicated overwhelmingly that they intended to "not pump illegal drugs into the vein" (97.6%) and "not have intercourse with more than one person" (97.2%). Once again, the findings of the study suggest that educational programs can effect intended changes in practice.

The question of whether or not the respondents understood the sexual and drug use items correctly on the pretest cannot be answered from the information available. If participants were accurately indicating their drug use and sexual practices, there are implications for the AIDS epidemic. Were the virus to be introduced into the Vietnamese population, these practices would greatly accelerate the transmission of AIDS in the community.

A limitation of the study was the lack of a retest. Changes in knowledge, attitudes, and intended changes in practices were measured immediately following the educational program. Whether these changes will persist over time is not known. Funding priorities set by the funding agency did not permit retesting at a later date. Studies duplicating this one should definitely include a retest.

The study is also limited by its location in one type of community setting. Conditions for conducting the study were optimal in the WIC sites. It may not be appropriate to generalize these findings to other community settings.

Other investigators who might consider duplicating the study at WIC sites in other cities or in other community settings should be particularly careful in translating items having to do with drug use and sexual practices. Before anything definitive can be said about these topics, future studies must further investigate these practices in the Vietnamese population.

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Acquired immunodeficiency syndrome in the black community: Focusing on education and the black male

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Acquired immunodeficiency syndrome (AIDS) is destined to go down in history as one of the most devastating diseases ever to plague mankind. In addition to its impact on personal and public health, it has had profound national and worldwide effects economically, ethically, and politically. Based on past and present trends in epidemiology, the Centers for Disease Control (CDC) have predicted 270,000 cases by 1991, with 179,000 deaths. The economic impact could be astronomical since as much as $147,000 is expended for the hospital care of each patient, excluding outpatient care, medication, and social costs.[1] In some cities, for single men between the ages of 24 and 44 years, AIDS is the leading cause of premature mortality as measured by years of potential life lost.[2] Already, in New York City, AIDS is the leading cause of death for males between the ages of 30 and 39 years, and the second leading cause of death for women aged 30–34 years.[3] Despite these disturbing statistics, the impact on blacks is even more alarming since there are proportionately more AIDS cases in the black community and the mortality rate for blacks with AIDS is significantly higher as compared to whites.[3,4] The black male stands as a key figure in the spread of the AIDS virus in the black community. It is imperative that he become a focus of AIDS education and that he play a major role in AIDS prevention.

BACKGROUND

The first cases of AIDS were reported by the CDC in 1981 in articles describing outbreaks of *Pneumocystis carinii* pneumonia and Kaposi sarcoma in homosexual men from New York City and California.[5,6] Since that time, the causative agent has been identified as the human immunodeficiency virus (HIV), formerly referred to as the human T-lymphotropic virus type III (HTLV-III), the lymphadenopathy-associated virus (LAV), and the AIDS-associated retrovirus (ARV).[7] Supportive evidence indicates that HIV may have originated in Africa and that it may be related to the simian immunodeficiency virus (SIV).[8-13]

HIV relentlessly disarms the immune system by attacking and destroying a patient's helper T-lymphocytes. AIDS represents the end stage of infection with this virus, but asymptomatic carriers and patients with AIDS-related complex (ARC) account for a much higher proportion of HIV disease. For every patient with AIDS, there are probably six or seven with ARC. For every patient with ARC, there are probably ten asymptomatic carriers. The virus is thought to be latent in this latter group. They are considered infected for life and are quite capable of transmitting the virus to others. Approximately 1.5 million Americans are currently infected with HIV, and 20–30% of them will develop AIDS over the next five to seven years.

TRANSMISSION

Transmission of the AIDS virus occurs mainly through sexual contact, blood transfusion, perinatal transfer, sharing needles, receiving infected semen, and exchange of other body fluids. The high-risk groups most likely to contract and transmit the virus include male homosexuals and bisexuals, intravenous (IV) drug abusers, hemophiliacs, children born to seropositive mothers, and sexual partners of those at risk for the disease. Casual contact with an infected person, including handshaking and sharing drinking glasses, toilets, or clothing, does not transmit the AIDS virus.[14,15] There is no documented evidence that insect vectors such as lice, ticks, and mosquitoes, or vaccines such as hepatitis B, are involved in the transmission of HIV.

AIDS IN BLACKS

Since AIDS was initially discovered in homosexual men, who represent the major risk group, there is a general perception that AIDS is nearly exclusively a "gay men's disease" and, more specifically, a "gay white men's disease."[16] This is certainly a misleading perception which requires prompt attention and correction since AIDS has disproportionately affected blacks.[4,17] Although blacks represent only 12% of the American population, they account for approximately 25% of the adult AIDS cases reported (Table I). It is estimated that 1–1.4% of the black population is infected with the virus compared to 0.3–0.5% of whites.[16] Using the Public Health Services model for future projections, it is estimated that by 1991 there

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TABLE I. Comparative Analysis of Human Immunodeficiency Virus Infection by Race

	Blacks	Whites*
US population	12.0%	88.0%
AIDS patients—total[4]	25.0%	75.0%
AIDS patients—homosexual/bisexual[16]	46.3%	88.9%
AIDS patients—IV drug abusers[16]	35.4%	5.2%
HIV positive recruits[21]	0.39%	0.09%
Estimated HIV prevalence[16]	1.0–1.4%	0.3–0.5%
AIDS life expectancy†	18 wk	24 mo

* Includes all non-blacks.
† From CDC.

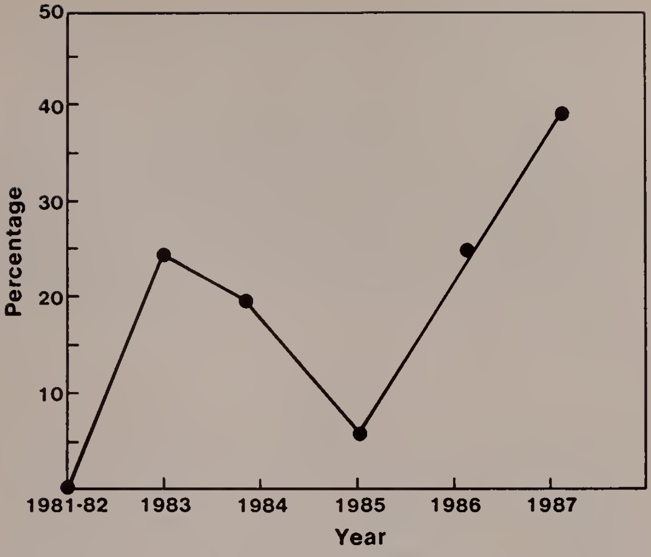

FIGURE 1. Percentage of new IV drug abuse-associated cases of AIDS per year—Michigan.

will be 67,500 blacks with AIDS, with 45,000 deaths.[18] In Michigan alone, blacks represent 13% of the population but 41% of the cases of AIDS.[19] In addition, 36.8% of all AIDS patients in Michigan are black males, 69% of all women with AIDS in Michigan are black, and 80% of the infants and children with AIDS in Michigan are black (personal communication, Michigan Department of Public Health, December, 1987). The impact on minority children is even more striking in New York City, where 97% of children with AIDS are black or Hispanic.[20] AIDS testing of military recruits reveals a higher percentage of seropositivity among black recruits when compared to their white counterparts. A recent study showed that white recruits had an HIV seropositive rate of 0.9 per 1,000, whereas black recruits had over four times this rate, at a seropositivity of 3.9 per 1,000.[21]

HIV TRANSMISSION IN BLACKS

Epidemiologic patterns of AIDS transmission in blacks differ from patterns observed in whites. Whereas whites experience the spread of this disease primarily through male homosexual contact, the black community is affected more by spread through intravenous drug use and heterosexual contact, and this poses greater levels of risk to the general black heterosexual population.[4,17,22] Black homosexual and bisexual men account for 46.3% of all cases in blacks and intravenous drug users account for 35.4%.[16] In contrast, among whites, 88.9% of cases are among homosexual or bisexual men and only 5.2% are among heterosexual IV drug users.[16]

BLACK IV DRUG ABUSERS AND AIDS

The disproportionate incidence of AIDS in the black community is at least in part related to heavy intravenous drug use in densely populated urban areas. More than 750,000 IV drug abusers are at risk for this infection and 200,000 are already infected.[23] The majority of women with AIDS are IV drug abusers between the ages of 13 and 39 and are in their peak childbearing years. Their newborn children have a 50–60% chance of being infected with the AIDS virus.[23,24] In the State of Michigan, an increasing proportion of AIDS cases in recent years are directly or indirectly due to IV drug abuse (Fig 1). According to the Michigan Department of Public Health, 71% of Detroit's newly reported cases in the first two months of 1988 were drug related.

The cocaine epidemic in Detroit and the rest of Wayne County has a significant impact on the risk of AIDS to blacks in Michigan because cocaine is frequently used intravenously alone or in combination with heroin (speedballing) (Fig 2). Statewide, two out of three people admitted for cocaine abuse are black and 85% of statewide "crack" admissions ("crack" is the smokable form of cocaine) are black.[25]

Heroin has a significant impact on the Michigan black urban community, since in Detroit and Wayne County 70% of patients admitted for heroin treatment are black and 67% are black males. Other intravenous drugs contributing to the spread of AIDS in the black community include Percodan, morphine, and Demerol™. The prison population is another site of IV drug use among black males. Drug paraphernalia such as syringes are in limited supply, encouraging the sharing of needles which facilitates the spread of the AIDS virus. Obviously, prison officials are reluctant to supply sterile equipment for illegal drug use. The transmission of HIV among IV drug abus-

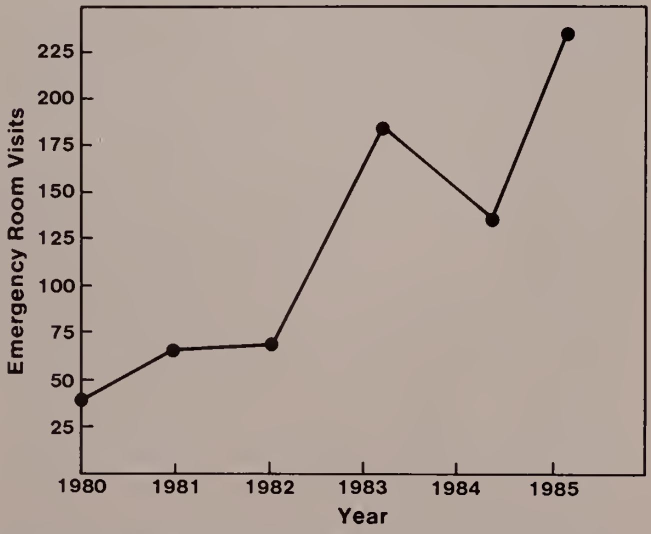

FIGURE 2. Use of cocaine and heroin in combination—Detroit (from Michigan Department of Public Health).

ers is related directly to the frequency of injections, the sharing of equipment, and the length of drug abuse.[23]

IMMIGRANTS

Another factor contributing to the distribution of AIDS in blacks involves AIDS in people from foreign countries, since this group comprises primarily blacks from Haiti and Central Africa.[26] Many are economically disadvantaged immigrants residing in New York or Miami, who either lack private health insurance or financial resources or are ineligible for government subsidized medical services.[26,27]

PRISONERS

It is well known and recognized that blacks make up a higher proportion of the prison population when the percentage of black prisoners is compared to the percentage of blacks in the general population. Although prison administrators try their best to provide supervision, there is still considerable homosexual behavior and intravenous drug use within the prison walls. Some of the homosexual behavior is forced and involuntary. Thus, prisoners represent another potential risk group and again blacks are potentially overrepresented with regard to HIV infections.[28]

HEALTH CARE COSTS

The rapidly escalating number of AIDS cases is putting a severe strain on many already floundering medical facilities, particularly public hospitals in urban areas. In New York's Bellevue Hospital, AIDS patients occupy over one third of the internal medicine beds.[29] Although hospitals treating AIDS patients have high occupancy rates, they are losing as much as 50–70 cents on each expense dollar.[30] It is expensive to care for AIDS patients and reimbursement does not cover all of the large daily costs. Public hospitals in urban areas are financially drained because of the large percentage of AIDS patients who are IV drug abusers. White homosexuals with AIDS tend to have a higher socioeconomic status than the general population and are more likely to have health insurance.[29] In contrast, the IV drug abuser is more likely to be unemployed and without health insurance. Of all cocaine addicts admitted to Wayne County hospitals, 77% were unemployed. The patients with IV drug abuse-associated AIDS are also more expensive to care for because they do not seek care until the latter stages of the disease, have more infections, and have a poorer prognosis.[16]

Many patients who do not have private insurance are covered by Medicaid. In San Francisco, 25–30% of those dying with AIDS are on Medicaid, while 65% of New York's AIDS patients are on Medicaid. Due to many socioeconomic factors, blacks are overrepresented as Medicaid recipients. Sixty-five percent of AIDS patients receiving Medicaid in Michigan are black. The lifetime cost of treating one AIDS patient in Michigan is approximately $29,000. Nationally, it is predicted that the cost to Medicaid for AIDS care will be $1 billion in 1991.[29] As time goes on, Medicaid, which covers approximately 23% of AIDS expenditures at this time, will bear more and more of the brunt of the cost of AIDS. Private insurers are opting to drop AIDS patients from their rolls. Ninety-one percent of commercial insurers feel that people testing positive for HIV are uninsurable, and there is evidence that the proportion of AIDS patients with private insurance is declining.[30] Medicare has not been an option for most AIDS patients because the waiting period for disability coverage is two years, and most AIDS patients do not survive that long.

PREVENTING AIDS IN THE BLACK COMMUNITY

The devastating impact of the AIDS epidemic is aggravated by the fact that this disease is incurable, and given enough time is probably uniformly fatal. The AIDS virus integrates its genetic code into the victim's cellular makeup and forces the patient's own cells to produce the products destined to destroy the host.[31] The virus can lay dormant in the unsuspecting host for years and be insidiously spread to others without either person's knowledge. Azidothymidine (AZT) has been approved by the Food and Drug Administration (FDA) for use as a treatment of selected patients, but AZT is highly toxic, has limited use, and is not a cure for AIDS.[32] The first clinical trials for a vaccine (VaxSyn HIV-1) against HIV will begin soon, but final stages of testing of large populations will not begin until the 1990s.[33] Thus, the most promising means of preventing the further spread of AIDS in the black community is education followed by responsible behavior.

Black men must begin to act more responsibly with regard to their sexuality and intravenous drug use. Those who are bisexual should inform their wives and any other prospective partners—male or female—about former and current sexual liaisons. They should practice "safe sex," including abstinence and use of condoms, and should consider voluntary HIV testing.[34] When positive, they should be vigilant to assure that they are not responsible for any additional HIV transmission. Heterosexual male IV drug abusers must likewise become more honest, concerned, and responsible towards their sexual partners. Many women who become infected with HIV are totally unaware that they are intimately involved with a bisexual male or an intravenous drug abuser. IV drug abusers should likewise consider voluntary testing and practice safe sex. They should avoid needle sharing, sterilize their equipment, and enroll in treatment programs to break the vicious hold of drug addiction. It appears that prostitutes are becoming a greater source of AIDS virus transmission, and this represents another area which will require more responsible behavior on the part of the black male if an effective war is to be waged against the spread of AIDS in the black community.[35] Black women and the black community at large should encourage, if not insist on, more credible behavior from the black man. This can be done by encouraging voluntary AIDS testing for known homosexual and bisexual men and known IV drug abusers, informal monitoring of those who are seropositive to encourage them to practice safe sex, and providing educational opportunities using physicians and other resources for a better understanding of AIDS and its prevention.[36] It is important to recognize that current education and prevention programs may be less effective in reaching minority populations, since they are not specifically designed for these groups.[16]

Targeted programs for AIDS prevention are needed for black men who engage in homosexual activity, and for blacks of either sex who engage in other high-risk behavior, including intravenous drug abuse. This identifies a central role for minority professional and community organizations in providing education about AIDS and its prevention in the black community. More health education and risk reduction projects are needed to actively involve black communities to accomplish overall community AIDS risk reduction activities. The key to the success of these programs will be the response of the black male.

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Public education to prevent the spread of HIV infection

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Acquired immunodeficiency syndrome (AIDS) may no longer be viewed as a disease which only poses a significant threat to homosexual men and intravenous drug users. Approximately 7% of cases diagnosed in 1986 were from among three groups in which the majority of cases are thought to result from heterosexual transmission. These were: heterosexual contacts, persons born outside of the United States, and persons with no identified risk factor. The proportion of cases accounted for by these groups is projected to increase to 10% in 1991.[1] If one considers only the more restricted classification of heterosexual contact cases, the proportion of all cases in this category is expected to grow to 5% in 1991.[1]

In response to these statistics, increasing attention has been turned to the problem of educating the wider public about AIDS. While ultimately a variety of strategies will have to be employed to bring about the necessary widespread behavior change, at this point, the principal focus of public health officials is on greatly increasing the general levels of knowledge about the modes of transmission of the disease and the precautions one can take to reduce the risk of infection. While a consensus exists on the central role that health education must play in stemming the epidemic among the heterosexual population, there is considerably less agreement on the issues of who should be educated, what messages should be communicated, and who should assume responsibility for the educating.

It must be stated at the outset that the amount of empirical support that can be marshalled for any recommendations concerning these issues, including those presented here, is far less than we would like. As Dr June Osborn,[2] Dean of the School of Public Health at the University of Michigan, has pointed out, we are paying a price in this crisis for letting the natural sciences get so far ahead of the social and behavioral sciences.

Still we can draw on the knowledge and insights we have accumulated from our previous public health education experience in three areas: (1) ongoing efforts to influence people to modify a variety of deleterious lifestyles and behaviors, such as smoking and diet; (2) efforts over the past six years to persuade established AIDS risk group members—especially homosexual men—to adopt changes in their high risk sexual behaviors; and (3) longstanding efforts to motivate individuals to take precautions to avoid other sexually transmitted diseases (STDs), such as syphillis or gonorrhea.

GENERAL CONSIDERATIONS

A few general points at the outset will help set the recommendations that follow into perspective. First, it is important to recognize the complexity of the task before us. The fear and dread that AIDS evokes in the public consciousness impedes effective educational efforts. It must be acknowledged that this disease is so frightening to much of the public that people have a strong need to dissociate themselves from it; to convince themselves that any AIDS-related communication does not refer to them. Some fear that to seem interested, or to acknowledge that such communications might have personal relevance, would implicate them as a risk group member.

It is essential to recognize the need to go beyond merely transmitting knowledge to the public.[3] In the past, health promotion campaigns have often been characterized by the naive belief that if you just give people information, they will behave in ways that the facts indicate will promote their health.[4] Available data, however, clearly demonstrate that while the relationship between knowledge and behavior is positive, it is weak.[4] The passive transfer of knowledge generally leads to only modest and short term changes in behavior, unless there is already strong motivation for change and social group reinforcement.[5]

There are many reasons why the mere transfer of knowledge is usually not sufficient to modify established behavior patterns. For example, people may understand and learn what you want them to know, but they may not regard the information as personally relevant.[6] We know that people create health schema or their own explanatory models for why certain people develop a disease and others do not.[7] Such explanations transcend statistical associations, and are largely based on idiosyncratic experience and observations of individuals with the disease.[8] These health schema can often be contradictory to scientific opinion.

Another reason why, in the case of AIDS, merely giving people information will frequently not be enough to alter behavior is the widespread public perception that AIDS is still a medical mystery. While in reality we have learned a

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great deal about this disease in a relatively short period of time, this is not the perception among much of the public. That answers to many urgent questions concerning AIDS must still be prefaced with phrases like, "At the present time," or "Based on currently available knowledge," or "It is unlikely," contributes to a public perception of uncertainty and hedging on the part of health officials.[9]

When, as in the case of AIDS, available scientific knowledge is regarded as indeterminate or provisional, its power to influence behavior and alter misconceptions is significantly diminished.[10] While we are likely to achieve some short term gains merely by enhancing the public's knowledge about AIDS, for them to be sustained and to gain momentum we need to create normative, organizational, and environmental supports for behavior change.[11]

WHO SHOULD BE EDUCATED?

Clearly, there is a need for everyone to understand certain basic facts about AIDS and its modes of transmission. Therefore, a certain part of our educational efforts must be directed to the widest possible audience. However, because we have limited educational resources, we are also compelled to prioritize our efforts. Educational activities must continue to be heavily focused on the populations with a high prevalence of HIV infection and whose lifestyle includes participation in high risk practices—especially homosexual or bisexual men, and intravenous drug users and their partners.

Much more extensive educational activities must also be targeted to minority communities. Available data indicate that blacks and Hispanics are greatly overrepresented among those with AIDS. At the present time, approximately 70% of the AIDS cases in heterosexual males, 70% of those in women, and 75% of those in children are accounted for by blacks and Hispanics.[12] Programs directed to these communities must have the active and ongoing support of minority professionals, community leaders, and community based organizations if they are to have any reasonable chance of success.

In addition, not enough attention has been given to the problem of adapting educational materials and messages aimed at minority communities. Too often "adapting" these materials to, say, Hispanics, has simply meant translating them into Spanish.

Especially important, health messages aimed at these minority populations must take into consideration and be adapted to their social attitudes and cultural norms. For example, within the Hispanic subculture, sex roles in sexual matters are quite rigidly prescribed. Women who appear too knowledgeable or assertive about sexual matters may find their character questioned. The honor of the Latin family is strongly bound to the presumed virtue and purity of its women.[13] Thus, efforts to persuade these women to negotiate sexual limits with their partners and to insist that they use condoms may confront strong cultural barriers.

If we are to induce and maintain behavior change among these women, or in any social group or subculture, we must find some way to tie the desired change into their own value and belief system. That is, we must present the health message in a way that permits them to view what we are asking them to do as compatible, rather than at odds, with established beliefs and values. For example, counseling infected minority women not to bear children may encounter strong opposition in a culture in which children are prized. However, precisely because of this cultural value, *uninfected* women may be strongly motivated to protect their child-bearing capacity and to bear healthy children. These values can be used to help persuade women to encourage their partners to use condoms as a precaution against AIDS or other STDs that could compromise their reproductive capacity and the health of their future offspring.

Adolescents are another group that must be the target of more concerted educational efforts. Even prior to AIDS, in 1979, in response to the alarming growth in the rates of STDs, the public health service had established as a goal that by 1990, "every junior and senior high school student in the United States should receive accurate, timely STD education."[14] The AIDS epidemic has only increased the urgency of achieving the goal. The necessity of reaching children early, before most have been confronted with decisions regarding sexual intercourse, has been stressed by Dr Ward Cates,[15] Director of the Division of Sexually Transmitted Diseases, at the Centers for Disease Control. In his view, classroom instruction should not be restricted to the communication of information, but should include the development of skills in decision making aimed at risk reduction.

Adolescence is a time of experimentation that often involves drug use and sexual activities. The subculture is one that frequently encourages and rewards risk-taking behaviors, which can present a significant threat to the physical well-being of our youth. Additionally, many adolescents have a need to rebel against their parents and other authority figures as they struggle to establish separate identities and satisfy their strong need for peer acceptance. Participation in sexual activity may be used by these youth to signify their independence from parents and movement into adulthood.

Taken together, these factors create a social environment in which AIDS-related risk behaviors are likely to be engaged in by a significant proportion of our youth, which makes it imperative that they be a principal target of our educational efforts.

WHAT MESSAGES SHOULD BE COMMUNICATED?

When we turn to the question of what messages to communicate, some lessons can be drawn from our previous efforts to educate established AIDS risk groups—especially homosexual men—about safe and unsafe behaviors. Available research[16,17] strongly suggests that some of the recommendations contained in risk reduction guidelines that have been used to educate homosexual men about risky and safe practices may have had some unintended and counterproductive consequences.

Among the most problematic of these have been the recommendations to "reduce" one's number of sexual partners and to "know" those partners. Existing evidence suggests that these recommendations are likely to have promoted a false sense of security among many homosexual men who have continued to engage in risky sex. It appears that these men believe that because they have, for example, significantly reduced their number of partners and now avoid anonymous contacts, they run little danger of infection. Their perception is that because they are in compliance with the recommendations of risk reduction guidelines they have adequately reduced their vulnerability.[16]

Reducing one's number of partners may have had protective value early in the epidemic when the prevalence of infection was still relatively low in most gay communities. However, as it has risen dramatically to perhaps as much as an estimated 68% in some cities with large gay male populations,[18] an unsafe encounter with even a single partner is more likely than not to carry a risk of exposure, and unsafe sexual contact with as few as two partners carries approximately a 90% risk of exposure.

The same recommendation, to reduce one's number of partners, is now being communicated to heterosexuals. As with the homosexual men early in the epidemic, a meaningful reduction in the chances of becoming infected may still at this time result from such a strategy. However, again as with the gay community, this circumstance may ultimately change as the prevalence of infection within the heterosexual population grows. If the percentage of the population infected does increase substantially, it will be extremely difficult to reeducate people later that this strategy of risk reduction has become considerably less efficacious.

The prescription to "know" one's partner is similar. Again, one can rarely take the kind of exhaustive sexual history from a prospective partner that would be necessary to even begin to evaluate the degree of risk inherent in an unsafe encounter with him. Nor is it likely that a prospective partner possesses the kind of information that would be needed about his previous partners' earlier sexual history. While querying an individual about a few points concerning his sexual history might permit one to identify a person with clear high risk characteristics, to conclude that any individual who does not share these characteristics is safe is unjustified.

Traditionally, individuals appearing at STD clinics have often been counseled that apart from condom use and reducing one's number of partners, a strategy that could effectively lower their risk of contracting a venereal infection was to be more selective in their choice of partners. Similarly, in response to the AIDS epidemic, heterosexuals are currently being urged to "know" their partners. More specifically, they are being told that if they "know or suspect" their partner is infected, they should use a condom.

This is a dangerous message because it encourages people to rely on their intuition about potential partners for deciding whether they are likely to be a risk group member or present a risk of exposure. Yet it is well known that many homosexual and bisexual men "pass" as straight their whole lives with few people even suspecting their sexual orientation. Similarly, as our awareness of the pervasiveness of drug use in our society has grown, it has become clear that many seemingly "respectable" people can use intravenous drugs for years without family, friends, or co-workers learning of their behavior.

People should be discouraged from trusting their instincts or intuition in evaluating the risk of HIV exposure associated with a prospective partner. Instead, the message should be that anyone (who has not been involved with their partner in a mutually monogamous relationship for at least eight years) who intends to engage in a sexual behavior that creates the potential for the exchange of body fluids should use a condom *regardless* of how safe they believe their partner to be.

Furthermore, many heterosexuals mistakenly believe that if they can be confident that their partner is not homosexual, bisexual, or an intravenous drug user, they run little risk of infection. Others recognize that infection is not limited to these groups, but erroneously assume that if in addition, they select partners who are white, middle class, and hold professional or white collar jobs they will remain uninfected.

Obviously, the use of social characteristics as screening criteria is a highly unreliable practice. Intravenous drug use, homosexuality and bisexuality, and patronization of prostitutes occurs in all social classes and ethnic groups. Educational messages must directly confront and debunk the notion that being more socially selective in the choice of prospective partners is a sufficient strategy for avoiding the risk of HIV infection.

Careful attention must also be given to avoiding ambiguous language in developing health education materials. The use of terms like "multiple partners" and "sexually active" are examples of language that may seem straightforward to health educators but may be misunderstood by much of the public. It is important to clarify for the public that multiple partners refers to two or more partners in a specified time frame—for example, over the past year. It must also be understood that the term "multiple partners" refers not only to situations in which the individual has two or more partners concurrently, but also to a pattern of serial monogamy, in which the individual has only one partner at a time, but each for a relatively short period of time—for example, less than one year.

The term "sexually active" may also not be well understood by many individuals. Because of the fact that early in the epidemic AIDS was associated with a gay lifestyle dubbed "fast lane" sex, characterized by a large number of partners, the disease has come to be associated in the popular mind with sexual excess and promiscuity. As a result, many people will equate the expression "sexually active" with promiscuity or having multiple partners. Thus, when they hear that sexually active heterosexuals are at risk for HIV infection and should adopt prophylactic actions, they may assume that these messages do not apply to them. The point is, simply, that health messages that may seem clear and unambiguous to the source—that is, the health educator—can carry unintended and erroneous implications to the receiver.

Additionally, if we want people to respond in only one or a restricted set of ways to some health communication, we must also inform people what those acceptable responses are or they may draw alternative conclusions.[4] For example, it has been pointed out that while we have been very effective in educating the public about the link between smoking and certain kinds of cancer, the public has drawn many different implications for their own behavior from this knowledge.[4] Some have cut down on the number of cigarettes they smoke, others have switched to low tar cigarettes, still others have changed to a pipe or cigar, and of course some have stopped smoking entirely. From a public health point of view, of course, all of these accommodations are not equally effective.

We should focus on communicating only one or two clear and consistent AIDS education messages to the public. An example would be that there are only two meaningful adaptations to the risk of HIV infection—abstinence or the use of a condom during every sexual encounter in which the exchange of body fluids can occur (and where the partners have not been in a mutually monogamous relationship for at least eight years).

It is important to limit the health recommendations we communicate to people, because while multiple messages may not seem to be competing with each other, they can be. When individuals are offered several different ways to deal with the threat of infection, they will usually choose the course of action that involves the least personal change or costs.[19] If the alternatives are all equally efficacious this is not a problem, but they rarely are. For example, reducing one's number of partners or knowing one's partners better are not as efficacious or reliable methods of self-protection against HIV infection as abstinence or consistent condom use. Furthermore, as discussed above, some of these other recommendations can promote a misfounded sense of security and invulnerability.

WHO SHOULD DO THE EDUCATING?

There is considerable evidence that the source of any message is an important determinant of the attention it will receive. The influence of the source is primarily determined by three characteristics: their credibility, attractiveness, and power.[5] Therefore, greater use should be made of opinion leaders (ie, well-known, respected, and trusted public figures) in trying to change public attitudes about the epidemic and norms concerning sexual behavior. Because these people are respected and trusted, much of the public will seek to emulate their values and attitudes.

Related to the use of opinion leaders is the idea of developing strategies for changing social attitudes and norms to support the adoption of behavior that will greatly slow the spread of infection. In focusing almost exclusively on health education approaches that emphasize inducing behavior change by altering the individual's level of knowledge or personal sense of vulnerability, we have not fully exploited the potential of social norms and sanctions to constrain or promote certain behaviors. The individual's need for acceptance and the approval of others can be powerful motives in shaping his behavior, perhaps even more potent than health motives.[5] We tend to assume that the most effective way to stimulate health behavior is to appeal to health motives; but in some cases, when these motives are weak compared to others, it may be more efficacious to promote health behavior by appealing to other motives, such as the need for social approval.[20] It is necessary to create a social environment in which abstinence and monogamy are positively sanctioned options. And for those individuals who choose to engage in intimate sexual relations with multiple partners, social approval must be conferred on those who behave in a sexually responsible way. Condom use during sexual intercourse must come to be regarded as a normative expectation.

Additionally, we must remember that while in the context of the AIDS prevention efforts sexual behavior is most prominently thought about as health behavior, sexual behavior is also an extremely important form of social behavior. For most people, sexual activities are used to satisfy important needs for affiliation, acceptance, attachment, and affirmation. Such needs are powerful and humanly fundamental. This may in part explain why some people will persist in sexual behavior that places them at risk for infection despite awareness of the inherent dangers.

Concerning our youth, while it would be desirable for parents to assume the principal responsibility for educating their children about STDs and effective precautions, it is unrealistic to expect this to occur in the large majority of cases. Some parents feel that to discuss such matters is to encourage sexual behavior; others are just too uncomfortable raising these issues; and still others believe these problems simply do not apply to their children. Additionally, because available poll data[21] indicate that a significant proportion of the adult population is still misinformed on important matters related to AIDS education, we must recognize that many parents are probably misinformed and cannot impart accurate information to their children.

For these reasons, at this time we must depend on the schools to educate children about AIDS, STDs, and drug use. To pretend that we can rely solely on parents to perform this role is really to abrogate our responsibility and knowingly leave many children unprepared to protect themselves against these threats.

The motivation to be a responsible parent and to protect one's children from harm is a very strong one in our society. We can use this to induce parents to better inform themselves about AIDS, STDs, and drugs and then to begin to assume a more active role in educating their children. In the meantime, it should be noted that available data suggest that sex education in the school does not undermine parental influence in this realm of behavior.[22]

At the junior and senior high school level, student-run AIDS education activities are likely to be more influential in many cases than those conducted by the school officials. Peers will generally be assumed to be more truthful and unbiased than school officials. Further, because peer acceptance is so important at these ages, the wish to conform with the message may be enhanced. Finally, when peers are the source of the message, the need to rebel against authority figures will not interfere with complying. Organizations like Students Against Drunk Driving (SADD) have demonstrated that certain messages are just more acceptable and powerful coming from peers than from elders.

The popular belief that there is widespread community opposition to sex education in the schools has served as a principal barrier to the implementation of these programs, and now AIDS education, in many places. In reality, however, there is evidence that the great majority of the population support such efforts. Research indicates that only about 1–3% of parents ever refuse permission for their child to take part in a sex education program.[23] Evidence of more specific support for AIDS-related sex education is also available. In November 1986, a representative sample of the national adult population was asked the following question: "Some people feel that an important way to combat AIDS is to provide more education in grade school about sex and sexually transmitted diseases, including the use of condoms, before students become sexually active. Others think this kind of education does more harm than good. Which comes closer to your view?" A full 70% endorsed more sex education.[24]

Part of the explanation for the popular misperception of widespread opposition to sex education appears to be a function of mass media coverage, which tends to overrepresent opponents' viewpoints and activities.[23] Research also indicates that historically, resistance to sex education curricula has come primarily from "citizens' groups" rather than parents, educators, or school administrators.[23,25]

Another important group of educators are physicians. Providing information about risky behaviors should become a standard part of every routine visit to a primary care physician. Because of the authority, status, and power invested in the role of the physician in our society, this group often is able to constrain or encourage behaviors merely by communicating to their patients what they would regard as desirable, especially when a good and trusting doctor-patient relationship has been established previously. Furthermore, Brandt[26] has noted that in the 20th century, Americans have increasingly turned to physicians for information, guidance, and treatment concerning sexual diseases and problems.

The workplace and the community must also become sites of AIDS education. Businesses as well as local civic and social organizations must share responsibility for public education on AIDS. The problem of selective attention to AIDS-related health messages can be reduced by a strategy of high saturation of the environment, which diminishes the opportunity for avoidance.[27] Such a strategy also helps demystify the disease, thereby facilitating more open discussion of the problem and greater information seeking. Additionally, when collective education takes place in organizations like schools and businesses where individuals have longstanding and ongoing relationships, peer and social relationships can be used to support and reinforce the health message.

There are no easy answers to the problem of educating the public about AIDS. Some people will inevitably resist any government organized or supported efforts to change behavior for political or ideological reasons. To them, such activities will represent unacceptable intrusions by the government into their private lives. Sexual activity has always been considered a private matter. Now, however, we must acknowledge that it can sometimes have important social consequences. As the human, social and ecomomic costs of the epidemic mount, it is no longer possible to take the position that what someone else does sexually is *always* only his own business.

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The role of the medical assistant

DONALD F. FOY

Despite all the specialization that has occurred in medicine, along with the development of many other allied health care occupations, there has remained a need for a generalist—someone trained specifically to assist the physician in running his or her office. That individual is, of course, the medical assistant, whose principal role is to look after the many needs of the physician's office. It is no wonder the medical assistant is often referred to as the "right hand of the physician"—standing as the patient's first point of contact with the medical care system. The medical assistant can be a combination of secretary, nurse, x-ray technician, and laboratory assistant, and ideally can be trained to perform a number of common procedures in each of these functional areas. Although not formally qualified in any of these fields, the assistant has enough general knowledge of what is routinely done in a physician's office to serve the needs of most practitioners.

Physicians should not underestimate the importance of medical assistants. The medical assistant is not only the nurse in the examining room, the receptionist, and the physician's executive secretary but, just as important, the morale officer for the physician and his or her patients. The assistant also serves as a public relations officer, an accountant, a bookkeeper, and sometimes a friend in need to worried patients.

In a medical setting, patients are subject to stresses that may alter their behavior. They may be apprehensive, distrustful, or belligerent because of their fear or their denial of possible outcomes. The responsibility for establishing an effective relationship rests with the medical assistant. This relationship often serves as the foundation for the patient's relationships with the physician and other staff members. The effective medical assistant must be skilled, tactful, and empathetic in dealing with patients and should display a genuine interest in the individual person.

The quality of interpersonal relations within the office soon becomes obvious to patients. Each office staff member has a different relationship with the physician-employer, which varies with their personalities and areas of responsibility. The medical assistant must be able to communicate effectively and work together with everyone to meet the primary goal of good patient care. The physician and the medical assistant must work together in a relationship based on consideration, trust, and professionalism. Positive relations in any work setting require compromise, support, and cooperation. Each staff member must contribute to the team effort. The team spirit will inspire confidence and trust on the part of the patient.

Public Relations. The medical assistant is the public representative of the physician or the employing facility. In this role the medical assistant can expect to have contact with outside physicians, staff members of other offices or facilities, salespersons, supply company representatives, and service representatives. Courtesy, patience, and effective communication skills will help present a positive public image of the physician's office or facility and its staff. These skills will also establish the medical assistant as an asset to the employer.

The medical assistant is often the first point of contact for the patient; hence, the importance of handling that person in a courteous, efficient, and sympathetic manner. One must keep in mind the old adage, "First impressions are lasting impressions." This is especially true in the medical setting, where patients are either sick or worried that they are.

The tempo of activities in a busy medical office can create a great deal of tension and subjects the medical assistant to constant stress. The medical assistant must therefore possess a high degree of tolerance to stress.

As a consequence, there must be organization in the work setting. In order to survive the day, time must be conserved, performance efficient, and patients kept satisfied. It is imperative that some type of routine work schedule be adhered to.

Personal Attributes. The patient expects the attention of individuals who are prepared to meet his or her special needs. The personal attributes required of medical assistants who wish to take their places as important members of the health care team include the following:

- Ability to communicate with others
- Diplomacy
- Interest in helping others
- Dependability
- Empathy
- Tolerance
- Consideration
- Attentiveness to personal appearance
- A calm manner
- Courtesy
- Common sense

Patient Education. As a member of the health care team the medical assistant has the opportunity to perform a vital service for all patients who enter the office or medi-

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cal care facility. This service is patient education. The benefit that the patient derives from this education is well worth the time and effort spent in providing it and will reflect favorably on the practice. An informed patient is generally more relaxed and cooperative than an uninformed one. In playing the role of medical educator, the medical assistant can also provide valuable information to the patient about other medical care facilities and providers of services with whom the patient may come in contact, such as laboratories, therapeutic facilities, and hospitals.

In addition to the stress of being in a medical setting, patients undergo anxiety when they do not understand why a procedure is necessary or how it will be accomplished. A receptive environment will provide the patient with a comfortable atmosphere in which to ask questions (for example, "Why am I having this kind of examination?" "What is the doctor going to do?" "How do I get my insurance company to pay for this?"). Medical assistants can work with other members of the health care team to create an atmosphere that will inspire patient confidence and trust.

It is possible to *anticipate* the patient's need for basic information regarding administrative and clinical procedures. The *administrative medical assistant* can volunteer information regarding appointment schedules, billing, insurance services, telephone hours, office hours, and emergency coverage. The medical assistant should also emphasize the office policy on confidentiality. All of these measures will help to establish rapport between office personnel and patients and give patients the added confidence to ask questions freely.

Patients often feel inhibited (because of shyness or whatever) about questioning their physicians directly. The medical assistant should recognize this common situation and let the patient know that he or she is available to transmit the patient's questions to the doctor. A good technique is to suggest that patients write down their questions before the appointment and show or read the list to the doctor. The role of the medical assistant as the patient's intermediary will help establish rapport between the patient, the doctor, the medical assistant, and other personnel in the medical care setting.

The *clinical medical assistant* can continue the patient education process by evaluating the patient's understanding of the various examination and treatment procedures. Explaining a process before it begins will alleviate anxiety and provide an atmosphere receptive to patient questions.

Medical assistants are also frequently called on to provide information about support services available in the community, such as transportation and visiting nurse services. They may also assist the patient in obtaining financial assistance for medical services through appropriate insurance coverage or through Medicaid for those unable to pay for care.

The more common situation in which the medical assistant may have to provide informational services occurs when a patient requires hospitalization or is referred to another health care facility for medical tests or consultation. The medical assistant can do a great deal to alleviate the patient's fears by telling the patient what to expect in the new setting and assisting with any arrangements.

Medical information and new treatment methods are favorite topics with the media (television, radio, magazines, newspapers). Patients may ask questions about the medical news they have heard on television or radio or bring articles with them when visiting the office. Although patients may be hesitant to approach the doctor, they may attempt to involve the medical assistant in a discussion of the validity of the information or the effectiveness of certain treatments. Again, the medical assistant can be of service to physicians by alerting them to the patients' concerns or by encouraging patients to speak directly to the doctor about them.

Information about fees and the office policy regarding dealing with third party payors, particularly Medicare, should be readily available. Is the doctor a participating physician under Medicare who accepts assignment for all Medicare patients or a nonparticipating physician who accepts assignment on a case-by-case basis? If assignment is acceptable on a case-by-case basis, the medical assistant may be expected to screen patients and render a judgment about their ability to be balance billed (billed up to the physician's MAAC). The medical assistant who understands Medicare and the tangled web of managed health care systems (PPOs, HMOs, IPAs, fee-for-service) is invaluable.

Employment-Job Opportunities. Medical assistants are most often employed in outpatient health care settings, particularly in physicians' private offices. Today's medical assistant, however, can select a position from among a vast array of opportunities in both outpatient and inpatient facilities depending upon training and credentials. The composition of the health care team in a medical office varies with the number of physicians, patient population served, and specialty of the practice.

The medical assistant career field has become a distinct health care occupation, despite some evidence that the career field is not clearly understood by the health care delivery system. Confusion regarding the role of the medical assistant has been, in part, the result of the rapid growth in formal medical assistant training programs, lack of standardization in training programs, generalized training given to the medical assistant in an industry characterized by growing specialization, wide use of the medical assistant in many functional areas, and emergence of the physician's assistant.

It is therefore not surprising that one of the key issues today is recognition of medical assistants as one of the allied health professions. This is necessary in order that medical assistants be included under federal health manpower legislation. Unfortunately, the Department of Health and Human Services and other government agencies have not included the medical assistant in their allied health career listings. Provision is made for the dental assistant, dental hygienist, physical therapist, occupational therapist, medical record librarian, dietitian, x-ray technician, laboratory technician, and several others, but not the medical assistant.

Moreover, the occupation is not a recognized health care occupation under Title 8 of the New York State Education Act. The Medical Society of the State of New York promised in 1987 to be of assistance with the legislature in gaining this well-deserved recognition. Perhaps with a more concerted effort next year we might even be successful.

Update: Universal precautions for prevention of transmission of human immunodeficiency virus, hepatitis B virus, and other bloodborne pathogens in health-care settings*

INTRODUCTION

The purpose of this report is to clarify and supplement the CDC publication entitled "Recommendations for Prevention of HIV Transmission in Health-Care Settings."[1†]

In 1983, CDC published a document entitled "Guideline for Isolation Precautions in Hospitals"[2] that contained a section entitled "Blood and Body Fluid Precautions." The recommendations in this section called for blood and body fluid precautions when a patient was known or suspected to be infected with bloodborne pathogens. In August 1987, CDC published a document entitled "Recommendations for Prevention of HIV Transmission in Health-Care Settings."[1] In contrast to the 1983 document, the 1987 document recommended that blood and body fluid precautions be consistently used for all patients regardless of their bloodborne infection status. This extension of blood and body fluid precautions to *all* patients is referred to as "Universal Blood and Body Fluid Precautions" or "Universal Precautions." Under universal precautions, blood and certain body fluids of all patients are considered potentially infectious for human immunodeficiency virus (HIV), hepatitis B virus (HBV), and other bloodborne pathogens.

Universal precautions are intended to prevent parenteral, mucous membrane, and nonintact skin exposures of health-care workers to bloodborne pathogens. In addition, immunization with HBV vaccine is recommended as an important adjunct to universal precautions for health-care workers who have exposures to blood.[3,4]

Since the recommendations for universal precautions were published in August 1987, CDC and the Food and Drug Administration (FDA) have received requests for clarification of the following issues: 1) body fluids to which universal precautions apply, 2) use of protective barriers, 3) use of gloves for phlebotomy, 4) selection of gloves for use while observing universal precautions, and 5) need for making changes in waste management programs as a result of adopting universal precautions.

BODY FLUIDS TO WHICH UNIVERSAL PRECAUTIONS APPLY

Universal precautions apply to blood and to other body fluids containing visible blood. Occupational transmission of HIV and HBV to health-care workers by blood is documented.[4,5] Blood is the single most important source of HIV, HBV, and other bloodborne pathogens in the occupational setting. Infection control efforts for HIV, HBV, and other bloodborne pathogens must focus on preventing exposures to blood as well as on delivery of HBV immunization.

Universal precautions also apply to semen and vaginal secretions. Although both of these fluids have been implicated in the sexual transmission of HIV and HBV, they have not been implicated in occupational transmission from patient to health-care worker. This observation is not unexpected, since exposure to semen in the usual health-care setting is limited, and the routine practice of wearing gloves for performing vaginal examinations protects health-care workers from exposure to potentially infectious vaginal secretions.

Universal precautions also apply to tissues and to the following fluids: cerebrospinal fluid (CSF), synovial fluid, pleural fluid, peritoneal fluid, pericardial fluid, and amniotic fluid. The risk of transmission of HIV and HBV from these fluids is unknown; epidemiologic studies in the health-care and community setting are currently inadequate to assess the potential risk to health-care workers from occupational exposures to them. However, HIV has been isolated from CSF, synovial, and amniotic fluid,[6-8] and HBsAg has been detected in synovial fluid, amniotic fluid, and peritoneal fluid.[9-11] One case of HIV transmission was reported after a percutaneous exposure to bloody pleural fluid obtained by needle aspiration.[12] Whereas aseptic procedures used to obtain these fluids for diagnostic or therapeutic purposes protect health-care workers from skin exposures, they cannot prevent penetrating injuries due to contaminated needles or other sharp instruments.

BODY FLUIDS TO WHICH UNIVERSAL PRECAUTIONS DO NOT APPLY

Universal precautions do not apply to feces, nasal secretions, sputum, sweat, tears, urine, and vomitus unless they contain visible blood. The risk of transmission of HIV and HBV from these fluids and materials is extremely low or nonexistent. HIV has been isolated and HBsAg has been demonstrated in some of these fluids; however, epidemiologic studies in the health-care and community setting have not implicated these fluids or materials in the transmission of HIV and HBV infections.[13,14] Some of the above fluids and excretions represent a potential source for nosocomial and community-acquired infections with other pathogens, and recommendations for preventing the transmission of nonbloodborne pathogens have been published.[2]

PRECAUTIONS FOR OTHER BODY FLUIDS IN SPECIAL SETTINGS

Human breast milk has been implicated in perinatal transmission of HIV, and HBsAg has been found in the milk of mothers infected with HBV.[10,13] However, occupational exposure to human breast milk has not been implicated in the transmission of HIV nor HBV infection to health-care workers. Moreover, the health-care worker will not have the same type of intensive exposure to breast milk as the nursing neonate. Whereas universal precautions do not apply to human breast milk, gloves may be worn by health-care workers in situations where exposures to breast milk might be frequent, for example, in breast milk banking.

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* These guidelines are reprinted from *Morbidity and Mortality Weekly Report* 1988; 37:377-382, 387-388. The Council of the Medical Society of the State of New York (MSSNY) has adopted these guidelines as MSSNY policy on the recommendation of the society's Committee on Preventive Medicine.

[†] The August 1987 publication should be consulted for general information and specific recommendations not addressed in this update.

Saliva of some persons infected with HBV has been shown to contain HBV-DNA at concentrations 1/1,000 to 1/10,000 of that found in the infected person's serum.[15] HBsAg-positive saliva has been shown to be infectious when injected into experimental animals and in human bite exposures.[16-18] However, HBsAg-positive saliva has not been shown to be infectious when applied to oral mucous membranes in experimental primate studies[18] or through contamination of musical instruments or cardiopulmonary resuscitation dummies used by HBV carriers.[19,20] Epidemiologic studies of nonsexual household contacts of HIV-infected patients, including several small series in which HIV transmission failed to occur after bites or after percutaneous inoculation or contamination of cuts and open wounds with saliva from HIV-infected patients, suggest that the potential for salivary transmission of HIV is remote.[5,13,14,21,22] One case report from Germany has suggested the possibility of transmission of HIV in a household setting from an infected child to a sibling through a human bite.[23] The bite did not break the skin or result in bleeding. Since the date of seroconversion to HIV was not known for either child in this case, evidence for the role of saliva in the transmission of virus is unclear.[23] Another case report suggested the possibility of transmission of HIV from husband to wife by contact with saliva during kissing.[24] However, follow-up studies did not confirm HIV infection in the wife.[21]

Universal precautions do not apply to saliva. General infection control practices already in existence—including the use of gloves for digital examination of mucous membranes and endotracheal suctioning, and handwashing after exposure to saliva—should further minimize the minute risk, if any, for salivary transmission of HIV and HBV.[1,25] Gloves need not be worn when feeding patients and when wiping saliva from skin.

Special precautions, however, are recommended for dentistry.[1] Occupationally acquired infection with HBV in dental workers has been documented,[4] and two possible cases of occupationally acquired HIV infection involving dentists have been reported.[5,26] During dental procedures, contamination of saliva with blood is predictable, trauma to health-care workers' hands is common, and blood spattering may occur. Infection control precautions for dentistry minimize the potential for nonintact skin and mucous membrane contact of dental health-care workers to blood-contaminated saliva of patients. In addition, the use of gloves for oral examinations and treatment in the dental setting may also protect the patient's oral mucous membranes from exposures to blood, which may occur from breaks in the skin of dental workers' hands.

USE OF PROTECTIVE BARRIERS

Protective barriers reduce the risk of exposure of the health-care worker's skin or mucous membranes to potentially infective materials. For universal precautions, protective barriers reduce the risk of exposure to blood, body fluids containing visible blood, and other fluids to which universal precautions apply. Examples of protective barriers include gloves, gowns, masks, and protective eyewear. Gloves should reduce the incidence of contamination of hands, but they cannot prevent penetrating injuries due to needles or other sharp instruments. Masks and protective eyewear or face shields should reduce the incidence of contamination of mucous membranes of the mouth, nose, and eyes.

Universal precautions are intended to supplement rather than replace recommendations for routine infection control, such as handwashing and using gloves to prevent gross microbial contamination of hands.[27] Because specifying the types of barriers needed for every possible clinical situation is impractical, some judgment must be exercised.

The risk of nosocomial transmission of HIV, HBV, and other bloodborne pathogens can be minimized if health-care workers use the following general guidelines:*

1. Take care to prevent injuries when using needles, scalpels, and other sharp instruments or devices; when handling sharp instruments after procedures; when cleaning used instruments; and when disposing of used needles. Do not recap used needles by hand; do not remove used needles from disposable syringes by hand; and do not bend, break, or otherwise manipulate used needles by hand. Place used disposable syringes and needles, scalpel blades, and other sharp items in puncture-resistant containers for disposal. Locate the puncture-resistant containers as close to the use area as is practical.
2. Use protective barriers to prevent exposure to blood, body fluids containing visible blood, and other fluids to which universal precautions apply. The type of protective barrier(s) should be appropriate for the procedure being performed and the type of exposure anticipated.
3. Immediately and thoroughly wash hands and other skin surfaces that are contaminated with blood, body fluids containing visible blood, or other body fluids to which universal precautions apply.

GLOVE USE FOR PHLEBOTOMY

Gloves should reduce the incidence of blood contamination of hands during phlebotomy (drawing blood samples), but they cannot prevent penetrating injuries caused by needles or other sharp instruments. The likelihood of hand contamination with blood containing HIV, HBV, or other bloodborne pathogens during phlebotomy depends on several factors: 1) the skill and technique of the health-care worker, 2) the frequency with which the health-care worker performs the procedure (other factors being equal, the cumulative risk of blood exposure is higher for a health-care worker who performs more procedures), 3) whether the procedure occurs in a routine or emergency situation (where blood contact may be more likely), and 4) the prevalence of infection with bloodborne pathogens in the patient population. The likelihood of infection after skin exposure to blood containing HIV or HBV will depend on the concentration of virus (viral concentration is much higher for hepatitis B than for HIV), the duration of contact, the presence of skin lesions on the hands of the health-care worker, and—for HBV—the immune status of the health-care worker. Although not accurately quantified, the risk of HIV infection following intact skin contact with infective blood is certainly much less than the 0.5% risk following percutaneous needlestick exposures.[5] In universal precautions, *all* blood is assumed to be potentially infective for bloodborne pathogens, but in certain settings (eg, volunteer blood-donation centers) the prevalence of infection with some bloodborne pathogens (eg, HIV, HBV) is known to be very low. Some institutions have relaxed recommendations for using gloves for phlebotomy procedures by skilled phlebotomists in settings where the prevalence of bloodborne pathogens is known to be very low.

Institutions that judge that routine gloving for *all* phlebotomies is not necessary should periodically reevaluate their policy. Gloves should always be available to health-care workers who wish to use them for phlebotomy. In addition, the following general guidelines apply:

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* The August 1987 publication should be consulted for general information and specific recommendations not addressed in this update.

1. Use gloves for performing phlebotomy when the health-care worker has cuts, scratches, or other breaks in his/her skin.
2. Use gloves in situations where the health-care worker judges that hand contamination with blood may occur, for example, when performing phlebotomy on an uncooperative patient.
3. Use gloves for performing finger and/or heel sticks on infants and children.
4. Use gloves when persons are receiving training in phlebotomy.

SELECTION OF GLOVES

The Center for Devices and Radiological Health, FDA, has responsibility for regulating the medical glove industry. Medical gloves include those marketed as sterile surgical or nonsterile examination gloves made of vinyl or latex. General purpose utility ("rubber") gloves are also used in the health-care setting, but they are not regulated by FDA since they are not promoted for medical use. There are no reported differences in barrier effectiveness between intact latex and intact vinyl used to manufacture gloves. Thus, the type of gloves selected should be appropriate for the task being performed.

The following general guidelines are recommended:

1. Use sterile gloves for procedures involving contact with normally sterile areas of the body.
2. Use examination gloves for procedures involving contact with mucous membranes, unless otherwise indicated, and for other patient care or diagnostic procedures that do not require the use of sterile gloves.
3. Change gloves between patient contacts.
4. Do not wash or disinfect surgical or examination gloves for reuse. Washing with surfactants may cause "wicking," ie, the enhanced penetration of liquids through undetected holes in the glove. Disinfecting agents may cause deterioration.
5. Use general-purpose utility gloves (eg, rubber household gloves) for housekeeping chores involving potential blood contact and for instrument cleaning and decontamination procedures. Utility gloves may be decontaminated and reused but should be discarded if they are peeling, cracked, or discolored, or if they have punctures, tears, or other evidence of deterioration.

WASTE MANAGEMENT

Universal precautions are not intended to change waste management programs previously recommended by CDC for health-care settings.[1] Policies for defining, collecting, storing, decontaminating, and disposing of infective waste are generally determined by institutions in accordance with state and local regulations. Information regarding waste management regulations in health-care settings may be obtained from state or local health departments or agencies responsible for waste management.

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Cavitation as an unusual roentgenographic manifestation of *Pneumocystis carinii* pneumonia in a patient with acquired immunodeficiency syndrome

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With the advent of acquired immunodeficiency syndrome (AIDS), *Pneumocystis carinii* pneumonia (PCP) is being encountered with increasing frequency. Although bilateral interstitial infiltrates remain the most common roentgenographic manifestation of this infection, several atypical radiographic findings have been described. We report a case with a very unusual roentgenographic presentation of this infection, which to our knowledge has not been previously described.

Case Report

A 33-year-old woman presented with progressive shortness of breath, fever, night sweats, and a 20-pound weight loss over the previous four to six weeks. In addition, she had a cough productive of clear sputum. Her medical history was only significant for childhood asthma. She had a history of intravenous drug abuse in the remote past but denied any such use in the past ten years. She had, however, smoked "crack" (a form of cocaine) on a few occasions in the previous year.

The initial physical examination was remarkable for sinus tachycardia of 130 beats/min, an oral temperature of 99.8°F, and a respiratory rate of 28 breaths/min. She was mildly cachectic in appearance. There were no other abnormal physical findings.

Gram's stain of expectorated sputum showed epithelial cells with a mixed microbial flora which were felt to be upper respiratory tract organisms. Laboratory evaluation revealed a mild normochromic normocytic anemia; the white blood cell count was 10,700/mm^3 with a slight shift to the left. The arterial blood had a pH of 7.52; PaCO, 22 mm Hg; PaO, 65 mm Hg, with an alveolar-arterial gradient of 87.5 mm Hg on room air. The initial chest roentgenogram (Fig 1) demonstrated several large, thin-walled cavities in both upper lung fields, with bilateral interstitial infiltrates which were most prominent in the lower lobes.

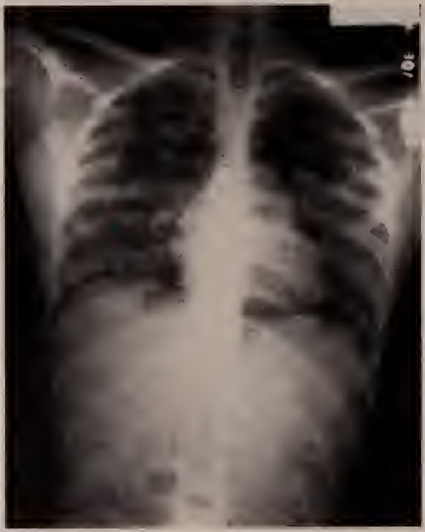

FIGURE 1. Initial chest roentgenogram demonstrating large thin-walled cavities in both upper lung fields and bilateral interstitial infiltrates.

On the first day of hospitalization, as microbiologic specimens were being collected, empiric therapy with isoniazid (INH), rifampin, and intravenous trimethoprim/sulfamethoxazole (TMP/SMX) was begun. Two days later, fiberoptic bronchoscopy with bronchoalveolar lavage was significant for *Pneumocystis carinii*. The lavage fluid was notably negative for acid-fast bacilli, fungi, malignant cells, and viral inclusion bodies. Immunologic studies for cytomegalovirus on the lavage fluid were also negative. All cultures (including fungal and mycobacterial cultures) of the bronchoscopic specimens showed no growth. Numerous cultures of the blood and bone marrow taken during the patient's hospital stay were all negative. An echocardiogram revealed normal cardiac function. The heart valves were free of vegetations. An upper gastrointestinal series demonstrated esophageal ulcerations suggestive of candidiasis. Consent for human immunodeficiency virus (HIV) serology was not granted and therefore could not be obtained.

During the first two weeks of her hospital stay the patient became progressively more anemic and leukopenic, without any evidence of bleeding. The TMP/SMX was stopped, and intravenous pentamidine was begun. A repeat chest film at this time showed that the cavities had become larger and more numerous (Fig 2). The INH and rifampin were discontinued after repeated sputum smears were negative for acid-fast organisms. Her condition markedly improved on pentamidine alone. Another chest radiograph several weeks later demonstrated resolution of the interstitial infiltrates and a reduction in size of the cavities (Fig 3).

The patient has been followed up for one

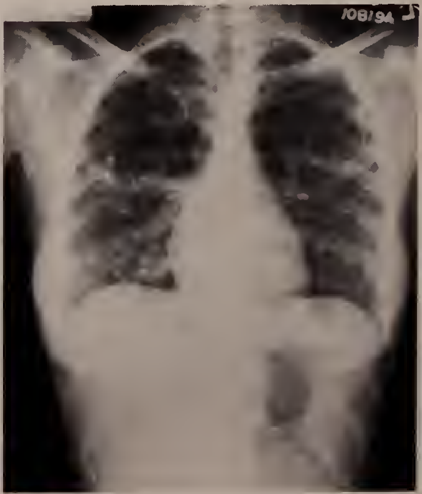

FIGURE 2. Follow-up chest roentgenogram showing an increase in the size and number of the cavities.

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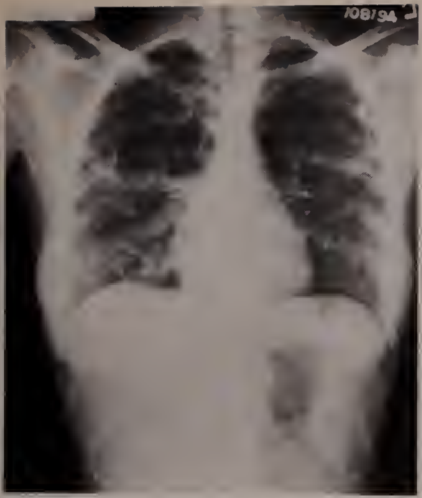

FIGURE 3. Last chest roentgenogram demonstrating resolution of the interstitial infiltrates and a reduction in size of the cavities.

year in the AIDS clinic; she has been noted to be feeling well and gaining weight.

DISCUSSION

This patient's clinical presentation and chest roentgenogram on admission were sufficiently suspicious for pulmonary tuberculosis that antituberculous therapy was begun at the outset. In view of her history of intravenous drug abuse, the radiographic presence of interstitial infiltrates and the increased alveolar-arterial gradient, empiric therapy for *Pneumocystis carinii* pneumonia was also begun. Septic embolization remained another consideration; however, the physical examination, numerous negative blood cultures, and unremarkable echocardiogram did not support this possibility.

Large bilateral multiple cavitations in relation to PCP have not, to our knowledge, been reported. Cavitation, which is a distinctly unusual feature of this infection, has been reported, but not to the extent that was observed here.

The classic roentgenographic expression of PCP has been the presence of diffuse bilateral infiltrates.[1] This has remained true even during the current AIDS epidemic.[2] Several atypical radiographic features in association with PCP have been described. These include unilateral or lobar infiltrates,[3,4] necrotizing pneumonia,[4] spontaneous pneumothorax,[5] and solitary pulmonary nodule.[6] PCP can also present with a normal chest roentgenogram.[7]

In a recent retrospective review of 104 patients with PCP, seven were reported to have small, thin-walled, air-containing "cysts" on initial chest films.[2] These occurred within a radiographic area of infiltrate. In another review of 52 patients with AIDS, one patient with PCP was found to have several small cavitations confined to the right upper lobe.[8] Single areas of cavitation have also been noted.[6,9]

Since the diagnosis in this case was not made histologically, there remains a slight possibility that another pathologic process may have been present. In view of the negative microbiologic, mycobacterial, and fungal studies, and the fact that this patient did well with pentamidine alone, we believe that this possibility is extremely remote.

Pneumocystis carinii pneumonia should be considered in the differential diagnosis of extensive pulmonary cavitary disease in patients who have risk factors for AIDS. This radiographic finding should not preclude efforts to establish a diagnosis of PCP.

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Invasive aspergillosis: An unusual manifestation of AIDS

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It is well recognized that exposure to *Aspergillus* is universal, but disease from invasive and disseminated infection is uncommon. Invasive aspergillosis usually occurs in patients with acute leukemia, cardiac transplant, renal homograft rejection crisis, neutropenia, or cytotoxic or steroid therapy.[1] In the leukemic population, infection occurs in up to 15–20% of cases.[2] Considering the frequency with which disseminated aspergillosis in the immunosuppressed non-AIDS population is reported, it is surprising that aspergillus or aspergillus-induced infection among patients with acquired immunodeficiency syndrome (AIDS) is rare. In this report, we present a case of disseminated invasive aspergillosis in a patient with epidemiologic risk and seropositivity for human immunodeficiency virus (HIV). We believe that invasive aspergillosis was the initial presentation of AIDS in this patient.

CASE REPORT

A 44-year-old man with a history of intravenous drug and chronic alcohol abuse was

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admitted to the hospital. He reported low-grade fever, chills, weight loss—30 lb in six months—and anorexia. He denied night sweats, headache, cough, shortness of breath, nausea, vomiting, abdominal pain, urinary complaints, or neurologic symptoms. He was seen in the emergency room three weeks prior to admission, after having been found unresponsive in the street. He responded to naloxone. At that time, he had a temperature of 105° F and was neutropenic. The white blood cell count was 1,600/mm^3, and the absolute neutrophil count was 480 cells/mm^3. The patient signed himself out of the hospital. The history was significant for a hospital stay in 1984 for left lower lobe pneumonia, cause unknown, and for alcoholic hepatitis in 1985, when the patient was also reported to be lymphopenic. The patient had grown up in New York City and denied recent travel. He lived in a men's shelter.

On admission to the hospital, the patient's temperature was 100.6°F; pulse, 83/min; respiratory rate, 20/min; and blood pressure, 92/52 mm Hg. The patient was 170 cm tall, and he weighed 49 kg.

On examination, he was cachectic, had pallor, bitemporal wasting, and was in mild respiratory distress. He demonstrated variable cooperation, poor attention span, and diminished cognitive function. Shotty axillary lymphadenopathy was palpable bilaterally. He had oral candidiasis and needle track marks on the extremities. No cutaneous abscesses were noted, and his neck was supple. Chest wall excursion was decreased, and no adventitious sounds were audible over both lung fields. The rest of the examination was essentially unremarkable.

Laboratory data revealed a white blood cell count of 2,700/mm^3 with 52% neutrophils, 6% band forms, 19% lymphocytes, 22% monocytes, and 1% basophils. The absolute neutrophil count was 1,566 cells/mm^3, and the lymphocyte count was 513 cells/mm^3. The hemoglobin was 8.7 g%; hematocrit, 26.3%; and platelet count, 268,000/mm^3. The serum sodium level was 131 meq/L; potassium, 2.7 meq/L; chloride, 104 meq/L; bicarbonate, 21 meq/L; glucose, 91 mg/dL; urea nitrogen, 12 mg/dL; creatinine, 1.2 mg/dL; bilirubin, 1.2 mg/dL; albumin, 1.8 g/dL; calcium, 6.2 g/dL, and cholesterol, 95 mg/dL. The SGOT was 33 units/L and γ-glutamyltransferase was 78 IU/L. Urinalysis revealed a specific gravity of 1.015 with trace protein; the sediment contained seven white cells per high power field and no red cells. Arterial blood gas studies on room air revealed pH, 7.52; Pa_{CO_2}, 25 mm Hg; Pa_{O_2}, 112 mm Hg; and saturation of 98%. Microscopic examination of a stained specimen of sputum for acid-fast bacilli was negative.

The chest radiograph revealed a cavity in the posterior segment of the right upper lobe with surrounding infiltrate (Fig 1). The patient was initially treated with pencillin, two million units intravenously every four hours. He continued to be febrile in the 101-102°F range. A culture of the sputum obtained at the time of admission yielded normal flora of the respiratory tract. Two blood cultures were negative. A tuberculin skin test (PPD, 5TU) and skin tests with candidal and mumps antigen were negative. An upper gastrointestinal series was normal. The hepatitis B surface antigen and antibody tests were negative.

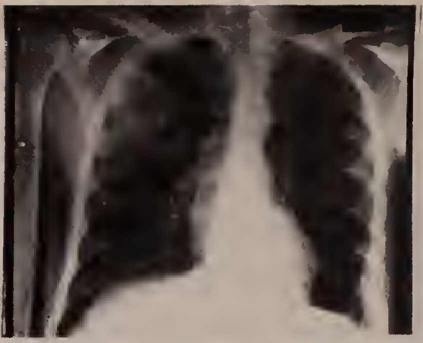

FIGURE 1. Radiograph of the chest taken at the time of admission, showing right upper lobe cavity with surrounding infiltrate.

Subsequent chest radiographs showed new cavities in the left mid-lung field. After eight days of penicillin therapy, the regimen was switched to isoniazid, rifampin, and pyridoxine. A bone marrow aspirate was done, and all cultures were negative. Bronchoalveolar lavage (BAL) was negative for pneumocystis, acid-fast bacilli, and viral inclusions.

On the 21st day of the patient's hospital stay there was significant roentgenographic worsening with multiple cavities and nodular densities in both lung fields (Fig 2). Intravenous vancomycin was added to his drug regimen to cover for presumed infective endocarditis with possible septic emboli. On hospital day 24, an echocardiogram showed normal heart valves and a small pericardial effusion. The white blood cell count gradually dropped to 600/mm^3 with an absolute neutrophil count of 60 cells/mm^3 and a lymphocyte count of 468 cells/mm^3. On day 25 the patient had a grand mal seizure. A spinal tap revealed five white cells (all monocytes); 366 red cells; protein, 66 mg/dL; glucose, 60 mg/dL (serum glucose, 120 mg/dL). India ink preparation, cryptococcal antigen, acid-fast, and fungal cultures were negative. Computerized axial tomography (CAT) of the head was normal. On day 27 intubation was performed for worsening gas exchange. Amphotericin B and pyrazinamide were added empirically. A repeat bone marrow aspirate and biopsy showed increased cellularity, plasmacytosis, and megaloblastic changes. All cultures were negative. On day 28 the patient underwent an open lung biopsy which revealed invasive aspergillosis (Figs 3, 4). Cultures on lung biopsy tissue grew *Aspergillus fumigatus*. On day 34, the human immunodeficiency virus (HIV) serology was reported positive. Evaluation of T-cell subsets was not done. On day 43 the patient died after receiving a total dose of 590 mg of amphotericin B.

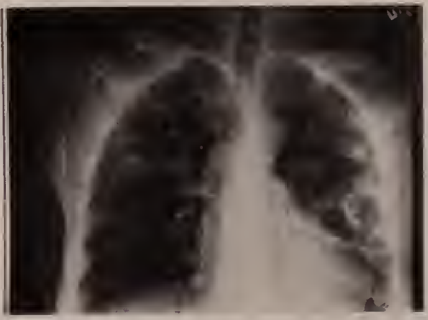

FIGURE 2. Radiograph of the chest taken three weeks into the hospital stay, showing bilateral multiple cavitations and nodular densities.

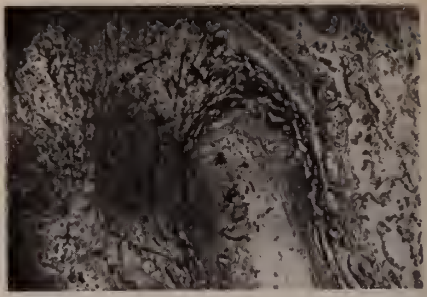

FIGURE 3. Histopathology of the lung biopsy specimen, showing invasion of pulmonary vascular bed by *Aspergillus*.

At autopsy, the body was that of a cachectic man. On gross examination there was extensive bilateral cavitation, necrosis, and abscess formation—especially in the left lung. There was approximately 1,000 cc of pericardial effusion; the heart was otherwise normal. The liver was smooth, and the esophagus, stomach, and intestines were unremarkable. There was no evidence of esophageal candidiasis. The fixed brain weighed 1,350 g with a mildly dilated ventricular system. Microscopic examination revealed pulmonary vascular invasion, acute and chronic pericarditis, and cerebral involvement on the undersurface of the brain stem, cerebellum, and the leptomeninges by organisms characteristic of *Aspergillus* species. There was no evidence of infection by another organism.

DISCUSSION

The genus *Aspergillus*, with its approximately 150 species, is common to soil, water, and decaying vegetation throughout the world. *Aspergillus* infection is usually acquired by inhala-

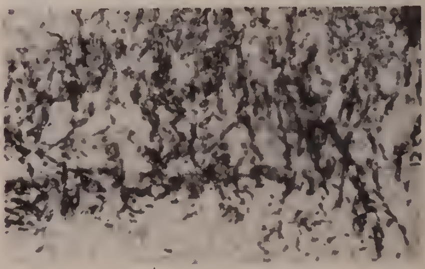

FIGURE 4. Higher magnification of the lung biopsy specimen, showing *Aspergillus* hyphae.

tion of airborne spores. Three main events occur in the pathogenesis of the disease: colonization of the tracheobronchial tree; superficial erosion and ulceration of bronchial mucosa; and transbronchial invasion with parenchymal damage.[3] The basic effect of invasive *Aspergillus* is parenchymal invasion of lung and blood vessels producing infarction, necrosis, and hemorrhage. *Aspergillus* can then hematogenously metastasize to other organs, such as brain, heart, kidney, liver, and spleen. The majority of patients with invasive *Aspergillus* have a hematopoietic-lymphoproliferative disorder and/or are receiving immunosuppressive therapy such as corticosteroids.[3] In a series of 91 patients with documented invasive infection due to an *Aspergillus* species reported by Fisher et al,[4] 78 (86%) patients had a hematologic neoplasm or aplastic anemia as the primary disease. The lung was the most common site of involvement (91%), and the most frequently affected extrapulmonary organ was the brain (18.3%).[4] Infections due to invasive aspergillosis are difficult to diagnose antemortem. The organism is isolated premortem in approximately one third of cases.[4,5] Blood cultures are usually negative, and respiratory secretions are positive in only 12–34% of cases.[6] Serologic studies may help but are often unreliable.[7]

Neutrophils and macrophages comprise the major host defense against *Aspergillus*. Although neutropenia has long been recognized to predispose to infection, neutrophil dysfunction also has been shown to predispose to frequent infections.[8] Corticosteroids inhibit mononuclear phagocytes and can impair mobilization of neutrophils needed to prevent invasion.[3] In the series of Fisher et al,[4] 72 (79%) patients with invasive aspergillosis were neutropenic. Invasive aspergillosis has been reported in presumably normal hosts,[9,10] and in those with alcoholism.[11]

To our knowledge, invasive aspergillosis has not been reported as the sole opportunistic infection in AIDS patients. The simultaneous occurrence of invasive aspergillosis with other opportunistic infections is reported in patients with AIDS.[12-14] In a report of a homosexual patient with an absolute neutrophil count of $935/mm^3$, multiple opportunistic infections were found: disseminated histoplasmosis, disseminated *Mycobacterium avium intracellulare*, genital herpes, oral thrush, and chronic hepatitis B carrier state; invasive aspergillosis was found at autopsy.[12] In another report of a patient with simultaneous *Pneumocystis carinii* pneumonia and pulmonary aspergillosis, who was treated with trimethoprim-sulfamethoxazole and amphotericin B, myocardial abscess due to *Aspergillus fumigatus* was seen.[13] In a review of 446 cases with AIDS, only seven patients had aspergillosis as an additional infection.[14]

The patient described here met the case definition for AIDS based on epidemiologic risk and seropositivity for HIV, along with features suggestive of HIV encephalopathy and wasting syndrome. His hematologic changes of anemia and leukopenia were consistent with those commonly observed in patients with AIDS or AIDS-related complex (ARC).[16] Lymphopenia was noted early in the course of AIDS, usually associated with inversion of the T helper (T_4) to T suppressor (T_8) ratio. Granulocyte abnormalities have also been observed in patients with AIDS. Neutropenia is observed in up to 20% of AIDS and 22% of pre-AIDS patients. This is attributed to neutrophil antibody activity or bone marrow suppression from decreased colony formation of granulocyte-macrophage progenitor cells or from depletion of interleukin-2 responsive lymphocytes. Neutrophil function may also be impaired in AIDS and pre-AIDS, especially chemotaxis and phagocytic responsiveness.[8,17] Bone marrow abnormalities reported in AIDS patients commonly include hypercellularity, plasmacytosis, erythrophagocytosis, and increased incidence of lymphoid aggregates.[16,17]

This patient's major problem was his hematologic profile. He had lymphopenia for over a year and neutropenia for three weeks prior to his final hospital stay. His neutropenia was the major risk factor for fungal infection. His bone marrow findings were consistent with prior AIDS cases.[16,17] It is possible that his malnutrition and disseminated infection in part contributed to his abnormal hematologic profile. However, it is difficult to establish the cause and effect relationship of the hematologic changes due to HIV infection and malnutrition/disseminated infection. But based on the clinical and laboratory profile of the case, our premise is that invasive aspergillosis occurred in this patient as a consequence of HIV infection. Furthermore, *Aspergillus* was the sole pathogen in this patient, unlike in other reports,[12-14] in which *Aspergillus* infection occurred in conjunction with other opportunistic infections in AIDS patients. Therefore, we believe that it might be appropriate that the AIDS definition include invasive aspergillosis. Due to the difficulty in culturing *Aspergillus*, more open lung biopsies and autopsies in AIDS patients with unclear pneumonia may elucidate the true frequency of aspergillosis in these patients.

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Brainstem invasion by *Aspergillus fumigatus* in a child with leukemia

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In recent years, there has been an increased incidence of severe fungal infections in patients with acute lymphocytic leukemia (ALL).[1-3] This appears to be related to the periods of profound immunosuppression caused by antineoplastic therapy and the prolonged use of broad spectrum antibiotics. These factors can change the composition of endogenous bacterial flora and create an environment conducive to invasion by opportunistic fungi.[4]

Aspergillosis is the second most common fungal infection seen in the immunocompromised cancer patient.[2] The mortality due to invasive aspergillosis is high and approaches 100% in disseminated cases.[5] Although the central nervous system (CNS) may be involved in these cases secondary to hematogenous seeding, brainstem involvement is unusual.[3] The present report describes the case of a child with ALL who presented with brainstem signs as the initial manifestation of invasive *Aspergillus fumigatus* infection.

CASE REPORT

A 10-year-old boy presented in 1984 with a history of persistent fever, sore throat, and earache despite a ten-day course of penicillin treatment. The initial findings included marked anemia, neutropenia, and thrombocytopenia with blast cells in the peripheral smear. A diagnosis of T-cell ALL was made based on bone marrow cell morphology and surface markers (T antigens positive 80%). He was referred to Roswell Park Memorial Institute for therapy.

On admission, physical findings included fever, ulceration of the upper lip, bilateral cervical lymphadenopathy, and hepatosplenomegaly. A neurologic examination was within normal limits. His white blood cell (WBC) count was 1.6×10^9/L with 2% blast cells and 98% lymphocytes; hemoglobin, 8.8 g/dL; and platelets, 39×10^6/L. Induction therapy for ALL was initiated with vincristine and prednisone. A chest film demonstrated a small infiltrate in the right upper lobe. Blood cultures were positive for *Staphylococcus aureus* and *Escherichia coli*, for which the patient was treated with nafcillin, mezlocillin, and tobramycin.

Because of persistent fever and the development of unexplained tachypnea on the fourth day of his hospital stay, the patient was treated with intravenous amphotericin B, acyclovir, and trimethoprim-sulfamethoxazole for possible fungal, viral, and *Pneumocystis carinii* pneumonia, respectively. By the next day, his condition had improved. On the 11th hospital day, after being afebrile for five days and having negative blood cultures, an absolute neutrophil count greater than 0.5×10^9/L, and a normal chest film, amphotericin B, trimethoprim-sulfamethoxazole, and acyclovir were stopped. All the antibiotics were discontinued by the 16th day of his hospital stay when the patient's absolute neutrophil count was greater than 1.0×10^9/L. He received prophylactic CNS chemotherapy at that time consisting of intrathecal methotrexate (15 mg), intrathecal hydrocortisone (15 mg), and intrathecal cytosine arabinoside (30 mg).

Within 24 hours of discontinuing all antimicrobials, the fever recurred, accompanied by dizziness, diplopia, and inability to swallow. The patient experienced a left hemiparesis with a left facial droop as well as the inability to close the left eye. His absolute neutrophil count dropped to less than 0.5×10^9/L. He was alert with dysarthric speech.

On neurologic examination, multiple cranial nerve involvement was evident. There was ptosis of the right eye with a dilated right pupil, but both eyes were reactive to light. Visual acuity and fundoscopic examination were normal. The sensory portion of the left fifth cranial nerve was diminished in all three branches. The sensory examination was otherwise grossly normal. There was also a left seventh nerve palsy and multidirectional nystagmus. A motor examination was normal except for the absence of deep tendon reflexes. Upper and lower extremity coordination were intact. Computerized axial tomographic (CAT) scans of the head with and without contrast were normal. A lumbar puncture showed 35 WBC/mm^3 with 85% neutrophils. In the cerebrospinal fluid (CSF), the glucose level was 66 mg/dL (blood glucose was 126 mg/dL) and the protein level was 36 mg/dL. The chest film revealed no abnormalities.

Despite treatment with intravenous amphotericin B, rifampin, isoniazid, septra, tobramycin, and cefotaxime, the patient's clinical condition was unchanged. He remained neutropenic and febrile, and on the 23rd hospital day, generalized tonic clonic seizures developed. A repeat lumbar puncture showed an opening pressure of 80 mm H_2O and contained 220 WBC/mm^3 with 60% neutrophils and 40% lymphocytes. CSF glucose was 40 mg/dL and protein 56 mg/dL. Acid-fast and India ink stains of the CSF were also negative. CSF *Cryptococcus* and *Candida* antigen tests were negative. A repeat CAT scan of the head was again normal. All CSF cultures remained negative for fungi, bacteria, and viruses.

On the 36th day of his hospital stay, the patient remained in relapse, with 90% blastocytes in his bone marrow. He continued to be febrile and his neurologic status was unchanged. A repeat CAT scan of the head now demonstrated an enhancing area in the posterolateral aspect of the fourth ventricle, inferior cerebellar peduncle, and left cerebellar hemisphere, which was interpreted as a probable CNS leukemic lesion. Intrathecal methotrexate, cytosine arabinoside, and hydrocortisone were administered, and amphotericin B was discontinued.

On the 42nd hospital day, severe respiratory distress developed, requiring mechanically assisted ventilation. Bilateral pulmonary infiltrates were now apparent on chest radiographs. An open lung biopsy taken from the lower border of the right middle

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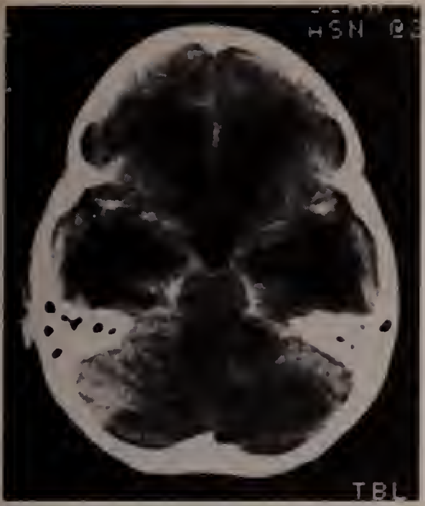

FIGURE 1. CAT scan of the brain with contrast medium technique showing low density lesion involving left cerebellar hemisphere and peduncles with ventricular dilation.

lobe showed areas of pulmonary hemorrhage both on routine histologic evaluation and by special stains. There was no evidence of fungi.

As the patient's condition had further deteriorated, amphotericin B and acyclovir therapy were reinstituted. A CAT scan performed at this time showed an expanding low density lesion in the left cerebellar hemisphere, involving the cerebellar peduncles, with signs of marked ventricular enlargement (Fig 1). On day 47, the patient became comatose. He died on the 52nd day in oliguric renal failure secondary to hypotensive shock.

At autopsy, the major pathologic findings were in the brain and lungs. The brain was edematous, showing gross involvement of the rostrade and caudal parts of the pons and medulla, superior spinal cord, and large part of the cerebellum (Fig 2). The fourth ventricle appeared compressed, with deviation of the cerebellar vermis, a proximal part of the fourth ventricle, and a portion of the cerebral aqueduct to the right side. The aqueduct and cerebral ventricle system were dilated. The histologic examination showed infarction of the aforementioned areas with invasion by the branched septated hyphae of *Aspergillus*. There was a striking invasion and thrombosis of the dorsal part of the lower brainstem (Fig 3). In the lungs there were multifocal hemorrhagic and solid yellow lesions. *Aspergillus* hyphae were present in lung parenchymal sections. Lung cultures at autopsy grew colonies of *Aspergillus fumigatus.*

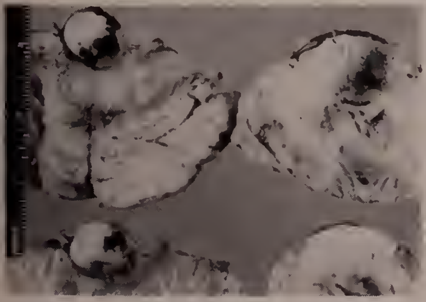

FIGURE 2. Coronal sections showing gross involvement of pons, medulla, superior spinal cord, and cerebellum by aspergillus.

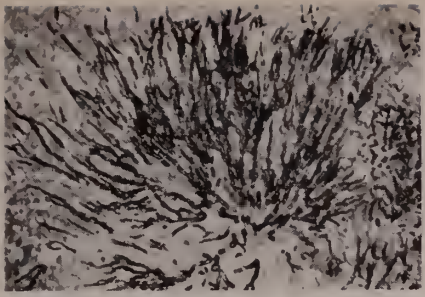

FIGURE 3. Aspergillus invading the brainstem (methenamine silver stain).

DISCUSSION

With the increasing number of cancer patients surviving prolonged periods of immunosuppression, there has been a corresponding increase in the number of cases of invasive aspergillosis.[1-3,6] Although disseminated cases of aspergillosis are common among immunosuppressed cancer patients, only 30–40% of such cases will have CNS involvement noted at autopsy.[1,7] Involvement of the CNS is usually the result of direct spread of infection from areas contiguous to the brain or secondary to hematogenous dissemination. *Aspergillus* seems to involve the brain via the posterior fossa structures when it spreads via hematogenous dissemination.[2,7] It should be noted that a pulmonary lesion is often the initial focus of *Aspergillus* infection from which hematogenous dissemination to the CNS will ensue.[8]

In the patient reported here, CNS aspergillosis was found with no definitive primary site of involvement. Although the patient exhibited evidence of lung invasion by *Aspergillus* at autopsy, no pulmonary involvement was noted at the initial presentation of brainstem signs. Nevertheless, the lungs may still be implicated as the primary site of microscopic invasion, which may have led to hematogenous dissemination to the CNS. The hemorrhagic lesions of the lung seen in this patient may have been an early manifestation of pulmonary aspergillosis. However, CNS aspergillosis without lung involvement has been reported.[9,10]

Brainstem aspergillosis, although rare, is a disease of devastating consequences. Depending on the area of involvement, presentation of CNS aspergillosis varies and may include hemiparesis, cranial nerve palsy, ataxia, or focal seizures.[9] The premortem diagnosis in immunosuppressed patients with cancer is difficult. Because the definitive diagnosis of brainstem aspergillosis can be made only by culture and/or histologic findings of aspergillus in tissue, the diagnosis is most often made postmortem. CSF findings are nonspecific, consisting of slight leukocytosis, and normal glucose and protein levels.[3,11] Isolation of the fungus from CSF has rarely been reported.[11]

The utility of CAT scanning for the diagnosis of brainstem aspergillosis has been disappointing. In this case, no radiologic abnormalities were detectable until late in the patient's hospital course. At that time, areas of low density were evident on CAT scan in the left cerebellar hemisphere extending down the cerebellar peduncles. This correlated with the postmortem findings of extensive invasion of blood vessels with areas of thrombosis and infarction involving the brainstem, cerebellum, and cerebellar peduncles. Our experience was similar to other reports, in which the CAT scan appearance of CNS aspergillosis was described as poorly circumscribed low density areas without capsule formulation.[12,13] One should be cognizant of the fact that contrast enhancement of CNS abscesses may not be detectable radiographically for several weeks after the onset of symptoms,[14-16] and contrast enhancement may be diminished by as little as two days of therapy with corticosteroids.[16-18] Also, inflammatory responses may be absent or decreased during periods of granulocytopenia. It is possible that magnetic resonance imaging may have a positive impact on the diagnosis of CNS aspergillosis.

Other diagnostic methods can arouse the clinician's suspicion of aspergillosis. Nasal surveillance cultures and periodic nasal examinations may be helpful in the early detection of invasive aspergillosis, potentially leading to early treatment and enhanced patient survival.[19] Although the detection of *Aspergillus* antibody is not useful in the diagnosis of invasive aspergillosis for immunocompromised hosts, a radioimmunoassay of *Aspergillus* antigen appears promising.[20]

Modes of therapy for CNS aspergillosis have not been well defined. Once there is CNS invasion with aspergillosis, treatment is rarely successful. The lack of drug delivery in high concentrations to infected brain tissue and the tendency of the organism to invade and occlude blood vessels probably explains the poor results of medical therapy.[2,7,16] Therapy depends on the recovery of host granulocytes. *Aspergillus* shows variable sensitivity to amphotericin B.[2] Successful therapy for CNS aspergillosis has been reported only once with systemic amphotericin B.[21] This agent, however, can at best only prevent progression of the lesions in patients with granulocytopenia. A combination of 5-fluorocytosine or rifampin with amphotericin B has demonstrated some success in anecdotal reports.[22] Intraventricular administration in conjunction with systemic amphotericin B has also been attempted.[11,23] More recently, liposomal amphotericin B has been tried in systemic fungal infections in immunosuppressed cancer patients.[24]

If the lesions are surgically accessible, the combined approach of resection and medical therapy may be effective. However, due to the location of the lesions and the patient's underlying condition, this combined approach can rarely be utilized.

In summary, it may be difficult to establish the diagnosis of brainstem aspergillosis. This diagnosis should be entertained in the clinical setting of a febrile cancer patient with granulocytopenia and with deterioration in central nervous system functions. This entity may mimic leukemic involvement of the brainstem with cranial neuropathies. Isolation of *Aspergillus* from the CSF is rarely accomplished, and conditions such as leukemic infiltrates, hemorrhagic processes, and antineoplastic agent side effects should be considered in the differential diagnosis. Therefore, the diagnosis requires a high index of suspicion, particularly in the setting of granulocytopenia in a cancer patient. Ancillary tests, such as positive nasal surveillance cultures for *Aspergillus* and the detection of *Aspergillus* antigen by radioimmunoassay, may heighten the clinician's suspicion of aspergillosis. The definitive diagnosis can only be made by histologic examination and/or culture of infected tissue. The success of therapy in leukemic patients is predicated on the recovery of granulocytes, surgical intervention, and the early institution of amphotericin B therapy.

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Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Doctors' penmanship

TO THE EDITOR: A copy of Heather Engel's letter[1] in the July 1988 issue on the problem of physicians' penmanship should be attached to each graduate MD's diploma. A goodly part of my time is spent reviewing medical records in the analysis of claims for malpractice. It is appalling how much time is wasted because of scrawl.

It is one matter if I waste my time in deciphering records long after the fact, but totally another if it occurs during the time of active patient care, especially in-hospital. Recently, I reviewed a case involving the State University of New York Health Science Center at Brooklyn (SUNY HSCB). I was truly amazed that *every* entry in 60+ pages was easily readable. What heresy are they preaching at SUNY HSCB?

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[1] Engel H: Physician penmanship. *NY State J Med* 1988; 88:392.

The changing environment of medicine

TO THE EDITOR: James Wright's article, "The changing environment of medicine," concisely and realistically describes the evolution of the medical profession.[1] This information is of help to prospective physicians. How to be a humanitarian, while learning as much as possible, now includes mastering a business where there are a number of outside influences and "big brothers." The hospital environment as well as medical meeting places have become preoccupied, not with discussions of the most advanced medical technology or ethical dilemmas, but with the misconception that doctors are not doing a good job and that doctors are out to make as much money as possible. Perhaps medicine may not be held in as high esteem as it used to be, but it still is the central vehicle for taking care of an individual's most precious commodity, his health. Dr Wright's essay should be among the handouts available in the premedical advisors' offices on college campuses throughout the country.

In recent years the press and media have been quick to disseminate criticism about physicians; rarely publicized are the many "goods" that occur in offices and hospitals throughout the country day after day. Recently, the *New York Times* reported that the socialized medical system in Great Britain is failing and is in deep financial trouble. The United States enjoys some of the finest health care in the world. But the system is being rocked by individuals who because of their influence can exert a negative impact on the profession. The unjustified granting of large financial awards for negligence or for presumed negligence is tempting to the public because the odds are probably greater for receiving a large award than for winning the New York State Lottery. Other individuals have underlying motives that may be economic, psychosocial, or political, and these individuals need to be countered. Many of the "helps" that have come on the scene are often reflective of an intent to rip people off. For instance, groups that exist to help the elderly fill out forms, or groups that advertise that they can truly relieve back pain or dramatically produce weight loss need careful monitoring. Legitimate deliverers of health services should work with their peers to assure quality.

The environment of medicine has changed but hopefully it will change back again. The "greenhouse effect" and its impact on climate, acid rain and its impact on lakes and trees, and "business" and its impact on medicine are all major health hazards. Individuals in medicine must literally and not figuratively help themselves or they will be like the characters in Beckett's play, *Waiting for Godot*.[2] As Vladimir concludes, "Let us do something, while we have the chance! It is not every day that we are needed. Not indeed that we personally are needed."

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[1] Wright J: The changing environment of medicine. *NY State J Med* 1988; 88:413-414.

[2] Beckett S: *Waiting for Godot*. New York, Grove Press, 1954.

Brucellosis in an 18-year-old man from Greece

TO THE EDITOR: Brucellosis is one of the world's most widespread zoonoses.[1] In the United States, human brucellosis is rare, with fewer than 300 cases reported annually since 1965.[2] The disease may be underdiagnosed and underreported, and some authorities estimate that for each reported case another 26 are unrecognized or unreported. We recently cared for a man who presented to the Queens Hospital Center with fever following exposure to farm animals. The patient was found to have brucellosis.

Case Report. An 18-year-old man developed intermittent high fever and back pain two weeks after he emigrated to the United States from Greece where he had worked on a farm caring for cows, pigs, and sheep. On some occasions he slept next to the animals in the barn and often drank unpasteurized goat's and cow's milk. Over a two-week period he experienced intermittent fevers, with temperatures ranging from 101°F to 105°F, progressively worsening sharp and spasmodic lower back pain, and a ten-pound weight loss. He was seen at three other hospitals, treated symptomatically, and sent home. He presented at our hospital with the same complaints.

On examination, the patient's temperature was 104°F; pulse, 86 beats/min; respirations, 18/min; and blood pressure, 100/50 mm Hg. The liver was enlarged to a total span of 14 cm, smooth, firm, and nontender. A firm, smooth spleen tip was felt below the left costal margin. The rest of the physical examination was normal except for congenital horizontal nystagmus.

Laboratory studies revealed a hemoglobin level of 12.8 g/100 mL, hematocrit 39%, platelet count, 225,000/dL; and leukocyte count, 6,800/dL, with 49% mature granulocytes, 40% lymphocytes, and 11% monocytes. Routine cultures of sputum, urine, blood, and stool were negative. Serum chemistry tests including electrolytes were normal. Radiographs of the chest and lumbosacral spine were normal. A technetium bone scan showed increased uptake over the seventh and eighth thoracic vertebra. Computed tomographic examination of that area was normal, but an enlarged spleen was described with a total 14 cm span.

Brucellosis was strongly suspected and the patient was treated with 2 g oral tetracycline daily. There was marked improvement with defervescence of fever. Several blood cultures confirmed the presence of *Brucella sp.* Because of the positive blood cultures, no serologic studies were done. The patient was discharged completely well and told to continue the antibiotic for a total of six weeks.

Discussion. Brucella is a small, gram-negative, nonmotile, nonsporeforming coccobacillus.[3] There are six known species of which four infect man: *Brucella melitensis*, *Brucella suis*, *Brucella abortus*, and *Brucella canis*. *Brucella melitensis* has as its preferred host sheep or goats, and is the most pathogenic species in human beings. Countries reporting more than 1,000 human cases annually in their indigenous populations include Argentina, Greece, Iran, Italy, Mexico, Peru, and Spain. In the United States, half of the cases are the result of contact with cattle and one quarter are contracted from swine; 10% of cases are caused by the ingestion of dairy products, mostly of foreign origin, and the remaining 15% of cases are evenly divided among contacts with sheep, goats, or dogs, laboratory workers, and incidental inoculation with vaccine.[2]

Clinically the interval between infection and appearance of symptoms in acute brucellosis is one to three weeks. Symptoms include fever, lethargy, weakness, weight loss, anorexia, and backache.[4] Also common are monoarticular arthritis of large joints, lymphadenopathy, and hepatosplenomegaly. The disease is rarely fatal since brucella endocarditis is rare.[5] Bone marrow, pulmonary, and central nervous system involvement by brucellosis is uncommon.

The organism can be cultured from various organs including blood. Cultures should be kept under observation for three to four weeks, if necessary. Blood cultures are positive in one third to two thirds of patients with active disease.[2] Specific treatment consists of tetracycline, 2 g daily in divided doses for four to six weeks.[2] Relapses or recurrences occur in 15–20% of cases and require retreatment. Fewer relapses occur if streptomycin is added to the tetracycline. Alternative drugs include chloramphenicol, erythromycin, and ampicillin. Rifampin may also be added to tetracycline as an effective combination.

The patient presented here had a classic picture of brucellosis with a typical history of exposure to farm animals, a two-week incubation period, and symptoms of fever, low back pain, weight loss, and hepatosplenomegaly. Treatment for brucellosis with tetracycline was initiated even before the positive blood culture results became known and the patient's condition improved dramatically in a few days. Physicians in the United States, where this disease is uncommon, should nevertheless consider this diagnosis in any patient who presents with the above constellation of features. The single most important determinant in arriving at the correct diagnosis is a good medical history eliciting the history of exposure to the organism.

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Fear, anger, and the hope of redemption in the age of AIDS

And the Band Played On. by Randy Shilts. 544 pp. New York, St Martin's Press, 1987. $24.95 (hardcover)

Crisis: Heterosexual Behavior in the Age of AIDS. By William H. Masters, MD, Virginia E. Johnson, and Robert C. Kolodny, MD. New York, Grove Press, 1988. $15.95 (hardcover)

AIDS, the Ultimate Challenge. By Elisabeth Kübler-Ross, MD, and Mal Warshaw. 160 pp, New York, Macmillan Publishing Company, 1987. $18.95 (hardcover)

Hundreds of volumes have been published on the AIDS epidemic, but, aside from informational treatises, most generally fall into three categories: the confrontational, the hysterical, and the inspirational. Here are three excerpts from the first few paragraphs of recent books on AIDS:

> Acquired Immune Deficiency Syndrome had seemed a comfortably distant threat to most of those who had heard of it before, the misfortune of people who fit into rather distinct classes of outcasts and social pariahs. But suddenly, in the summer of 1985, when a movie star was diagnosed with the disease and the newspapers couldn't stop talking about it, the AIDS epidemic became palpable and the threat loomed everywhere.
>
> (from *And the Band Played On*)

> In later chapters we will point to many instances of misinformation that the general population has been fed about AIDS. First, however, we will introduce the alarming conclusion we have reached, based on our own research and studies conducted by others: contrary to claims by various government agencies and public health experts that infection with the AIDS virus is still largely confined to the original "high-risk" groups (gay and bisexual men and intravenous drug users), the epidemic has clearly broken out into the broader population and is continuing, even now, to make its silent inroads of infection while many maintain an attitude of complacency . . .
>
> (from *Crisis*)

> Is it possible that our AIDS patients, children and adults alike, chose to contribute their short life spans on the planet Earth to help us open our eyes, to raise our consciousness, to open our hearts and minds, and finally to see the light?
>
> (from *AIDS, the Ultimate Challenge*)

Randy Shilts, the author of *And the Band Played On*, is a reporter widely regarded as the United States' most informed journalist on the topic of AIDS. He has watched the epidemic unfold from the vantage point of San Francisco, a city known for its politically active and cohesive gay community. It hardly seems surprising that he should call for more public awareness, support, and governmental action to stem the AIDS epidemic. His feeling that "AIDS did not just happen to America—it was allowed to happen by an array of institutions, all of which failed to perform their appropriate tasks to safeguard the public health," is an understandably angry indictment of a nation which has appeared to be disinterested in a segment of its populace. By confronting the apathy of Americans, Shilts hopes to move them to action—or at least understanding.

The author of *AIDS, the Ultimate Challenge*, is a famous researcher from the 1960s. Elisabeth Kübler-Ross's 1969 book, *On Death and Dying*, forever changed the way we look at death. Now she focuses on AIDS, but she seems to place no blame on anyone. This is not, she says in her calm, soothing way, a conspiracy of the straight peoples against the deviant, nor a scourge from God against sinners. Rather, AIDS is people dying, dying and trying to understand life itself in the action.

Two of the three authors of *Crisis* are also world-famous researchers from the '60s and '70s. To some it may seem surprising that the ground-breaking sex-researchers Dr William Masters and Virginia Johnson, along with their co-author, Dr Robert Kolodny, should adopt so hysterical a tone. But the subtitle of *Crisis*, "Heterosexual Behavior in the Age of AIDS," tells much of their story. It seems to accept AIDS as understandable (and acceptable?) in the homosexual community, but a "crisis" in the straight community. This near-homophobia may be unintentional. Perhaps Masters and Johnson have felt guilty all these years that their physiological research was adopted as a rationale for the so-called "sexual revolution." And now, in the "Age of AIDS," they feel that the revolution has gotten so out of hand and dangerous that its etiology resembles the reign of terror in 1790s France, with AIDS as the guillotine.

Each of these books has a particular point to make, and each point is valid, up to a point: Even hysteria can be useful, if only to remind us how often even scientists can sound shrill and off-target. Just a quick scan of the chapter titles in each volume gives a rough outline of the authors' theses.

Randy Shilts entitles his sections "Behold, A Pale Horse" (Part I); "The Gathering Darkness" (Part IV); "Battle Lines" (Part V); "Lights and Tunnels" (Part VII), and "The Butcher's Bill" (Part VIII), among others. His use of catchline metaphors indicates a kind of irony, with overtones of doom. The first section begins with a quotation taken from the biblical book of Apocalypse.

Masters and Johnson cover "Breaking Out" (Chapter

1); "Infected Heterosexuals, New Evidence" (Chapter 4); "How Safe Is the Nation's Blood Supply?" (Chapter 5); "Can You Catch AIDS from a Toilet Seat?" (Chapter 6); and "Beyond the Sexual Revolution" (Chapter 8). Clearly, Masters and Johnson are worried; and they want to alarm the reader.

Kübler-Ross discusses the "Ultimate Challenge" in terms of people. Her chapters have such titles as "Working with AIDS Patients" (Chapter 1); "Children and AIDS" (Chapter 3); "Women and AIDS" (Chapter 7); and "Letting Go" (Chapter 8). From the moment she quotes Teilhard de Chardin until her epilogue, Dr Kübler-Ross focuses on the humans and their suffering, and on the religious overtones of the experience.

A closer look will illuminate the values and strengths of each volume.

And the Band Played On. Randy Shilts began covering the AIDS story in San Francisco in 1982, as the first full-time reporter on an "AIDS beat." He has traveled through a dozen countries and conducted hundreds of interviews investigating the story. *And the Band Played On* is the result of his five years' coverage.

Shilts follows the story from several points of view: the politics, the people, and the history of the AIDS epidemic. He begins in 1976, in New York and in Africa, and retells the story of promiscuous male homosexual partying and a new disease striking a physician working in the backwoods of Zaire. Then he skips to 1980, San Francisco, in a section he entitles "Before." Once again Shilts recounts the partying lifestyle of young gay male San Franciscans, but at the same time he reminds us of their political muscle.

He does a very subtle and intriguing bit of writing in depicting the gay lifestyle of his city. His main thesis is that government fails these people—that despite gays' growing political acumen and power, the city, state, and federal authorities felt secure in ignoring them—but Shilts also places blame on the homosexual community itself. By the actions of some irresponsible gay men, by the secretiveness and fear of some of the gay leaders, and by the greed of some of San Francisco's sex-entrepreneurs, the AIDS epidemic was also augmented. The stories of Gaetan Dugas, Paul Popham, Bill Kraus, and others illustrate this theme.

The gay community, Shilts makes clear, is like any other community—diverse and sometimes antagonistic even to segments of itself. When Gaetan Dugas, known by some as "Patient 0," had sex with other gay men, he would sometimes tell them afterwards, "I've got gay cancer. Maybe you'll get it now, too," but he did not stop to examine his behavior. Paul Popham watched his lover die and went on with a gay lifestyle, unaware of the danger he put himself and others in. Bill Kraus worked in the mayor's office in San Francisco, getting zoning variances for gay sex establishments.

The story, of course, is also one of response as well as challenge. And if the initial gay response was poorly conceived, then the governmental response, Shilts feels, was abysmal. The actions of the Centers for Disease Control were slow and fragmented, and the Reagan administration seemed uninterested in the problems of the gay community. Even after Rock Hudson's death, which put AIDS into the mainstream of America's consciousness, money and research and possibly life saving drugs were slow to be released.

It would be easy to recount Shilts' long telling of the story, but it is his point that is important. Toward the end of *And the Band Played On*, Shilts tells of an encounter between Cleve Jones, one of the pioneers of the AIDS Foundation in San Francisco, and Todd Coleman, a beautiful young gay man. They meet on Super Bowl Sunday (a bit of Americana irony), make love, and go their separate ways. A few days later, Jones discovers that Coleman has AIDS. He confronts the younger man and tries to remind him of his responsibilities to his lovers, his community, himself. Coleman replies, " . . . it's important for me to have sex."

As Shilts put it: "Jones felt the tide of despair begin to shift again in his stomach. Everybody, it seemed, was going crazy."

This seems a metaphor for Shilts' feelings about the AIDS epidemic. It seems so clear, so logical that a disease this deadly should evoke a crisis response, practically a Manhattan Project effort to find a cause and a cure. But since AIDS is about three things that the American public feels least comfortable with—sex, death, and homosexuality—the threat has been minimized or ignored entirely. Everybody, indeed, is going crazy.

And the Band Played On is a very disconcerting book to read. But its lessons are vital. The human community is a small world. AIDS will not remain a disease of only a small segment, and even if it did, we must recall the words of John Donne: "No man is an island. Every man is a piece of the continent, a part of the main."

Crisis: Heterosexual Behavior in the Age of AIDS. Masters and Johnson perhaps have read their Donne, also. But while Shilts takes to heart the message of community, they seem to remember a different portion of that famous piece. They "Do not ask for whom the bell tolls" so long as the tolling denotes the passing of a gay man or a drug abuser. But when "the bell tolls for thee" they object. Why else discuss heterosexual behavior nearly exclusively?

It is not hard to understand why Masters and Johnson might want to sound such an alarm if they truly believed (as evidently they do) that the crisis was at hand. But it is exceptionally hard to understand why they believe that the crisis is upon us, given the evidence they present.

In preparing their book, Masters and Johnson chose samples of 800 sexually active adults between the ages of 21 and 40 who met three criteria: they had received no blood transfusions since 1977; they reported no use of illicit drugs via injection; they reported no homosexual or bisexual contact from 1977 on. The 800 adults were evenly split into four cohorts. Two hundred men and 200 women who reported having been in long term monogamous relationships (either marriage or cohabitation) for five years prior to being interviewed were selected. To compare with those groups, Masters and Johnson also selected 200 men and 200 women who reported a minimum of six sexual partners a year over the preceding five years.

These 800 were selected from a group that "completed a self-administered questionnaire" and who were later "interviewed to obtain a detailed sex history covering the preceding five years," wrote Masters and Johnson. "A blood sample taken from each subject was tested for antibody to the AIDS virus," the authors explain, but they did not analyze or double-check the accuracy of the data presented by the subjects.

Furthermore, Masters and Johnson published their data first in book form. They did not give the medical and scientific community a chance to examine and question their results by publishing them in a scientific journal. This omission they blame on lack of time and money. The first excuse sounds once again hysterical, like Chicken Little screaming about the intact sky. The second excuse just sounds specious. That Masters and Johnson announced their findings in a press conference on the day of publication of *Crisis* only increases the cynicism with which one views the work.

> The prevalence of infection with the AIDS virus among the 400 strictly monogamous men and women was, not surprisingly, very low: only 1 out of the 400, or 0.25%, had evidence of such an infection.
>
> In contrast, the prevalence of infection among the 400 men and women with numerous partners was strikingly higher: 14 women (7%) and 10 men (5%).

These facts come from their first presentation of data, on page 9 in the chapter entitled "Breaking Out." They seem to be straightforward enough.

But the "not surprising" one HIV-positive individual in the monogamous 400 really should be surprising, because if all the criteria outlined above were truly met, how did he or she get AIDS?

The choices are limited. We know that AIDS is a blood-borne disease, usually sexually transmitted, but not always. It can be transmitted by contaminated needles used by drug abusers—but the 400 are not supposed to be drug users. It can also be found in contaminated blood used for transfusions, but the 400 all swore that they had not had transfusions for at least nine to ten years.

So how did that one unfortunate individual come down with AIDS? There are no other mechanisms of transmittal. It could, of course, be that this person has been faithful to a spouse or lover who has not been as monogamous, or who has been abusing drugs and sharing needles, and thus transmitted HIV. But the 400 were supposedly 100% monogamous. One question asked of them was, "Have you been completely faithful to your sex partner during the entire time of your relationship?" Another was, "Do you think your partner has been completely faithful to you during the entire time of your relationship?" Masters and Johnson chose their 400 from a much larger group based on these questions.

Or it could be that this individual lied. People do lie, and especially about things like marital infidelity, drug abuse, or homosexuality.

Masters and Johnson do not vouch for the total fidelity of their monogamous 400, so we have to allow for that possibility as the path of transmission in that one case.

But the idea that at least a single individual could have told less than the truth is a disturbing one, especially given the claims Masters and Johnson make for their results. If they have truly eliminated the possibilities now thought of as the usual venues of infection for HIV, then the high incidence they find in the nonmonogamous 400 perhaps does indicate a conspiracy of silence and misinformation from the health officials of this nation.

Estimates are that one in every ten Americans is a drug abuser, and that many Americans males have had some homosexual experiences. These personal life choices are still considered shameful by many—who nonetheless make them, and hide their secret by lying about them.

Let us assume, as apparently Masters and Johnson did not, that the 10% figure is accurate. Then some 40 individuals in 400 might be drug abusers. Some men might be at least occasionally practicing homosexuals or bisexuals. Given the dynamic of current sexual mores, an individual who is homosexual probably has less inclination to lie about it than a drug abuser (who after all is breaking the law) would lie about his or her problem. But although estimates are hard to come by, it's reasonable to assume that at least some homosexuals are still in the closet, say 5%, which would mean ten men in the nonmonogamous 200 are gay. Either way, the 50 individuals who very possibly belong in a "classic" AIDS high-risk group are enough, certainly, to account for the 24 HIV positive cases found by Masters and Johnson without accusing the government of deliberate lying about the AIDS epidemic.

The point is not that Masters and Johnson are just as open to the accusation of bad faith as the government is, but rather that in a crisis such as the AIDS epidemic, crying "wolf!" is a dangerous and shortsighted indulgence.

When the book was published, Masters and Johnson generated a great deal of publicity. Their names alone ensured that response, and the enormity of their claims filled out the notoriety appeal. But much of the publicity was not complimentary. One bit of news that came out when the book was published was that Masters and Johnson had been given $25,000 in seed money by the Ortho Pharmaceutical Company to develop a commercial spermicide that would also kill the AIDS virus.

Dr Masters was asked about his data. Not having, as he himself admitted, a truly random, representational sampling of the American people makes his data suspect for more reasons than those outlined above. Asked why, then, he made his claims for AIDS "breaking out" into the heterosexual community, he asserted, "I simply believe it."

Just as *And the Band Played On* is a disturbing book, so is *Crisis*, but for very different reasons. Whether Masters and Johnson can be accused of creating panic to further any sales of a potential anti-AIDS product is really not the question. But why they would make such claims without having their data peer-reviewed or even releasing the names of the laboratories that did the testing is a disservice to science itself. And finally, the underlying homophobic attitude they take detracts from the serious message they could deliver from the vantage point of their worldwide renown. They write, "Thus the implications of the spread of the AIDS virus into the general population are frightening. As others have said, the AIDS epidemic carries the potential to be the greatest natural tragedy in human history" (p 11). But AIDS already is a disaster for those now dying. To ignore them is to be callous in extremis.

AIDS, the Ultimate Challenge. Elisabeth Kübler-Ross does not espouse a point of view that makes a stand or carves out turf. She seems to say, "We are all in this together" while Randy Shilts says, "Why have you pushed us away to die in silence and exile?" and Masters and Johnson scream, "We're all going to die and it's somebody's fault!"

In her book, Kübler-Ross talks about the people with AIDS that she has known, and she has met many at her death and dying workshops in the past six years. It is to Kübler-Ross that people with AIDS and their relatives have found they can turn. "Someone whose next of kin is dying of AIDS may desperately need to lean his head on a shoulder, but he will not even get as much as a handshake out of fear that the disease can be spread this way," she writes. Her job, she tells us, is to provide that shoulder.

Elisabeth Kübler-Ross is a profoundly spiritual person, and her calling dovetails with her spirituality. When she thinks about people with AIDS she comes to this conclusion: "Over the next ten years, as changes take place on Earth, those who choose love *now* will have nothing to fear. They will be spiritually uplifted and above all the turmoil. Those who stick to their old negative, judgmental ways will blame everybody and everything and will be too blind to see the destiny they have created for themselves."

Not everyone will be comfortable with her vision of AIDS as a trial by the Lord of we puny humans. But no loving human being can fail to see that Elisabeth Kübler-Ross is doing a saint-like job working with the dying and the sick. In her book she recounts her difficulties in getting an AIDS hospice started, and her interactions with the citizens, AIDS patients, physicians, health care workers, and politicians who called for the home or who opposed it. She shows how she works with people with AIDS—and she never classifies them nor quantifies them as gays, drug abusers or others. To Kübler-Ross, they are all human souls, and therefore worthy of love and respect.

After reading a work that shakes one's faith in the social institutions the way *And the Band Played On* does, or after the slightly distasteful effect of *Crisis*, the calm serenity of the acceptance of death as part of humanity can be very comforting. Kübler-Ross does not offer the hope of cures, nor even seem to long for them—which may be the only weakness of her book. She accepts AIDS perhaps too stoically, as a challenge from God to rise above our earthly pretensions.

Shilts, certainly, would have little sympathy for this view. And his call for more action may be the truest human response. We must try to relieve the sufferings of our fellows through science, if possible. The meaning of life is life, after all: The strongest drive in man is not the drive for sex but the drive for survival.

But Kübler-Ross's philosophy, that love itself is a human drive that must be acceded to, is a deep wellspring of hope in a time of plague. "What are we to make of this hell of AIDS?" she writes in her epilogue.

> Let us look again at the prophesies, from the Holy Scriptures, to the Hopi Indians, to Nostradamus. It has been foretold that there will be a time of great plague; there will be a time of the separation of the wheat from the chaff prior to great changes on this planet earth. We have also been taught, over endless time, that *love* is always stronger than anything else and can literally conquer all evil. If we are to believe all of this, wouldn't it be simple to spend all our joint energies and resources (on every level) to organize a worldwide team, not only for research but for care centers, support groups, treatment centers, counseling centers, and bereavement groups?"

Perhaps we cannot all believe that AIDS is the plague of biblical prophesy, but surely we ought to be able to see that the actions outlined by Kübler-Ross in that short paragraph are the morally, socially, and scientifically correct way to proceed. Love, with a good leavening of common sense and the resources of a rich and powerful nation behind it, just may conquer all.

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Tetracycline-resistant *Neisseria gonorrhoeae*

Gonococcal resistance to antibiotics has been well documented over the past 40 years. Sulfonamides, the first antibiotics used to combat gonorrhea, were rendered almost useless by the end of the 1940s due to development of gonococcal resistance. When penicillin first became available, virtually all gonococcal strains were susceptible to this new antibiotic. As resistance to penicillin emerged, it was first countered by use of progressively increased dosages and eventually, when the first approach was no longer practical, by use of other antibiotics. This ability of the gonococcus to protect itself so effectively from eradication comes about by resistance which is either plasmid mediated, chromosomally mediated or both. Penicillinase producing *Neisseria gonorrhoeae* (PPNG) is a major example of plasmid mediated resistance wherein the bacteria gain a plasmid that produces an enzyme (beta-lactamase) that destroys penicillin. In 1983, chromosomally mediated resistant *N gonorrhoeae* (CMRNG) was documented. In this instance, beta-lactamase is not produced but rather reduced susceptibility to penicillin comes about through nonspecific mutations. Whereas PPNG strains are absolutely resistant to penicillin, CMRNG typically display varying degrees of decreased sensitivity.

As a response to the decreased usefulness of penicillin to cure gonorrhea, other antibiotics have been introduced for treatment including tetracyclines, ceftriaxone and spectinomycin. Of these, tetracycline is the least expensive and also has the advantage of treating concomitant *Chlamydia* infection. Centers for Disease Control (CDC) guidelines recommend dual treatment with penicillin and tetracycline but in many cases this has been abbreviated into treatment by tetracycline only. Such an approach is definitely not appropriate, as the following report by Dr Menegus and colleagues emphasizes. This detection of a rapid increase of tetracycline-resistant *Neisseria gonorrhoeae* (TRNG) in Monroe County was originally reported in the *Clinical Microbiology Newsletter* of Strong Memorial Hospital, Rochester. This study demonstrates that dramatic changes in the local prevalence of TRNG can occur quickly and emphasizes the need to discourage the use of tetracycline monotherapy for gonorrhea.

An Outbreak Study

"An Outbreak of Tetracycline-Resistant *Neisseria gonorrhoeae* in Monroe County." M.A. Menegus, F.S. Nolte, M. Nasello and J. Knapp. University of Rochester Medical Center, Rochester, and Centers for Disease Control, Atlanta.

"*N gonorrhoeae* strains with high level resistance to tetracycline (TRNG) were first described in 1985.[1] This form of tetracycline resistance is due to the insertion of a resistance determinant, *tet* M, into the gonococcal conjugative plasmid.[2] Screening *N gonorrhoeae* for resistance to drugs other than penicillin is not routine, but to develop some data for our community, we examined all strains of *N gonorrhoeae* isolated in our laboratory during June 1986 and June 1987 for high level tetracycline resistance as well as other resistance phenotypes. The prevalences of TRNG were 0.6% and 1.4% in 1986 and 1987, respectively.

"Over 100 strains were tested in each year. However, in September 1987, we began isolating TRNG from a number of apparent treatment failures. This observation suggested an increase in the prevalence of TRNG and prompted routine screening of all *N gonorrhoeae* for high level tetracycline resistance.

"From 12/28/87 to 1/31/88, we screened 168 isolates and found 35 (21%) TRNG. This is a surprisingly high prevalence when one considers that just six months ago only 1.4% of our isolates were TRNG. Also, national figures (8/86–7/87) reported recently in *MMWR* suggest lower prevalence rates (range 0–7.9%, mean 2%) in most US cities. The prevalence of TRNG exceeded 10% in only one of 15 cities participating in the National Surveillance Project, Baltimore, where the rate was 15%.[3]

"Because of the apparent high prevalence of TRNG in our community, the Monroe County Department of Health has recommended that practitioners *not use tetracycline alone for gonorrhea infection.* In the past, tetracycline monotherapy was recommended as an option because it was inexpensive and treated both gonococcal and chlamydial disease. However, with the emergence of TRNG, single-agent therapy with tetracycline is now considered imprudent. In its 1987 guidelines for the management and control of gonococcal disease, CDC discourages the use of tetracycline monotherapy and recommends several alternatives for penicillin allergic patients.[4]

"To aid in therapeutic decisions for gonococcal infections, Strong Memorial laboratory will soon begin reporting the results of screening for high level tetracycline resistance in addition to the high level resistance to penicillin associated with beta-lactamase production."

Guidelines

New York State Department of Health guidelines for adult treatment of uncomplicated gonococcal infection and for PPNG are listed below. For further information and for treatment schedules for other gonococcal infections, eg, ophthalmia, disseminated gonorrhea, gonorrhea in pregnancy, consult the Sexually Transmitted Diseases Treatment Guidelines, 1986, which is available from the New York State Department of Health, Bureau of Communicable Disease Control, Sexually Transmitted Disease Control Program, Room 649, Corning Tower, Empire State Plaza, Albany, NY 12237.

Treatment Guidelines

1. Uncomplicated urethral, endocervical, or rectal infection: Amoxicillin 3.0 g or ampicillin 3.5 g by mouth OR aqueous procaine penicillin G (APPG) 4.8 million units IM OR ceftriaxone 250 mg IM. Amoxicillin, ampicillin, and penicillin (but not ceftriaxone) are accompanied by probenecid 1.0 g by mouth.

Comment: APPG may be less desirable because of associated pain and toxicity.

Plus

Tetracycline HCl 500 mg by mouth four times daily for seven days OR doxycycline 100 mg by mouth twice daily for seven days

Or

For patients in whom tetracyclines are contraindicated or not tolerated, the single-dose regimen may be followed by erythromycin base or stearate 500 mg by mouth four times daily for seven days OR erythromycin ethylsuccinate 800 mg by mouth four times daily for seven days.

2. Penicillin-resistant *Neisseria gonorrhoeae*.

Penicillinase-Producing Neisseria gonorrhoeae (*PPNG*). Patients with proven PPNG infection or who are likely to have acquired gonorrhea in areas of high PPNG prevalence and their sex partners should receive spectinomycin 2.0 g IM, OR ceftriaxone 250 mg IM, both followed by tetracycline OR doxycycline, OR erythromycin as outlined above. To treat pharyngeal gonococcal infection due to PPNG: Ceftriaxone 250 mg IM OR nine tablets of trimethoprim/sulfamethoxazole (720 mg/3600 mg) per day in one daily dose for five days.

Chromosomally Mediated Resistant Neisseria gonorrhoeae. Patients who fail standard treatment for gonorrhea or who have infection with penicillin-resistant strains that do not produce beta-lactamase (CMRNG) should be treated with spectinomycin 2.0 g IM or ceftriaxone 250 mg IM.

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Cancer mortality among Native Americans in New York State

Deviations in cancer mortality among specific population subgroups may be overlooked due to masking of differences within the more homogeneous general population. Therefore, it becomes important to consider patterns of cancer mortality among defined racial groups.

Based on the 1980 US Census enumeration,[1] New York State ranks tenth with regard to Native American populations. However, since most Native American groups reside in the southwestern United States, New York State ranks second in total Native population among states on the east coast, exceeded only by the Native population in North Carolina. This sizable Native American population makes New York State well suited for studying patterns of mortality among this group.

Two studies, a standardized proportional mortality study[2] and a retrospective cohort study,[3] have examined mortality patterns among Native American groups in New York State between 1955 and 1984. Both of these studies were restricted to a single Native American tribe which represents less than 20% of the total Native American population in New York State. This Native group, the Seneca Nation of Indians, is located in western New York State. A third study, a standardized proportional mortality study,[4] examined the mortality experience of all Native Americans in New York State, exclusive of New York City, between 1980 and 1986. In each of these studies, mortality among Native groups was compared with sex, age, cause, and calendar year specific mortality of the general population in New York State, exclusive of New York City. Analytic procedures adjusted for any differences in the age and sex distribution between the study group and the general population of New York State, exclusive of New York City, allowing an assessment of mortality patterns.

Overall, cancer mortality among both Native males and females was significantly low in each of the studies.[2–4] This observation is consistent with diminished cancer mortality reported among Native American males in other parts of the United States[5,6] and in Canada.[7–9] Lower cancer mortality has also been reported among Native females in the United States[5,6] and in Canada.[8]

A deficit of lung cancer deaths was observed among male Native Americans in New York State.[2,4] Previously published studies of Native males[5–10] have also reported lung cancer mortality deficits. Decreased lung cancer mortality has also been observed among Native females,[6,10] however, other studies[5,7–9] and investigations[2–4] have shown no variation for this cause of death. These deficits may reflect a lower prevalence of smoking among these groups.[11,12]

All of the New York State studies have reported a significant deficit of breast cancer deaths among Native females.[2–4] This is consistent with low breast cancer mortality reported among other Native female populations.[5,6,8] Lower breast cancer mortality is likely associated with lower breast cancer incidence attributable to both earlier age at first pregnancy and higher fertility patterns reported among Native American females.[13]

Deficits in colon cancer mortality were noted among both Native males and females in New York State between 1980 and 1986.[4] While other studies of Native groups in New York State[2,3] did not identify a deficit of deaths due to this cause, deficits of colon cancer deaths have been reported among Native populations in other areas.[5,6,8,9]

A deficit of pancreatic cancer deaths was observed only among Native females in New York State.[2,3] This mortality deficit has been previously described among Indian females in Canada,[9] however, excess pancreatic cancer mortality has been reported among Native females in New Mexico.[5,6]

An excess of cervical cancer deaths has been reported among Native females in New York State.[4] While this excess may be partially attributable to underutilization of cervical cytology screening programs,[9-12] knowledge of cervical screening behavior was unavailable.

In summary, studies of Native American populations in New York State are consistent in reporting a deficit of cancer deaths among both males and females for all sites combined.[2-4] Among Native males, site-specific deficits have been reported for colon[4] and lung[4] cancer deaths and for deaths due to lymphatic/hemopoietic malignancies.[2] Native females in the state have demonstrated site-specific deficits in mortality due to cancers of the colon,[4] pancreas,[2,3] and breast,[2-4] and elevated cervical cancer mortality.[3]

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Sporotrichosis outbreak

Sporotrichosis is a fungal infection caused by a microscopic fungus called *Sporothrix schenckii*. The disease generally presents as single or multiple skin nodules which ulcerate and may persist for months. Treatment may consist of iodides for the lymphocutaneous form of infection; in other forms, amphotericin B is administered.[1]

Sporotrichosis is an occupational illness that tends to occur among people in the horticultural industry, such as tree nurserymen, rose gardeners and greenhouse workers. Sphagnum moss has been identified a number of times as a vehicle for the fungus.

In May 1988, an outbreak of cutaneous sporotrichosis was reported to the New York State Department of Health, Bureau of Communicable Disease Control (BCDC), which occurred among various county soil and water district workers and some members of the public who handled pine tree seedlings packed in sphagnum moss harvested from Wisconsin. Investigation by BCDC staff identified one group of 107 workers who handled the seedlings and moss between April 11 and 28, 1988. Of them, 13 clinically confirmed cases of sporotrichosis were identified with onset of symptoms between May 1 and June 1, 1988. *Sporothrix schenkii* was cultured from seven ill workers as well as from five samples of the implicated moss.

Following this incident, a letter describing the outbreak and a fact sheet on sporotrichosis were mailed to 12,000 people who had purchased pine tree seedlings from various county soil and water district offices as part of the state's spring reforestation program. To date, 12 people have contacted the New York State Department of Health describing symptoms consistent with sporotrichosis—six have been culture confirmed.

A literature review revealed that four previous outbreaks associated with Wisconsin sphagnum moss have been reported since 1965. Sporadic cases associated with sphagnum moss have also been reported. Nurseries in 16 states including Alabama, Delaware, Florida, Illinois, New Jersey, New York, Ohio, Pennsylvania, Rhode Island, Tennessee and Texas are known to have received shipments of sphagnum moss from Wisconsin during the months May through July.

Other state health departments are currently evaluating whether this problem exists in their states and are notifying the public of the occupational as well as avocational risks of handling pine tree seedlings packed in sphagnum moss and the precautions which should be taken. Due to the lack of physician familiarity with this disease, case definition may be important to ensure proper diagnosis and appropriate treatment.[2]

In an effort to assess the national scope of this outbreak, representatives from the Centers for Disease Control have expanded the investigation to other states. The New York State Department of Health, Bureau of Communicable Disease Control, would appreciate notification by any local health unit that identifies cases of sporotrichosis associated with sphagnum moss. Contact Stan Kondracki or Dr Bruce Coles, Bureau of Communicable Disease Control, at (518) 474-3186. Copies of a fact sheet on sporotrichosis are available on request.

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Playground injuries

Public and home playground equipment accounted for approximately 177,000 medically attended injuries in the United States in 1981, for an overall rate of 83 per 100,000 population.[1] The highest rates were in children ages 0-4 (327 per 100,000) and 5-9 (302 per 100,000). Male rates exceeded those for females. Falls were the most frequent hazard reported, accounting for approximately 47% of home and 72% of public playground-related injuries. The high incidence of these injuries places playground equipment fifth on the list of 100 hazardous consumer products from the US Consumer Product Safety Commission.[2]

Most federal reports of injury relate primarily to studies of public playgrounds.[3,4] The type of surface on the playground was a major factor affecting the number and severity of injuries. Both paved and natural surfaces had higher proportions of severe injuries (fractures, dislocations, strains/sprains, concussions and internal injuries) than protective surfaces including sand, gravel, rubber matting or wood chips.

Analysis of the percentage of most common public playground equipment-related injuries by the manner in which they occurred showed 59% from falls to surface and 11% falls—struck some piece of equipment. Other categories were: impact with moving equipment (7%), falls against/onto or into stationary equipment (8%), contact with protrusions, pinch points and sharp edges and points (5%) and unknown/other (9%).[4]

New York State pilot demonstration projects have helped guide the development of preventive programs. Following a child playground equipment injury prevention project in New York State in 1977 which trained playground supervisors to identify and correct hazards in public playgrounds, accompanied by a public awareness media campaign, there was a 22.4% reduction in playground-related injuries treated in the two largest hospitals in one of the program areas. Knowledge about unsafe playground practices and equipment hazards improved after the workshops for playground personnel.[5] This approach to injury prevention deserves further application.

Preventive actions can include:

1. Identification of Existing Problems and Dangers. It is essential that play settings and equipment be carefully scrutinized for hazards. Surface materials such as asphalt and concrete are especially dangerous. Wood equipment should be checked for rotting and splintering. Metal equipment should be checked for sharp edges, rusting, exposed bolts and screws. Ropes must be sound and show no evidence of fraying. All equipment should be evaluated for stability. Concrete footings should be checked for cracking.

2. Correction and Adjustment in Standard Play Settings and Equipment. A safely designed playground must have a carefully planned circulation system to and around equipment. The playground should have a clearly defined ball or running game area, a play area for younger children separate from that used by older ones. Corrections to an existing playground may require reorganization of the entire play setting. Adjustments to equipment might include replacement of plank swing seats with rubber or canvas; replacing concrete and asphalt surfaces with sand, woodchips, cocoa shell, pinebark, pea gravel, shredded tires or thick rubber mats. Dangerous equipment should be removed. Playgrounds should have constant supervision of children and regular inspection of equipment and surface conditions.

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WOMEN'S WORK, FAMILIES, AND HEALTH: THE BALANCING ACT

Edited by Kristen M. Swanson-Kauffman, RN. 98 pp, illustrated. New York, Hemisphere Publishing Corp, 1987. $24.50 (hardcover)

As a woman balancing the multiple roles of physician, wife, mother and daughter, I looked forward to reviewing Kristen M. Swanson-Kauffman's *Women's Work, Families, and Health: The Balancing Act*. The volume is a compilation of papers presented at a symposium of the Western Society for Research in Nursing at the University of Washington in Seattle. The main theme centers around the juggling of commitments related to parenting and working for women today.

Each chapter summarizes research efforts presented by individual authors, all of whom, including Dr Swanson-Kauffman, are registered nurses with advanced doctoral degrees. Editor Kristen Swanson-Kauffman's enthusiasm for this project is obvious in her introductory chapter, "Overview of the Balancing Act: Having It All." The chapters all open with a succinct abstract and present the contents and conclusions of the research in easily read text with clearly presented tables and graphs and extensive reference lists. Topics examined include "Women's Lives: Pressure and Pleasure, Conflict and Support," "Childbearing Choices of Professional Women," and "Work and Family Roles of Women." Other topics include "Social Support and Parental Adaptation" and "Infertility and Its Influence on Women's Careers."

A particularly pertinent chapter by Marie Annette Brown, ARNP, PhD, examines employment during pregnancy. This study compared employed and unemployed women in their experiences of support and health during pregnancy in a sample of 313 adult females. Findings suggest that working is in no way detrimental, and that employed women may experience a better support system and a better sense of well-being while pregnant.

The research appears well documented, with large samples and adequate controls. Statistical analyses were performed where indicated to examine the significance of differences between categories studied. There is little clinical or anecdotal material which at times makes the reading a little dry. This work differs from similar publications in its emphasis on the interaction of women's work and families with the health care field, particularly nursing. The importance and relevance of this work lies in the insight it provides into the delicate balancing act that so many women are handling so successfully in today's society. The information is pertinent to health care providers, employers, and to all women balancing multiple roles.

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HEMATOLOGIC PROBLEMS IN PREGNANCY

Edited by David Z. Kitay, MD. 420 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $39.95 (softcover)

In an era of unprecedented bibliographic proliferation, one can hardly look forward to yet another textbook on the shelf. However, when it comes to the subject of blood disorders in pregnancy, one is struck by the sparsity of references on the subject. Indeed, a search of the National Library of Medicine CATLINE did not reveal any text addressing a similar topic. Perhaps this is the reason why the authors have introduced this text and called it a timely book, the purpose of which is to provide a practical organization of the most important hematologic problems confronting the obstetrician. It is also presented by the authors as a benchmark so that 20 years from now another generation of obstetric hematologists may assess the progress which has been made in the intervening years.

The authors appear to have accumulated a fair amount of experience in the hematologic aspects of pregnancy and spent more than four years preparing the text. However, there is a paucity of authors who by their titles are in the hematologic arena, only six of 33. Of these six, one is in blood banking, one is in pediatric hematology, and the remainder are practicing adult hematology. All have made a sincere effort to write a comprehensive text. The contents of the book cover most of the hematologic disorders encountered in pregnancy. The individual chapters end with a summary and often an editor's note. The book is replete with illustrations including tables and diagrams, and includes long lists of references. Anemias, which are believed to be the most common complication of pregnancy, are discussed with a fair amount of detail; iron deficiency is well introduced and covered exhaustively, but unfortunately the discussion displays occasional errors. A registered dietitian coauthored the section on nutrition, and in the chapter on genetics, the authors attempt to simplify and bring down to earth a rather complex subject. The hematologist managing a pregnant patient with leukemia or lymphoma may draw some help from this book but will probably be looking for more specific information concerning the implications of chemotherapy to mother and fetus. The section on blood banking and isoimmunization is exceptionally well written, serving as a model for other authors approaching this topic. It conveys practical recommendations to physicians interacting with the blood bank. Coagulation disorders are adequately presented and enriched with good illustrations; but in managing disseminated intravascular coagulation in pregnancy, the reader should be warned about the danger of using epsilon aminocaproic acid. In the management of acute thrombotic thrombocytopenic purpura, there should be more emphasis on the details of plasma infusion and plasma exchange. Administering plasma infusion every other day, as stated in the text, is often inadequate.

Is this a timely book? Indeed it is. Will it achieve its purpose as a "bedside consultant" and "useful reference?" Only physicians utilizing the text can answer that appropriately. Its title makes it unique, and the price invites procurement of the text. It should not be the sole reference, especially in some areas of management, and some of its chapters need revision. All things considered, the book should be within an arm's reach of the physician who will use it not only as a guide to management, but also as a gate to a large list of references.

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WOMEN AND CANCER

Edited by Steven D. Stellman, PhD. 277 pp, illustrated. New York, The Ha-worth Press, Inc, 1987. $39.95 (hardcover)

Cancer, the second leading killer of Americans, has characteristics that uniquely affect women. Breast cancer is epidemic in this country, for example, and the risk of cervical cancer is in part determined by women's sexual behavior. Therefore, the title, *Women and Cancer*, offers auspicious promise. Sadly, this volume is an uneven attempt to meet that promise.

Steven Stellman has edited a collection of 17 "research papers, essays and 'thought pieces,'" organized in five sections: basic statistics (epidemiology—3 papers), screening and early detection (3 papers), implications of treatment and its aftermath (5 papers), risks and benefits (including ethics, 2 papers), and tobacco (4 papers).

From the National Cancer Institute's (NCI) Biostatistics Branch, Susan Devesa's piece begins the volume with a comprehensive summary list of cancer statistics for American women. Unfortunately, as these articles initially appeared as volume 11 (Fall/Winter 1986) of *Women and Health*, much of the data is outdated, running through 1979 or 1980 as compiled in 1984 or 1985. Many of us who view the evening news are more current. The next five pieces in this volume would disappoint the casual reader hoping for a broad overview of this topic; they constitute formal research pieces of relatively limited interest. Only the hardy would venture beyond.

This, however, would be a shame. The remainder of the volume, while not comprehensive, is largely informative. The "therapeutics" section commences with a poignant piece by Grace Ulbricht detailing the concerns of women who have undergone laryngectomies. Frances Feldman raises alarming concerns about cancer victims' and their caregivers' experiences in the workplace. Jana Hach and Sarah Wilson review the development and current status of the hospice movement in this country. And Helene Brown concisely reviews the risks of and methods for recognizing and helping patients deal with unproven cancer therapies ("quackery").

The "risks and benefits" papers are again mixed. Elizabeth Barrett-Connor provides a thoughtful and comprehensive assessment of the pros and cons of postmenopausal estrogen with or without progesterone replacement for osteoporosis prophylaxis. This, however, is also dated—the large *New England Journal of Medicine* study on birth control pills and the risk of breast cancer appeared subsequent to this article. Unfortunately, Colin Soskalne's piece on the ethics of the use of human subjects in cancer studies, while raising salient points, is unduly wordy and tedious.

Where this volume truly shines is in the four pieces on smoking. As a group, they offer a comprehensive review of the topic and excellent information on the history and statistics of this health hazard, potentially useful to those clinicians attempting to aid their patients in dealing with this risky habit. With lung cancer deaths surpassing those of breast cancer, in spite of the 1/10 lifetime risk of the latter disease, this section unfortunately remains sadly appropriate.

With all but two of the included papers having been invited, one would wish that the entire volume conformed to the standard that the majority of the "thought pieces" exhibit. As it stands, the volume largely fails as a significant addition to the clinical oncologist's library, although social scientists and primary care physicians may be largely pleased with its contents.

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LEARNING ELECTROCARDIOGRAPHY: A COMPLETE COURSE

Third Edition. By Jules Constant, MD. 640 pp, illustrated. Boston, Little, Brown & Co, 1987. $48.00 (clothbound)

It is an honor to be asked to review the newest edition of Jules Constant's book, *Learning Electrocardiography*. Dr Constant is a world renowned expert in electrocardiography, and well known for his previous editions of this authoritative book. He is Associate Clinical Professor of Medicine, State University of New York at Buffalo School of Medicine; his electrocardiographic expertise and teaching skill are widely known by all practitioners of this art.

As a "complete course," the book begins with brief chapters on cardiac anatomy and physiology, electrical activity of the heart, and another brief chapter on "How Electrocardiography Works." This is followed by several chapters dealing with lead nomenclature and conventions, frontal plane axes, dipole concepts, and the like. There is a very nice chapter on "How to Produce a Good ECG Tracing."

The main body of the text is devoted to recognition and diagnosis of standard ECG waveforms. This is followed by two chapters dealing with arrhythmias, less than 25% of the text, and two brief chapters on electronic pacemakers and His bundle recordings. There is a very brief chapter entitled "Systematic Approach to Reading an Electrocardiogram," and a relatively brief index.

Two stylistic features used by the author may affect the book's usefulness. First, the entire text is written using the so-called "Socratic" method, with questions posed by the author followed by the answer. At times, this device works well. At other times, it does not. Example: "If concealed VA conduction from an interpolated PVC prolongs the subsequent P-D interval, what does it do to the R-R interval of the sinus beats surrounding the PVC; i.e., what happens to the distance between the R waves preceding and following the PVC?" Aside from the implausibility of such rhetorical questions, the style has another adverse effect. When one attempts to use the book as a reference source, one has to find the right Socratic question and then hope that the answer applies to one's search. If one does not ask the same question as the author, one may be out of luck.

There is another question of style which, in this reviewer's opinion, adversely affects the book. Nearly all the ECG examples are printed with the limb leads distributed in a circle around the hexaxial coordinate and the precordial leads printed at the bottom. Although some authors prefer this display pattern for teaching beginners, it is not the format in which most ECGs are viewed in the everyday world. One might get used to such a display; however, this reviewer found it annoying and unnecessary. It is not a format to which most of us are accustomed, and it takes a few seconds to find the lead one is looking for when they are so printed.

The beauty of this book lies in the author's insightful, clearly written, and concise comments on many standard ECG waveform features. For example, his commentary on terminal QRS changes in myocardial infarction is illuminating. The author's long experience in clinical electrocardiography shows in a number of areas, where he inserts comments such as "this is seldom seen in practice." The author is clearly an experienced and authoritative expert.

There are several areas in which the reviewer might disagree with the author. Statements are made in the book which are not universally accepted. For example, the author states that the normal range of frontal plane QRS axis is from 0° to +90°, whereas most electrocardiographers accept −30° to +90° as the normal adult range. Other similar examples appear throughout the book. Many of the commentaries are dated. For example, as much space is devoted to the Master two-step exercise test as is devoted to treadmill testing, and the treadmill exercise test discussion is not very complete. The chapter on electronic pacemakers is dated, and is really insufficient even for a fourth-year medical student. Many of the references date back to the "golden age" of electrocardiography in the 40s and 50s, although there are some recent references.

The index calls for special comment. It is not only too brief, but it is not very useful and is misleading. For example, the page numbers in the index may refer to an electrocardiogram, with the indexed word appearing nowhere on that page. Those page numbers in italics are said to refer to electrocardiograms, but the reviewer often found it impossible to distinguish which page numbers were in italics and which were not.

With these qualifications and reservations, it is difficult to put this book into perspective as an addition to the medical literature. I believe that for medical students, house staff, and cardiology fellows, the standard electrocardiographic texts of Chung, Friedman, and Marriott are much to be preferred. The Constant book has too many drawbacks and limitations to its use as a reference source. However, this reviewer found it fascinating to read the book for protracted periods, enjoying Dr Constant's insightful descriptions and clear prose. I personally have improved my knowledge of electrocardiography by reviewing this book, and have spent several pleasurable hours reading it. The book can be enthusiastically recommended as a supplement to one of the standard texts described above. The limitations described above prevent its enthusiastic recommendation as a sole source, or as a "complete course" sufficient by itself.

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UROLOGY: PROBLEMS IN PRIMARY CARE

By Elroy D. Kursh, MD, and Martin I. Resnick, MD. 344 pp. Oradell, NJ, Medical Economics Books, 1987. $32.95 (softcover)

As indicated by the title, this is a work intended for primary care physicians and medical trainees, as a quick, "first-hand" reference for most of the urologic problems they may encounter. The editors are from Cleveland, Ohio, at Case Western Reserve University, where Dr Resnick is Chairman of Urology and Dr Kursh is Associate Professor. They have authored numerous publications and made significant contributions to urology. Dr Resnick has done pioneering work in stone disease and its treatment and is an active and well known researcher and educator. The other nine contributors are well qualified and represent Case Western Reserve, the Cleveland Clinic, Brown University, and Northeastern Ohio School of Medicine.

The book is divided into four sections: "Assessment," "Adult Urology," "Pediatric Urology," and "Urologic Emergencies." Each part contains various sections from four to eight pages in length, each dealing with specific urologic problems. The first section covers urologic anatomy, history, laboratory investigation, and diagnostic imaging. The section on adults covers such topics as hematuria, renal masses, stones, infections, malignancies, infertility, impotence, skin conditions, and others. Pediatric topics such as vesicoureteral reflux, undescended testes, congenital anomalies (especially hypospadias and urethral valves), malignancies, and more are included. The part on emergencies covers scrotal pain, urinary retention, urosepsis, and trauma. Despite the small size of the book it covers the entire spectrum of urology.

The subjects are presented in a clear and logical manner. Each author adheres to the strict outline: clinical significance, signs and symptoms, laboratory tests, diagnosis, and management. Particularly pertinent is the end of each section: consultation timing and patient education. It makes it quick and easy to find and to read about a particular question, right in the office or emergency room. At times, details are sacrificed for brevity. This is actually the aim of the work and is done in a careful manner so that pertinent information for the primary care provider is emphasized. The information about carcinoma of the prostate is a good example. It is a controversial topic that is debated widely. In a short space, Dr Kursh gives the salient features and clear, straightforward advice.

An additional strength of this book is that it is up to date. There have been changes in urology in recent times that range from minor to revolutionary. The management of stone disease, impotence, surgery for carcinoma of the prostate, chemotherapy of some tumors, much of pediatric urology, and other conditions are well covered.

This is an enjoyable book and it is heartily recommended for the shelf of any primary care physician, pediatrician, nurse practitioner, physician's assistant, or emergency room specialist.

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VIETNAM: A CASEBOOK

By Jacob D. Lindy, MD. 353 pp, illustrated. New York, Brunner/Mazel Publishers, 1987. $40.00 (hardcover)

This is the tenth book in the Brunner/Mazel psychosocial stress series, which focuses on the immediate and long term psychosocial consequences of extraordinary stressors including war, divorce, parenting, separation, social isolation, acute illness, drug addiction, death, sudden unemployment, rape, natural disasters, crime victimization, and others.

Vietnam: A Case Book was developed and written through a collaborative effort by Jacob D. Lindy, MD, who is an internationally known research psychiatrist; a team of skilled clinicians and researchers in the area of post-traumatic stress; and the patients, veterans of the Vietnam War, who were being treated for post-traumatic stress disorders. Dr Lindy has served as consultant to survivors of a wide variety of highly stressful incidents including the 1974 Buffalo Creek Dam break, the West Philadelphia (MOVE) bombing, and a number of other post-traumatic stress studies in which the University of Cincinnati's Traumatic Stress Studies Center has been involved.

This book provides a unique view for clinicians, researchers, and other health care providers who care for the psychological needs of the survivors of catastrophic experiences. It is a comprehensive presentation of the story of the therapeutic dyad involved in the process of comprehending, communicating, and mutually understanding the many problems involved with patients who have survived a catastrophic experience such as war. The book is also a scientific presentation which sets out to measure differing components of post-traumatic stress and the relative efficacy of treatments for this disorder. The unique way this book weaves together the perspectives of the patient, the therapist, and the scientist allows us to explore the historical military events of the past and understand the outcome of the therapeutic intervention for these patients.

The book is divided into three parts. The first portion discusses the clinical case studies themselves. The second provides a clinical discussion involving the veteran and therapist relationship. The third section examines the research component, detailing guidelines and instruments utilized for each of the clients. Carefully woven into each of the presentations is a historical perspective on the veterans' role in the Vietnam War. Each portion of this book allows us to hear from the veteran, the therapist, and the researcher, providing a first-person viewpoint regarding the interactions and expectations of therapists and clients. In the clinical case studies, each of the seven veterans is represented both while in Vietnam and during their psychotherapy sessions. This case book makes a very strong attempt at allowing us to understand, through the veterans' own words, their images, and even poetry reflecting their experiences. This results in an almost existential perspective as the therapist reveals to us through the first person singular the veterans' thoughts and reflections.

The veteran/therapist relationship is explained in part two of the book, in which the clinical aspect of the working alliance between veteran and therapist is examined. Issues such as confidentiality, trust, self-disclosure, ethics, and concern are all carefully reviewed from the perspective of each. As one veteran says, responding is "almost as if [my doctor] were by my side and we were digging a foxhole together. I would go down a certain distance, and he would go down even further, so I would need to go deeper, and slowly proceed going deeper than I ever imagined. It was as if we weren't afraid together facing any of it until we were at the bottom; then, with his help I could climb out."

The last portion of the book addresses research issues in a treatment project through the combined narrative voice of the University of Cincinnati's Trauma Stress Center Group. This section discusses the instruments used for post-traumatic stress disorders and those newly created that have been developed specifically for studies at the Cincinnati Stress Center.

This case book allows for an inside, almost *in vivo* view, of how the veteran, therapist, and researcher contribute to the knowledge that we currently have regarding the process of recovery for people who have survived a catastrophic event. During this process, the therapist is utilized as a model for pacing, concern, and acceptance. He becomes a real figure whose attitudes can be differentiated from the negative experiences which the veteran had with the same authority figures during and after the Vietnam War. If ever there were a textbook which allows us to break the wall of silence between patient and therapist, this certainly is an example. The veteran helps others to help him understand his own traumatic event and its sequelae.

This is a useful textbook for any clinician, researcher, or other practitioner who may be caring for those with post-traumatic stress disorders. Certainly, it will assist in the ongoing effort to understand what happens to survivors of a catastrophic event. It offers us an inside historical view of the veterans of the Vietnam War, their aftermath, and the healthy environment that is necessary for them to recover from the ill effects they suffered. It does not make excuses; it does not provide all the answers. It looks at the issues clearly, concisely, and openly.

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AN ACCOUNT OF BELLEVUE HOSPITAL

Edited by Robert J. Carlisle, MD. 381 pp, illustrated. New York, The Society of the Alumni of Bellevue Hospital, 1983. $17.50 (hardcover)

Bellevue Hospital began as an almshouse in 1736 and has faithfully served the City of New York as a penitentiary, mental institution, alcoholic treatment center, and general hospital for two and a half centuries. It survived many changes and emerged as a world-famous municipal hospital, an integral part of an outstanding medical school, and an excellent teaching center. This book, like a family Bible, tells its story. The original manuscript, written in 1873, was updated and published in 1893 by the Society of the Alumni of Bellevue Hospital. In 1986 this edition was reprinted as part of the celebration of the Society's 100th anniversary and the 250th birthday of Bellevue Hospital.

Although of primary interest to those who served within its walls, it will fascinate lovers of medical history as well as those politically involved in the governance of municipal hospitals and city departments of health.

I found instructive the vivid accounts of death-dealing diseases that were frequent visitors to the city in the 18th and 19th centuries. Physicians and nurses had to contend with epidemics of typhus, typhoid, smallpox, Asiatic cholera, erysipelas, tuberculosis, yellow fever, pneumonia, and puerperal fever. The leading cause of death in 1842 was delirium tremens (25.6%) and in second place was erysipelas (17.2%). In 1861–1864 a typhus epidemic took the lives of six from the medical staff of 21. Armed only with hope, dedication, and prayer, there was no talk of striking and no one refused to treat highly contagious patients. As I read this book, patients with AIDS come to mind. How can any member of our profession refuse to treat them?

The last half of the book contains a catalog of those who served on the medical staff at that time. A short biographical sketch follows each name, with the causes of death when available. Many of the names are those of famous men and include Flint, Wood, Dalton, Mott, Clark, Van Buren, Parker, Ogden, Dubois, Francis, and Hamilton. The causes of death provide the most dramatic clue to the state of the medical arts during those difficult years. Typhus, typhoid, tuberculosis, heart disease, and apoplexy were not surprising, but fatigue, exhaustion, tonsillitis, and carbuncle stir one's emotions.

Finally, Bellevue was probably the first hospital to operate an ambulance service, horse-drawn, of course. This fine book will appeal to only a few but it should be part of the library of anyone interested in the history of medical care in a large urban setting.

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BOOKS RECEIVED

AIDS

Someone Was Here: Profiles in the AIDS Epidemic. By George Whitmore. 211 pp. New York, NAL Penguin, Inc, 1988. $17.95 (hardcover)

AIDS: A Guide to Clinical Counselling. By Riva Miller and Robert Bor. 127 pp. London, Hammersmith Riverside, 1988. $23.50 (paperback)

Radiology of AIDS. Edited by Michael P. Federle, MD, Alec J. Megibow, MD, and David P. Naidich, MD. 163 pp, illustrated. New York, Raven Press, Ltd, 1988. $89.00 (hardcover)

Confronting AIDS: Update 1988. Institute of Medicine, National Academy of Sciences. 239 pp. Washington, DC, National Academy Press, 1988. $15.95 (paperbound)

ALLERGY

Food Allergy. Volume 17. Edited by Dietrich Reinhardt and Eberhard Schmidt. 301 pp, illustrated. New York, Raven Press, 1988. $49.00 (hardcover)

CARDIOLOGY

How to Read an ECG. Fourth Edition. By Margaret C. Blowers, RN, and Roberta Smith Sims, RN. 72 pp, illustrated. Oradell, NJ, Medical Economics Books, 1988. $18.95 (spiralbound)

GERIATRICS

Medical Ethics and the Elderly. A Case Book. By Mark H. Waymack, PhD, and George A. Taler, MD. 250 pp, illustrated. Chicago, Ill, Pluribus Press, Inc, 1988. $29.95 (clothbound)

Management of the Frail Elderly by the Health Care Team. By John R. Walsh, MD, Ruth Ann Tsukuda, RN, and Judy Miller, RN. 206 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1989. $32.50 (softcover)

HEALTH CARE ADMINISTRATION

Renewing the Promise: Medicare and Its Reform. Edited by David Blumenthal, Mark Schlesinger, and Pamela Brown Drumheller. 215 pp. New York, Oxford University Press, 1988. $29.95 (hardcover)

HEMATOLOGY

Handbook of Blood and Blood Products. By S. Ghosh. 117 pp. Dobbs Ferry, NY, Sheridan House, Inc, 1988. $19.95 (paperback)

INFECTIOUS DISEASE

Infective Disease in Primary Care: A Symptomatic Approach. By Robin Hull, MD. 234 pp, illustrated. Dobbs Ferry, NY, Sheridan House, Inc, 1987. $17.50 (paperback)

INTERNAL MEDICINE

Drug Interactions Index. Second Edition. By Robert T. Weibert, PharmD, and William A. Norcross, MD. 374 pp. Oradell, NJ, Medical Economics Books, 1988. $27.95 (softcover)

LEGAL MEDICINE

Testifying in Court: A Guide for Physicians. Third Edition. By Jack E. Horsley, JD, and John Carlova. 150 pp. Oradell, NJ, Medical Economics Books, 1988. $34.95 (hardcover)

Judging Medicine. By George J. Annas, JD, MPH. 448 pp, Clifton, NJ, The Humana Press, Inc, 1988. $24.95 (hardcover)

MEDICAL HISTORY

Perfecting the World. The Life and Times of Dr. Thomas Hodgkins, 1798–1866. By Amalie M. Kass and Edward H. Kass. 624 pp, illustrated. New York, Harcourt Brace Jovanovich, Publishers, 1988. $34.95 (hardcover)

Maimonides' Medical Writings. By Fred Rosner, MD. 218 pp. Haifa, Israel, The Maimonides Research Institute, 1987. $14.95 (hardcover)

MISCELLANEOUS

The Truth About Herpes. Third Edition. By Stephen L. Sacks, MD. 216 pp, illustrated. Seattle, Wa, Gordon Soules Book Publishers, Ltd, 1988. $14.95 (paperback)

Living With Stress. What Is It? Stress at Work, Stress at Home, Dangerous Life Styles, Learning to Relax, Overcoming Stress. By Cary L. Cooper, Rachel D. Cooper, and Lynn E. Eaker. 250 pp, illustrated. New York, Penguin Books, 1988. $7.95 (paperback)

Herbal Medicine for Everyone. What It Is, How It Developed, How It Can Help You, Where to Find Treatment. By Michael McIntyre. 176 pp, New York, Penguin Books. $5.95 (paperback)

The Care of Patients: Perspectives and Practices. Revised Edition. By Mack Lipkin, MD. 235 pp, New Haven, Conn, Yale University Press, 1987. $10.95 (paperback)

Podiatric Marketing and Practice Management. Edited by Barry H. Block, DPM. 265 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $37.95 (softcover)

Hidden Victims. An Eight-Stage Healing Process for Families and Friends of the Mentally Ill. By Julie Tallard Johnson, MSW, ACSW. 192 pp, New York, Doubleday, 1988. $16.95 (hardcover)

A Bridge to Peace. By Kevin M. Cahill, MD. 101 pp. New York, Haymarket Doyma, Inc, 1988. $15.00 (hardcover)

Minding the Body, Mending the Mind. By Joan Borysenko, PhD. 241 pp, illustrated. New York, Bantam Books, 1987. $8.95 (paperback)

Selling or Buying a Medical Practice. By Gary R. Schaub, MBA. 250 pp, illustrated. Oradell, NJ, Medical Economics Books, 1988. $31.95 (paperback)

Transcendence of Loss Over the Life Span. By Patricia Weenolsen, PhD. 559 pp. New York, Hemisphere Publishing Corp, 1988. $70.00 (cloth)

M.D. Doctors Talk About Themselves. By John Pekkanen. 294 pp. New York, Dell Publishing, 1988. $18.95 (hardcover)

NUTRITION

The Low Cholesterol Oat Plan: The Lower Cholesterol, Lower-Blood Sugar Cookbook. By Barbara Earnest and Sarah Schlesinger. 352 pp. New York, William Morrow & Co, Inc, 1988. $17.95 (clothcover)

PEDIATRICS

Pediatric Medicine. Edited by Mary Ellen Avery, MD, and Lewis R. First, MD. 1,408 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1989. $79.95 (hardcover)

Pediatric Head Injuries. A Handbook. By A. Loren Amacher, MD. 129 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1988. $24.50 (softcover)

PSYCHIATRY

The Psychiatric Treatment of Alzheimer's Disease. By the Committee on Aging, Group for the Advancement of Psychiatry. 175 pp, New York, Brunner/Mazel Publishers, Inc, 1988. $20.00 (hardcover)

RADIOLOGY

Interventional Radiology. Edited by Wilfrido R.

Castaneda-Zuniga, MD, and S. Murthy Tadavarthy, MD. 873 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $14.95 (hardcover)

SOCIAL WORK

Paradigms of Clinical Social Work. Edited by Rachelle A. Dorfman, MSS. 460 pp. New York, Brunner/Mazel Publishers, 1988. $45.00 (hardcover)

SURGERY

Experimental Surgery and Physiology: Induced Animal Models of Human Disease. Edited by M. Michael Swindle, DVM, and Robert J. Adams, DVM. 350 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $59.95 (hardcover)

Principles of Surgical Technique: The Art of Surgery. Second Edition. By Gary G. Wind, MD, and Norman M. Rich, MD. 280 pp, illustrated. Baltimore, Md, Urban & Schwarzenberg, 1987. $34.50 (softcover)

UROLOGY

Current Trends In Urology. Volume 4. Edited by Martin I. Resnick,MD. 190 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $48.50 (hardcover)

FROM THE LIBRARY

MEDICAL LIBRARIES OF NEW YORK STATE

The first medical libraries of the State were private libraries belonging to physicians, but the need for collections of medical knowledge for the use of all was felt even before the Revolutionary War. Writing in 1769, Peter Middleton recommended, "to those Gentlemen in practice whose judgment and experience in the diseases of the country may have enabled them to undertake the task, that they would not only study and improve upon the observations of those who have gone before them, but also collect others for their own use and for the benefit of their successors."

He gave several reasons for this step being necessary. Many changes were taking place in the practice of medicine. Many diseases then existed which were entirely unknown to the ancients: "the names both of diseases and drugs by custom are differently applied; improvements and discoveries are daily made; medicines in the highest reputation at one time are laid aside and others substituted in their place, and the forms and manners of using those retained are perpetually diversified." The most important reason given was that "our climate, way of living, and other circumstances, which ought always to be regarded in the treatment of diseases are so very much different from those of the countries whence the most useful and judicious observations are transmitted to us, that the young practitioners of this continent must be under the greatest difficulties to accommodate those foreign observations. . . . Such a collection would not only tend to the benefit of mankind in general, but would greatly redound also to the honor of the profession in America. . . ."

WESLEY DRAPER
(*NY State J Med* 1957; 57:584–594)

FDA continues search for AIDS therapies and vaccines

The US Food and Drug Administration (FDA) is overseeing more than 100 ongoing human studies to evaluate a variety of AIDS therapeutics and vaccines at various stages of development. While it is the role of pharmaceutical manufacturers, the National Institutes of Health, and other research institutions to test experimental drugs, the FDA supervises the testing process, evaluates the results, and determines whether a drug is sufficiently safe and effective for general use. All potential AIDS drugs receive a 1-AA classification, giving them the highest priority in the FDA's drug review system.

Three basic types of drug treatments dominate the investigational new drug (IND) applications approved by the FDA: antiviral agents which interfere with HIV synthesis; immunomodulating agents which help strengthen the immune system; and drugs that treat the opportunistic infections and cancers associated with AIDS, including *Pneumocystis carinii* pneumonia, candidiasis, Kaposi sarcoma, and cytomegalovirus.

Dextran sulfate, which has demonstrated some antiviral activity against human immunodeficiency virus (HIV), has received FDA approval for expanded clinical testing in patients with AIDS. The National Institute of Allergy and Infectious Diseases is conducting the multicenter trial.

The FDA recently gave the go-ahead for clinical testing of recombinant soluble CD4 protein, an experimental AIDS therapy. The CD4 protein, located on the surface of T4 helper cells, interacts with HIV and allows the virus to enter the cells, where it reproduces and destroys the host cell. By synthesizing large quantities of the CD4 protein using recombinant DNA technology, researchers hope to inject the protein into AIDS patients and use it as a decoy to divert HIV from actual T4 cells.

Congress passes first AIDS bill

It took nearly two years for the US House of Representatives to agree on a major bill aimed at strengthening research, testing, and counseling programs for AIDS (*Nature* 1988; 335:386). Combining this bill with the bill passed by the Senate this past spring, which stresses educational issues, would produce a comprehensive program for controlling the spread of AIDS.

In its final form, the House of Representatives bill demonstrates the importance and urgency of AIDS research. Specifically, the bill mandates that federal agencies participating in AIDS research hire more staff—for a total of 780 new positions—and that research grants related to AIDS be reviewed within six months of the final date for application.

Congressman Henry Waxman from California, principal sponsor of the bill, pushed for an amendment that would make experimental drugs more readily available to AIDS patients. Once an experimental drug has been approved for investigational use, patients with AIDS would have access to the drug without having to participate in a formal clinical trial.

For researchers to monitor the spread of HIV and better understand its transmission, the bill requires all states that receive federal funding provided under this bill to supply demographic data on all people found to be seropositive for HIV. The House appropriated additional funds to establish an epidemiologic data bank at the Centers for Disease Control and an international AIDS resource center at the National Library of Medicine.

The congress earmarked $400 million a year for testing and counseling of individuals at high risk for acquiring HIV. Issues such as compulsory testing and confidentiality of test results sparked intense debate. Amendments that would have required AIDS tests for all persons applying for marriage licenses, all inmates in US prisons, and all hospital patients were rejected. Members of the House also finally agreed that protecting the anonymity of persons with positive test results would encourage the population at risk for AIDS to seek testing.

Study implicates HIV as a cancer-causing virus

A recent study by Vogel et al (*Nature* 1988; 335:606–611) produced a startling conclusion: A regulatory gene of human immunodeficiency virus (HIV), when purified and introduced into the genetic make-up of mice, can induce tumors that are histologically and developmentally similar to those seen in AIDS patients with Kaposi sarcoma. "Regardless of the mechanism, the ability of the HIV *tat* gene to induce tumours in mice would suggest that HIV is a cancer-causing virus," stated the authors.

Although AIDS has been associated with several lesions, including Kaposi sarcoma, B-cell lymphoma, squamous cell carcinoma, and carcinoma of the rectum, there has been no evidence to suggest that HIV directly causes any of these malignancies. Rather, the lesions have been attributed to the underlying immunodeficiency of AIDS patients, infections with viruses other than HIV, and environmental factors.

Vogel and colleagues sought to determine whether the *tat* gene, which has been shown to regulate viral gene expression in cultured cells, can also affect expression of host genes and thereby cause diseases associated with HIV infection. They developed a line of transgenic mice by introducing the *tat* gene into the mouse germline via microinjection of DNA into fertilized eggs. After studying numerous tissues, they found that only skin cells expressed the *tat* gene. (Why other tissues did not express the gene is not clear.)

When the transgenic mice were four months old, histologic analysis of the back skin was performed. "The most prominent finding was hypercellularity of spindle-shaped cells in the dermis of male mice . . . .The histological appearance of these changes in the skin was suggestive of the early patch-type lesions of Kaposi's sarcoma seen in patients with HIV infection." In about 15% of the male transgenic mice, skin tumors developed at 12–18 months of age. "Histologically their appearance suggests a pro-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

gression or a derivation from the focal proliferation of spindle cells seen in earlier lesions," noted the authors.

Cell lines derived from the tumors did not express detectable levels of the *tat* gene product. Therefore, the authors concluded that the *tat* gene product was probably acting indirectly to produce the lesions, rather than directly affecting the neoplastic cells. The *tat* gene product would be produced in suronnding non-neoplastic skin cells and could induce chemical mediators, such as growth factors, that would initiate the malignant cell growth.

The investigators did not attribute the tumors to an immunodeficient state, because lymphoid tissue did not appear to express the *tat* gene, and the absolute number of T cells and the ratios of T cell subsets were not abnormal.

Based on the similarities between the mouse skin lesions and Kaposi sarcoma (KS) in humans, the authors proposed "that HIV, and specifically the *tat* gene product, contributes to the development of KS."

Another interesting finding concerned sexual differences in tumor development. Although the *tat* gene product was expressed at equivalent levels in both male and female transgenic mice, nearly all of the male mice suffered lesions, while none of the female mice exhibited lesions. "This male preponderance has not yet been explained but may be due to hormonal or other genetic factors."

Do screening programs for HIV yield an unacceptable false positive rate?

One obvious means of monitoring and controlling the spread of AIDS is to institute routine testing for HIV infection. Public health officials appear to agree on the need for screening of populations at high risk of acquiring HIV, including homosexuals and intravenous drug abusers. However, there has been no consensus on the value of routine HIV screening for groups at relatively low risk of infection.

"One major concern has been that the predictive value of a positive test for HIV would be very low among populations with low prevalence rates of HIV (i.e., that the ratio of false positive to true positive diagnoses would be unacceptable)," according to Burke et al (*N Engl J Med* 1988; 319:961-964). Noting the lack of actual data on the rate of false positive diagnoses in HIV screening programs, the authors sought to determine that rate in a population with a very low prevalence of HIV.

Since 1985, all civilian applicants for US military service have undergone a rigorous testing program for HIV infection, which consists of four phases, with each subsequent test dependent on a positive result for the previous test: an enzyme-linked immunosorbent assay (ELISA); a duplicate ELISA; Western blotting; and, finally, Western blotting with a second, new blood sample.

The investigators retrospectively identified a low-prevalence subpopulation based on three characteristics: "an age of 17 or 18 years, residence in a state with an applicant seroprevalence rate lower than 1.5 per 1000 and a relatively low cumulative incidence of the acquired immunodeficiency syndrome . . . , and residence in a county with a population density of less than 500 persons per square mile."

This retrospective study involved reviewing photographs of all Western blots from HIV-positive applicants, and retesting the corresponding frozen serum samples, together with positive and negative controls, using four different assay systems. The authors required that all four assays be reactive and diagnostic to constitute a true positive result; nonreactive and/or nondiagnostic findings on all four assays characterized a false positive result. Although all four assays determined the presence of HIV antibodies, they differed in the antigen source and in the test configuration.

Sixteen of the applicants in the low-prevalence subpopulation had tested positive for HIV. There was no photograph of the Western blot and no serum available for one applicant, preventing any further testing. Of the remaining 15 samples presumed to be positive for HIV, assay results were confirmatory for 14 samples, and all assay results were negative for one sample. Therefore, one in 135,187 applicants had a false positive result, for a false positive rate of 0.0007%.

The investigators than extrapolated this false positive rate for the low-prevalence subpopulation to the whole applicant population. For all applicants since October 1985, the prevalence of HIV infection was 1.48 per 1,000. Therefore, overall, the risk of a false positive diagnosis is one in 200 positive diagnoses. "The predictive value of a positive diagnosis in this program is 99.5%." It is important to note that this extrapolation process is based on the assumption that the low-prevalence subpopulation is representative of the general applicant population with respect to the prevalence of false positive test results.

The testing program used to screen military applicants has such a high specificity for several reasons: It is based on a serial testing procedure that produces multiplicative increases in specificity; Western blots are evaluated based on relatively conservative criteria; more than one blood sample is tested; and testing procedures are performed in laboratories that maintain strict quality assurance standards.

In view of these findings, the authors believe "that with adequate attention to program design and quality control, the false positive rate can be kept at an acceptably low level" to permit a reliable screening program for HIV infection in a low-prevalence population.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Roy Garrison Aiello, MD, Port Washington. Died February 14, 1988; age 77. Dr Aiello was a 1937 graduate of Boston University School of Medicine, Boston. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Catherine Dwinelle Bentley, MD, Madison, Wisconsin. Died September 26, 1987; age 82. Dr Bentley was a 1928 graduate of the State University of New York Upstate College of Medicine, Syracuse. She was a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

Adolph Frederick Bien, MD, Hanover, New Hampshire. Died September 12, 1988; age 86. Dr Bien was a 1926 graduate of the New York University School of Medicine, New York. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Fred R. Bromberg, MD, Bay Shore. Died September 7, 1988; age 78. Dr Bromberg was a 1935 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Surgeons. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Clarke T. Case, MD, Utica. Died September 12, 1988; age 70. Dr Case was a 1942 graduate of the Harvard Medical School, Boston. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the New York Academy of Medicine, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Frank Cifarelli, MD, Floral Park. Died September 6, 1988; age 70. Dr Cifarelli was a 1942 graduate of the Medical College of Wisconsin, Milwaukee. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery and the American Board of Thoracic Surgery. His memberships included the New York Society for Thoracic Surgery, the Society of Surgical Oncology, the American Association for Thoracic Surgery, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Angelo Serafe D'Eloia, MD, Penfield. Died June 23, 1988; age 82. Dr D'Eloia was a 1931 graduate of the New York Medical College, New York. He was a member of the American Geriatrics Society, the Medical Society of the County of Cattaraugus, and the Medical Society of the State of New York.

Lewis Grant Denman, MD, Livingston Manor. Died August 10, 1988; age 79. Dr Denman was a 1945 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a member of the Sullivan County Medical Society and the Medical Society of the State of New York.

Benjamin Samuel Fishman, MD, New York. Died August 27, 1988; age 79. Dr Fishman was a 1938 recipient of the Registrable Qualification Granted by English Conjoint Board. He was a member of the American Rheumatism Association, the New York County Medical Society, and the Medical Society of the State of New York.

Joseph Jerome Frank, MD, Manlius. Died September 19, 1988; age 75. Dr Frank was a 1939 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Diplomate of the American Board of Ophthalmology. His memberships included the New York Academy of Medicine, the American Academy of Ophthalmology and Otolaryngology, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Bernard D. Freundlich, MD, Brooklyn. Died September 9, 1988; age 59. Dr Freundlich was a 1954 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the New York Academy of Medicine, the American Academy of Orthopaedic Surgeons, the American Rheumatism Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Joseph John Fried, MD, New York. Died September 7, 1988; age 91. Dr Fried was a 1922 graduate of the Deutsche Univerzita Medizinische Fakulta, Praha, Czechoslovakia. He was a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Ophthalmology. His memberships included the New York Academy of Medicine, the New York Society for Clinical Ophthalmology, the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the New York County Medical Society, and the Medical Society of the State of New York.

Arthur Friedman, MD, New York. Died September 11, 1988; age 60. Dr Friedman was a 1952 graduate of the Hahnemann Medical College of Philadelphia. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Andrew J. Giambrone, MD, Rochester. Died August 12, 1988; age 79. Dr Giambrone was a 1937 graduate of the Hahnemann Medical College of Philadelphia. He was a Fellow of the American College of Anesthesiologists. His memberships included the American Society of Anesthesiologists, Inc, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Walter A. Gunther, MD, Troy. Died August 12, 1988; age 77. Dr Gunther was a

1936 graduate of the Albany Medical College of Union University, Albany. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. He was a member of the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Rensselaer, and the Medical Society of the State of New York.

Leigh J. Harris, MD, New York. Died August 24, 1988; age 36. Dr Harris was a 1977 graduate of the New York Medical College, New York. He was a Diplomate of the American Board of Ophthalmology and a member of the New York County Medical Society and the Medical Society of the State of New York.

Benjamin B. Hershkowitz, MD, New York. Died September 13, 1988; age 94. Dr Hershkowitz was a 1921 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Otolaryngology and a member of the New York County Medical Society and the Medical Society of the State of New York.

Paul S. Ingrassia, MD, Nanuet. Died August 26, 1988; age 77. Dr Ingrassia was a 1936 graduate of the New York Medical College, New York. He was a Qualified Fellow of the International College of Surgeons. His memberships included the New York Academy of Medicine, the American Society of Abdominal Surgeons, the Medical Society of the County of Rockland, and the Medical Society of the State of New York.

Francis Kwok, MD, Rego Park. Died August 24, 1988; age 72. Dr Kwok was a 1943 graduate of St Johns University (Pennsylvania Medicine School), Shanghai, Kiangsu, China. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the New York Academy of Medicine, the American Academy of Otolaryngology, the New York County Medical Society, and the Medical Society of the State of New York.

Edward Lehman, MD, New York. Died September 3, 1988; age 94. Dr Lehman was a 1918 graduate of Columbia University College of Physicians and Surgeons, New York. He was a member of the New York Pediatric Society, the American Psychiatric Association, the Association for the Advancement of Psychotherapy, the New York County Medical Society, and the Medical Society of the State of New York.

John Henry Mehrling, MD, Stony Brook. Died September 3, 1988; age 74. Dr Mehrling was a 1937 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Academy of Allergy, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Luis G. Moreno, MD, Staten Island. Died August 30, 1988; age 71. Dr Moreno was a 1945 graduate of the Facultad de Medicina de la Universidad Nacional de Colombia, Bogota, Cundinamarca, Columbia. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the American Academy of Orthopaedic Surgeons, the Richmond County Medical Society, and the Medical Society of the State of New York.

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Harry L. Mueller, Jr, MD, Binghamton. Died September 10, 1988; age 62. Dr Mueller was a 1949 graduate of Cornell University Medical College, New York. He was a Fellow of the American College of Obstetrics and Gynecology and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

George Edward Sanders, MD, Rochester. Died September 5, 1988; age 96. Dr Sanders was a 1917 graduate of the University of Illinois at Chicago Health Sciences Center, Chicago. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Robert I. Walter, MD, New York. Died August 15, 1988; age 81. Dr Walter was a 1932 graduate of the University of Rochester School of Medicine-Dentistry. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the New York Obstetrical Society, the New York County Medical Society, and the Medical Society of the State of New York.

David E. Zehner, MD, Fountain Valley, California. Died August 26, 1988; age 78. Dr Zehner was a 1938 graduate of the University of Arkansas School of Medicine, Little Rock, Arkansas. He was a member of the American Academy of Family Physicians, the Bronx County Medical Society, and the Medical Society of the State of New York.

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CLASSIFICATION CODES

CC = Clinical Conference
CPC = Clinicopathologic Conference
CR = Case Report
ED = Editorial Commentary
EP = Essay in Pediatrics
ER = Essay Review
HIST = History of Medicine
LE = Letter to the Editor
LEN = Leads from *Epidemiology Notes*
NF = News Feature

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FOR MY PATIENTS

NUMBER 5/1988

DID YOU READ YOUR COPY OF "UNDERSTANDING AIDS"?

Dear Patient,

Like all the other households in the country, your family received a copy of the brochure "Understanding AIDS." In June of this year, a copy was mailed to every home by the U.S. Public Health Service. Read yours from cover to cover. The more you know about how AIDS is spread and how it can be prevented, the more secure you will feel about your health and the less worry you will have.

Getting the AIDS Virus

The three basic ways of being infected with the AIDS virus are:

- Having sex with a person who is infected with the virus.
- Sharing drug needles and syringes with a person who is infected with the virus.
- Having had one or more blood transfusions before widescale screening of donations began in 1985

An infected mother can also pass the AIDS virus to a fetus in utero, and thus a child can be born with AIDS.

Whether you are male or female, heterosexual, bisexual or homosexual, you can get the virus by sexual intercourse. The virus may be in the semen or vaginal fluids of the infected person. Although the largest number of cases of AIDS continue to occur in male homosexuals, the number of heterosexual cases grows each year. Anal intercourse is considered the riskiest form of behavior. You won't become infected with AIDS from ordinary, everyday activities such as: handling telephones or drinking glasses, sitting on toilet seats, swimming in pools, simple kissing, or being bitten by a mosquito. It's also not transmitted by contact with saliva, sweat, tears, urine, or bowel movements of AIDS patients.

Donating blood at a blood bank has **never** been a risk for getting AIDS because each needle used is brand-new and is discarded right after the blood is drawn. ***Receiving*** blood from a blood bank does not carry a real risk of AIDS because all donors are carefully screened for risk factors. Since 1985, all donated blood has been routinely tested for the AIDS antibody. (The AIDS risk in New York State is estimated to be as low as 1 in 250,000 donations.)

Risky Behavior

Medical experts used to believe that only "certain high risk groups" of people became infected with AIDS. Now they say that ***who you are*** does not place you in danger of AIDS but rather ***what you do*** can endanger you. That means you must avoid:

- Sharing drug needles and syringes.
- Anal sex (with or without a condom).
- Vaginal or oral sex with someone who "shoots" drugs or practices anal sex.
- Sex with a pickup or prostitute or with someone you know who has several sex partners.
- Unprotected sex (that is, without a condom).

Using a Condom

If you are sexually active and don't limit your sexual activity to one partner, use a condom regularly to help prevent the spread of AIDS and other sexually transmitted diseases. Buy the ones made of latex rubber. Spermicides (which can kill the virus) or lubricants will provide extra protection.

Who Should Get the AIDS Test?

The AIDS test doesn't show that one has the AIDS disease, but it does show if you have been infected with the virus in the past.

- Be tested if you or your partner belongs to one or another of the Risky Behavior groups I listed above. I especially urge you to be tested if you plan to have a baby or are not using birth control measures and you're in a Risky Behavior group.
- Be tested if you are a hemophiliac or had received a blood transfusion between 1978 and 1985.

If you test positive for AIDS infection, you must be counseled about your future health and steps to protect your partner. People who have always practiced safe behavior — that is, no drug "shooting" and sex with one, faithful, uninfected partner — don't need to be tested.

To prevent AIDS, avoid exposure to the virus. Only you and your intimate friends can control your own exposure risks. Make a habit of Safe Behavior.

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This issue of the *New York State Journal of Medicine* contains a special additional item, "For My Patients." This feature will be run in future issues as well.

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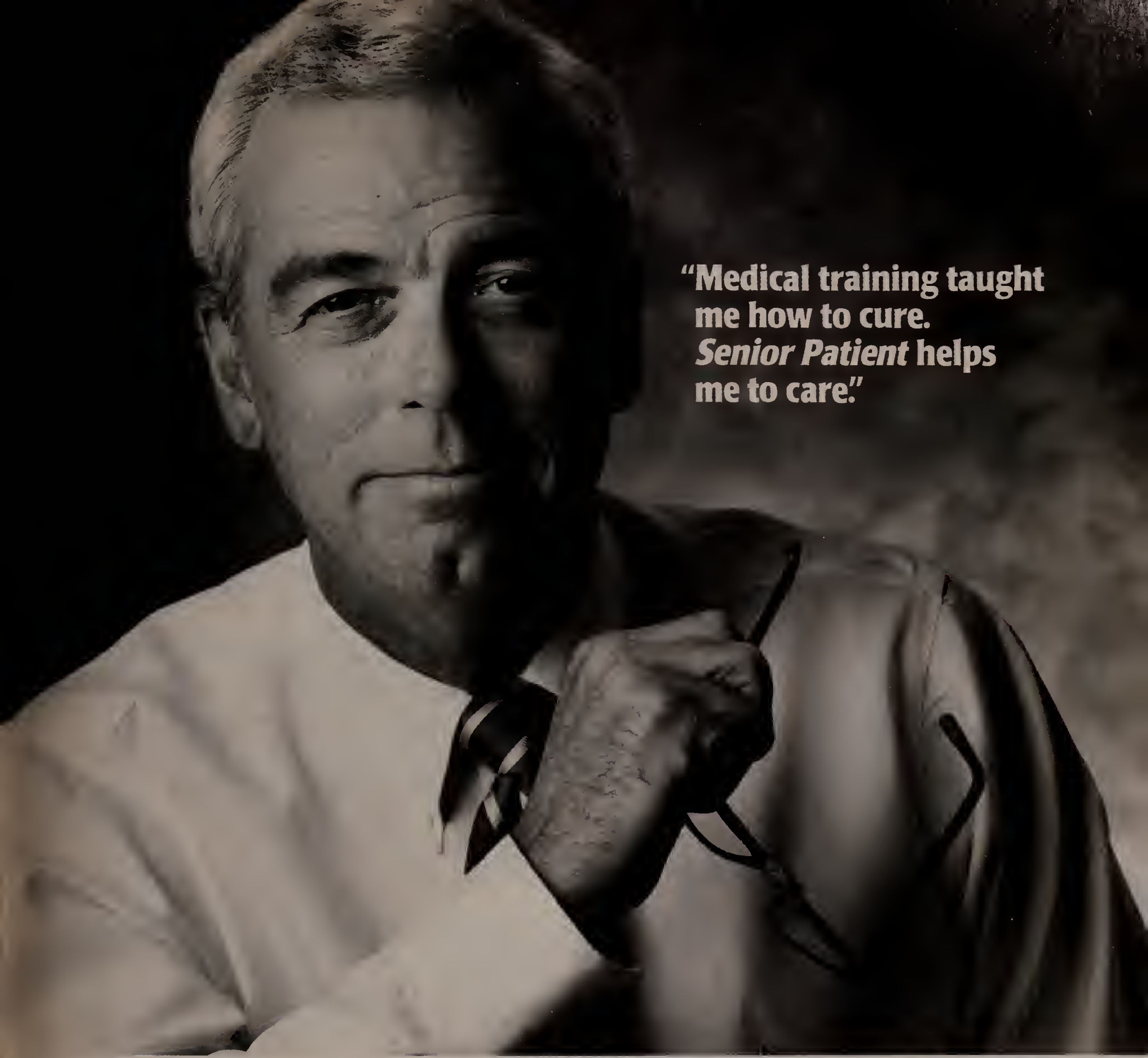
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(*continued from p 4A*)

Resort, Vail. Contact: CME Registration, American Academy of Pediatrics, PO Box 927, Elk Grove Village, IL 60009-0927. Tel: (312) 981-7884.

HAWAII

Jan 9–11. **Urogynecology.** *Jan 12–14.* **Obstetric Controversies.** Waiohai Hotel, Kauai. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024-2188. Tel: (800) 533-8811.

VERMONT

Jan 30–Feb 2. **International Pediatric Update Conference.** Trapp Family Lodge, Stowe. Contact: Lynne Wechsler, Office of CME, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

VIRGINIA

Jan 26–28. **Ethical Issues in Military Research: What Are the Issues? How Can They Be Handled?** Marriott Gateway, Arlington. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

AROUND THE WORLD

CANADA

Jan 19–20. **Second International Symposium on the Prevention of Catastrophic Sports and Recreational Injuries to the Spine and Head.** Toronto. Contact: Dr Charles H. Tator, Director, Canadian Sports Spine and Head Injuries Research Centre, 399 Bathurst St, Toronto, Ontario, Canada M5T 2S8. Tel: (416) 369-5890.

MEXICO

Jan 3–8. **18th Annual Pediatric Postgraduate Course. Pediatric Update.** 18 Cat I Credits. Pierre Marques, Acapulco. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Schneider Children's Hospital of Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Jan 14–21. **Seventh Annual Symposium Clinical Update in Anesthesiology.** Puerto Vallarta. Contact: H. Phillips, Mt Sinai Medical Center, 1 Gustave L. Levy Pl, New York, NY 10029. Tel: (212) 241-7630.

Jan 23–27. **Diagnostic Radiology.** Camino Real, Ixtapa. Contact: Radiology Postgradute Education, Registration Office, University of California, Room 569U, San Francisco, CA 94143-0766. Tel: (415) 476-5731.

VIRGIN ISLANDS

Jan 6–10. **ECG-STAT!** Frenchman's Reef Resort Hotel, St Thomas. Contact: Anne Criss Taylor, Executive Coordinator, Rogers Heart Foundation, PO Box 12588, St Petersburg, FL 33733. Tel: (813) 894-0790. □

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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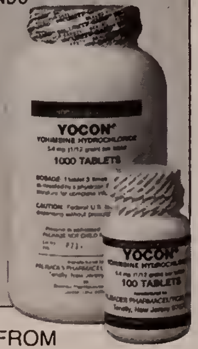
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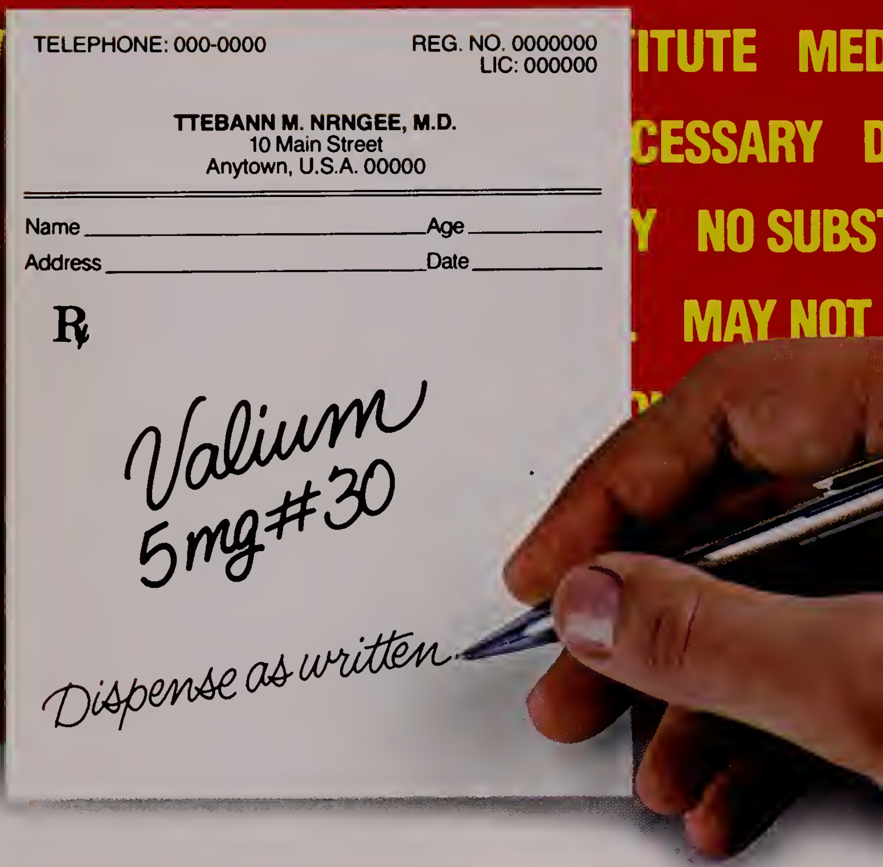

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